**Working Memory and Sensory Memory in Subclinical High Schizotypy: An Avenue for Understanding Schizophrenia?**

Sarah M Haigh1\*, Marian E. Berryhill1\*, Alexandrea Kilgore-Gomez1, Michael Dodd2

\*Co-First authors

1 Department of Psychology, Center for Integrative Neuroscience, Programs in Cognitive and Brain Sciences, and Neuroscience, University of Nevada, Reno, NV 89557

2 Department of Psychology, University of Nebraska, Lincoln, NE 68688

Corresponding author:

Sarah M Haigh, PhD

1664 North Virginia Street,

Reno, NV 89557-0296

Email: shaigh@unr.edu

Keywords: schizophrenia, schizotypy, working memory, sensory memory

Abbreviations: SM= sensory memory; WM=working memory; SSD=schizophrenia and schizophrenia spectrum disorders; EEG=electroencephalography; ERP=event-related potential; MMN=mismatch negativity; fMRI=functional magnetic resonance imaging; SN=salience network; DMN=default mode network; FPN=frontoparietal network; MATRICS= Measurement and Treatment Research to Improve Cognition in Schizophrenia; TMS= transcranial magnetic stimulation; tDCS=transcranial direct current stimulation; FM=frequency modulated; PFC=prefrontal cortex

**Abstract**

The search for robust, reliable biomarkers of schizophrenia remains a high priority in psychiatry. Biomarkers are valuable because they can reveal the underlying mechanisms of symptoms and monitor treatment progress, and may predict future risk of developing schizophrenia. Despite the existence of various promising biomarkers that relate to symptoms across the schizophrenia-spectrum, and despite published recommendations encouraging multivariate metrics, they are rarely investigated simultaneously within the same individuals. In those with schizophrenia, the magnitude of purported biomarkers is complicated by comorbid diagnoses, medications, and other treatments. Here, we argue three points. First, we reiterate the importance of assessing multiple biomarkers simultaneously. Second, we argue that investigating biomarkers in those with schizophrenia-related traits (schizotypy) in the general population can accelerate progress in understanding the mechanisms of schizophrenia. We focus on biomarkers of sensory and working memory in schizophrenia and their smaller effects in individuals with *nonclinical* schizotypy. Third, we note irregularities across research domains leading to the current situation in which there is a preponderance of data on auditory sensory memory and visual working memory, but markedly less in visual (iconic) memory and auditory working memory, particularly when focusing on schizotypy where data are either scarce or inconsistent. Together, this review highlights opportunities for researchers without access to clinical populations to address gaps in knowledge. We conclude by highlighting the theory that early sensory memory deficits contribute negatively to working memory and vice versa. This presents a mechanistic perspective where biomarkers may interact with one another and impact schizophrenia-related symptoms.

**Introduction**

Schizophrenia and schizophrenia spectrum disorders (SSD) remain a prominent public health risk. There are persistent gaps in knowledge surrounding the biological mechanisms of symptoms affecting cognition, behavior, and emotion. To track the emergence and progression of symptoms, there is a need for reliable biomarkers of schizophrenia and SSD. These biomarkers can reveal underlying mechanisms as well as complementing existing clinical interviewing methods. Biomarkers may also be used to predict conversion from high-risk to diagnosis (Näätänen et al., 2016). Currently there is no gold standard biomarker that reliably tracks symptom severity (Lieberman et al., 2019), although there is convergence regarding risk factors and cognitive deficits (Mohn-Haugen et al., 2022). This has led to many calls for multivariate biomarker metrics to more comprehensively assess an individual’s risk level (e.g., (Cannon et al., 2016; Price et al., 2006; Seidman, Shapiro, et al., 2016). Although this opinion is not controversial, there remains a tendency to focus on single biomarkers and their predictive power. This may be due to the simplicity of understanding how a single biomarker reflects underlying processing schizophrenia and SSD, and to the added technical difficulties and financial challenges involved when measuring multiple biomarkers. Here, we support the call for a multivariate biomarker, and we extend this view by suggesting that to accelerate our understanding of the neural mechanisms associated with symptoms, it would be valuable to test across the entire SSD, including neurotypical individuals with *high subclinical schizotypy* symptomatology in the general population. This suggestion has the added benefit of increasing the pool of researchers available to investigate questions of clinical relevance, but who do not have access to clinical populations.

Here, we define *schizotypy* as the subclinical characteristics associated with schizophrenia that are prevalent within the general population. Schizotypy in this context is not a diagnosis as the schizophrenia-related traits are not severe enough to warrant intervention. It is unknown if participants with high schizotypy traits will develop schizophrenia and/or psychosis in the future, as many of these studies are cross-sectional in design and recruit from the general population. However, one value of focusing on schizotypy is that participants with psychiatric or neurological diagnosis can be removed. This reduces the likelihood of comorbid conditions and medications that may confound the findings. Therefore, it would be expeditious to include a larger swath of the spectrum to include the larger *subclinical* population, and to combine discrete biomarkers that track symptom severity.

Existing biomarkers include: genetic polymorphisms (Ettinger et al., 2014), oculomotor abnormalities (reviewed in: (Levy et al., 2010)), and atypical neural responses detected using EEG and fMRI. Unfortunately, many of these biomarkers were identified in isolation rather than in tandem with other biomarkers associated with a particular sensory or cognitive domain. This is despite success when taking a multifactorial approach to monitoring multiple neural (Kent et al., 2004; Price et al., 2006; Ranlund et al., 2018; Taylor et al., 2017) and behavioral (Seidman, Shapiro, et al., 2016) measures to help distinguish between schizophrenia and other psychosis-related conditions such as bipolar disorder. Due to the success seen in those diagnosed with schizophrenia, we recommend the same multifactorial approach for the subclinical schizotypy population. For the current review, we revisit EEG biomarkers from several related literatures: abnormal *sensory* memory (SM) and *working* memory(WM), in both auditory and visual modalities. These topics have well-developed literatures with a substantial foundation in the clinical schizophrenia and SSD populations. We focused on sensory and working memory specifically, to explore how these biomarkers may interact due to downstream deficits impacting later processing. We discuss the use of sensory and working memory measures as biomarkers of symptom severity from a cross-sectional perspective - how do these markers reflect on their internal processes at this moment? We conclude by discussing how some of the biomarkers we discuss may be promising as predictive biomarkers of the onset of psychosis and schizophrenia, and briefly summarize some of the literature up to this point.

**Scope of Review: Auditory *and* Visual, Sensory *and* Working Memory**

Perturbed bottom-up functioning impairs upstream cognition (Javitt, 2009; Javitt & Sweet, 2015). If *sensory* encoding is impaired then the information derived from the impoverished signal will produce further problems. An immediate consequence of impaired SM is reduced information available for WM. In addition, WM deficits in schizophrenia could negatively impact SM too: If WM is inefficient, then manipulating incoming sensory information will also be impaired. Improvements anywhere along this loop could have cascading consequences (Figure 1). A first step is to characterize the relationships between SM and WM biomarkers and how they are impacted along SSD.

**[Insert Figure 1 here]**

Because psychiatric populations are difficult to access, recruit and retain in research studies we must leverage all data points including findings from the subclinical population. We subscribe to the view that the general population falls along a spectrum of schizotypal symptoms (theoretical review in (Grant et al., 2018); see Figure 2 for illustration). People with more symptoms, but within neurotypical range, are referred to as having ‘high schizotypy’. First degree relatives of those with schizophrenia typically have high schizotypy ratings, and are considered to be at high clinical risk (Kalmady et al., 2020; Le et al., 2020). As noted, studies in first degree relatives and in neurotypical individuals with high schizotypy scores avoid complications and confounds such as medication type, dosage, duration, and mental health comorbidities. An important challenge in *subclinical* populations is that any biomarker would be weaker. We began our review by searching specific sensory and working memory terms in PubMed and counting the number of studies identified; see Table 1. We then summarize the reports of abnormal auditory and visual SM in SSD and turn to WM before discussing whether empirically derived biomarkers can serve as indicators of clinical remediation.

**[Insert Figure 2 here]**

**[Insert Table 1 here]**

# Sensory Memory in Schizophrenia Spectrum Disorders

Sensory memory (SM) refers to the window over which the neural response to a sensory stimulus remains active after the stimulus is withdrawn. The duration of the memory trace is brief (e.g., (Sperling, 1960) and considered to be preattentive (Naatanen et al., 1982). Measures of SM are appealing as a clinical biomarker because they are unaffected by attentional focus - which is compromised in schizophrenia. The SM trace is an input for predictive coding and very sensitive to changes in sensory stimuli (Winkler, 2007; Winkler et al., 2009). It is also extremely adaptive to the current environment (May & Tiitinen, 2010; May & Tiitinen, 2007). Abnormal SM in those with schizophrenia *may* be domain general (Neuhaus et al., 2013; Saint-Amour et al., 2007). To date the most research has been conducted in audition and vision, although sensory memory in the somatosensory and olfactory domains is reduced in schizophrenia (Somatosensory: (Ludwig et al., 2016) Haigh et al., 2016; Olfactory: (Moberg et al., 2003; Wu et al., 1993)) and can be tracked using similar methods to vision and audition (Restuccia et al., 2009). Despite the numerous reports of oculomotor dysfunction in schizophrenia and SSD, relatively little has been reported in visual SM. We will now discuss each in turn.

## Auditory Sensory Memory

Poor auditory SM impairs the encoding of simple parameters of sounds. Neural measures of *auditory* SM are a clinically useful biomarker. They are minimally affected by attention and can be measured before the participant is aware of the percept, thereby isolating early sensory processing from upstream cognitive processes.

In people with schizophrenia there are numerous examples of deficits in auditory SM, including impaired pitch discrimination (distinguishing between tones of different pitches; (McLachlan et al., 2013), tone-pair matching (Rabinowicz et al., 2000), sound localization (Donde et al., 2020; Donde, Martinez, et al., 2019), discrimination of intensity, duration and sequences (Schnakenberg Martin et al., 2018) and auditory scene segregation (Ramage et al., 2012; Weintraub et al., 2012). Moreover, there is evidence that mismatch negativity amplitude is lower in those with schizophrenia, reflective of a general auditory sensory memory deficit (Catts et al., 1995), lower educational achievement before the onset of schizophrenia, worse cognitive symptoms, poorer global function, and longer duration of illness (Friedman et al., 2012). There is some question over whether these abnormal auditory responses are due to sensory *memory* or if they are due to problems encoding the information in the first place. While it is difficult to disentangle how poor early encoding impacts the ability to encode short-term memory traces, there is evidence that both individuals with chronic schizophrenia and individuals at their first-episode of psychosis show deficits in encoding deviants in complex patterns (Haigh et al., 2016; Haigh, Coffman, et al., 2017; Salisbury et al., 2018), suggesting that memory impairments persist after encoding. However, other aspects of audition are preserved, including the time-course of auditory SM (March et al., 1999) and the ability to categorize syllables (Dale et al., 2010; Haigh et al., 2019). Categorization can also show the expected shifts when participants are habituated to one syllable (for example, adapting to one syllable category makes it easier to detect a syllable in another category) (Haigh et al., 2019) suggesting residual plasticity in the auditory system. Finally, more closely associated with specific symptoms of schizophrenia, auditory hallucinators reporting phenomenon of speech and voices exhibit worse auditory SM (McCleery et al., 2018), highlighting the link between auditory processing and symptomology.

There is evidence of similar, but smaller, SM abnormalities in schizotypy that are similar to those reported in schizophrenia. However, the number of studies focusing on schizotypy are relatively small and often identify potential biomarkers in isolation. For example, sensory cortices send a ‘corollary discharge’ indicating a self-generated response compared to external in origin (Sperry, 1950). Auditory corollary discharges are produced in the superior temporal gyri (Creutzfeldt et al., 1989; Muller-Preuss & Ploog, 1981) and are manifest by reduced auditory N1 ERP responses to *self-generated* speech compared to the N1 response to externally generated speech (Ford & Mathalon, 2005). Individuals with schizophrenia show equivalent N1 amplitudes for self-generated and externally generated speech, suggesting they cannot differentiate between conditions. Impaired corollary discharge could reflect a confusion between self-generated auditory hallucinations and externally generated speech (Ford & Mathalon, 2005). In schizophrenia there is a reliable reduction in N1 amplitude (Salisbury, 2010; Shelley et al., 1999); reviewed in: (Rosburg et al., 2008) that correlate with the perceptual deficit (Javitt et al., 2000). Similarly, individuals with high schizotypy show equally large N1 responses to external and self-generated speech, whereas neurotypical participants with low schizotypy show N1 suppression to self-generated speech (Oestreich et al., 2016). Those high in schizotypy show reduced precision in auditory sensory memory despite normal rates of acquisition and decay (Bates, 2005).

In addition, reductions in well-characterized cognitive-related ERPs in combination with sensory ERPs to simple auditory stimuli have shown some utility. For example, reductions in the P3 complex (Javitt et al., 1995; Pritchard et al., 1985), are evident early in the disease course (Devrim-Ucok et al., 2016; Salisbury et al., 2020), as are reductions in steady-state potentials (Light et al., 2006), and in the slow-wave potentials associated with auditory scene segmentation (Coffman et al., 2018; Coffman et al., 2017) that correlate with hypoactive midcingulate cortex (Coffman et al., 2018). Reduced amplitude of P50, mismatch negativity, P3, and antisaccades (making eye movements in the opposite direction to a cue) are reported in individuals with schizophrenia, and significant reductions in P50 and P3 in relatives, compared to controls. However, combining all four measures in a multivariate analysis permitted the model to accurately (80%) classify group assignment (Price et al., 2006). In short, throughout early auditory sensory processing there are atypical ERPs in the schizophrenia population. Further investigation in high schizotypy individuals to expressly investigate auditory SM would be valuable to understand the boundary conditions between intact and impaired function in SSD.

## Auditory Mismatch Negativity

The most robust neural measure of SM is the **mismatch negativity (MMN).** In neurotypical individuals, fronto-central electrodes record larger (more negative) responses to an infrequent deviant stimulus than to the standard stimulus at around 100-200ms after stimulus-onset (Naatanen, 1985). The MMN is calculated by subtracting the EEG response to the standard tone from response to the deviant tone producing a difference waveform (Näätänen, 1985). In individuals with chronic schizophrenia, the MMN is *smaller* (Cohen’s d: 0.99) (Javitt & Sweet, 2015; Umbricht & Krljes, 2005), and magnetic activity in the planum temporale (secondary auditory cortex) is reduced during auditory SM tasks that generated a smaller (magnetic) MMN (Kircher et al., 2004). Reduced MMN generalizes across stimuli (Erickson et al., 2016; Umbricht & Krljes, 2005) and includes responses to pattern deviants based on grouping (Haigh et al., 2016), pitch (Haigh, Matteis, et al., 2017), rules (Haigh et al., 2019), and phonemes (Fisher et al., 2019).

In SSD the degree of MMN reduction correlates with symptom severity (Fisher et al., 2019; Light & Braff, 2005). Greater reductions are apparent in chronic schizophrenia compared to individuals at high-risk (Shin et al., 2012). Local and global change detection have both been found to be poor in schizophrenia suggesting evidence of abnormal predictive coding that cannot be due to abnormal adaptation to simple stimulus parameters (Kirihara et al., 2020). Together, these data highlight a pervasive deficit in preconscious auditory SM that occurs early in the auditory processing stream (Escera & Malmierca, 2014). MMN is sensitive to the balance between excitatory and inhibitory neurotransmitters in the auditory cortex (Holliday et al., 2018). There is some debate regarding neurotransmitter systems’ roles in the auditory MMN. Some evidence indicates a restored MMN with nicotinic agonists, implicating acetylcholinergic modulation (Baldeweg et al., 2006) but see (Knott et al., 2014), whereas other studies implicate acetylcholinergic (Kantrowitz et al., 2018) or serotonergic influence (Sehatpour et al., 2022).

In SSD more broadly, there is some evidence of reduced MMN. For example, MMN to pitch oddballs successfully discriminates between neurotypicals and people diagnosed with schizotypal personality disorder (Niznikiewicz et al., 2009). Furthermore, combining MMN responses to multiple deviants: duration, gap or silence instead of a tone, or location of tone, lead to identification accuracy up to 80.5% when using multivariate machine learning techniques. The combined MMN accuracy correlated with global assessment of functioning, highlighting auditory MMN as a biomarker of symptoms in chronic schizophrenia (Taylor et al., 2017). MMN deficits are evident in early course schizophrenia (within 2-years of the first episode of psychosis; (Salisbury et al., 2007)). There are inconsistent findings in those within 6-months of initial psychosis (Erickson et al., 2016; Haigh, Coffman, et al., 2017), and in those at-risk of developing schizophrenia (compare (Shin et al., 2012) to (Hsieh et al., 2019)), or who are unaffected first-degree relatives (Magno et al., 2008). Several accounts address why MMN deficits are greater in those with advanced disease. One theory is that MMN is tied to symptom severity (Kantrowitz et al., 2018), as MMN correlates with negative symptoms in chronic (>5 years) patients (Catts et al., 1995). Of relevance, the MMN correlates with negative symptoms in *first*-episode patients, and with measures of social functioning (Murphy et al., 2020). Certainly, individuals with chronic schizophrenia have the most severe symptoms and neurological consequences. For example, MMN correlates with reduced grey matter volume in primary auditory cortex and both MMN deficits and the great matter volume reductions increase with the duration of time past first-episode (Salisbury et al., 2020; Salisbury et al., 2007). A recent meta-analysis indicated that MMN shows progressively greater abnormality, with first degree relatives showing the smallest deficit, followed by individuals at high-risk of developing schizophrenia, and finally recently diagnosed (early-stage schizophrenia), compared to those with chronic schizophrenia (Erickson et al., 2016); Figure 3).

**[Insert Figure 3 here]**

A few inconsistent MMN findings are worth mentioning. First, some people ‘recover’ without repeated psychotic episodes, and they do not exhibit an abnormal MMN (Kim et al., 2020). This highlights a key gap - few longitudinal studies have been conducted to ascertain how biomarkers change over time or with vacillating symptoms. Second, a small study in the subclinical population reported that high scorers on the suspiciousness subscale had *larger* MMN amplitudes than low scorers (Broyd et al., 2016). Thus, the current literature presents competing perspectives: 1) that increased symptom load reliably predicts abnormal MMN (e.g., (Claridge & Beech, 1995), but 2) in the subclinical population more subtle patterns of preserved and impaired MMN emerge. Clearly, more research is needed to address these gaps in our understanding.

## Oculomotor and Low-Level Visual Deficits

Despite the wealth of data cataloging impaired auditory SM in schizophrenia and SSD, there is relatively little in the visual SM domain. This is surprising given the prevalence of oculomotor deficits in schizophrenia, suggesting that the visual system is affected. Before describing the studies that have reported on visual SM, we note research pinpointing oculomotor deficits in SSD. We address this issue first because oculomotor deficits lead to atypical sampling of the environment and these contribute to abnormal visual sensation. Oculomotor responses are disrupted along SSD with atypical performance in saccadic tasks (e.g., double-step, adaptation, anti-saccade) and smooth pursuit (O'Driscoll & Callahan, 2008; Thakkar & Rolfs, 2019; Wolf et al., 2021). A proposed mechanism is that abnormal corollary discharge (e.g., (Feinberg, 1978) disrupts knowledge about eye position, resulting in inaccurate eye movements with more interruptions during visual tracking. Findings in nonhuman primates and lesion studies implicate impaired signaling in the mediodorsal thalamus, which serves as an intermediary between subcortical eye movement control areas (e.g., superior colliculus) and frontal eye fields (Thakkar & Rolfs, 2019). Inactivating this pathway creates a mismatch between where an organism believes their eyes are directed and where they are actually looking and thus alters visual perception (Cavanaugh et al., 2016). In those with schizophrenia the abnormal visual scan patterns of faces and social settings contribute to aberrant social interactions (Patel et al., 2020). Oculomotor deficits contribute to higher order cognitive deficits because those with schizophrenia sample and perceive the environment abnormally.

Approximately ~60% of individuals with schizophrenia have distortions in visual perception (Phillipson & Harris, 1985) and >33% experience visual hallucinations (Silverstein & Lai, 2021). The range of perceptual deficits is broad and includes worse performance in assessments of contrast sensitivity (Harper et al., 2020), detection of contour (Keane et al., 2014), color (Fernandes et al., 2019), biological motion (Okruszek & Pilecka, 2017) but see (Keane, Peng, et al., 2018), faces (McCleery et al., 2015), and stronger afterimages (Thakkar et al., 2021). The extent of afterimage deficits is clinically relevant as they predict illness severity (Keane, Cruz, et al., 2018).

Similarly, accounts for visual deficits in schizophrenia spectrum disorders identify abnormalities in the anatomy of the retina (reviewed in reviewed in (Bernardin et al., 2017; Silverstein et al., 2020), as well as atypical network-level connectivity, both hypo- and hyper- connectivity, including cortical - medial temporal lobe abnormalities (reviewed in: (Silverstein & Lai, 2021). Together, the consistent oculomotor and low-level visual deficits suggest that processing from as early as the retina is abnormal in schizophrenia.

## Visual Sensory Memory Biomarkers of Symptom Severity

Given the range of oculomotor and low-level visual deficits identified in SSD, it is surprising how little work has been conducted in iconic memory. Several studies investigating iconic memory decay in those with schizophrenia have reported no differences in schizophrenia (Hahn et al., 2011; Knight et al., 1978). We know of no research evaluating iconic SM in schizotypy in the general population.

Similarly, it is noteworthy that in comparison to the high volume of research on the auditory MMN, there is little research assessing the visual MMN in schizophrenia and in SSD. However, atypical visual MMN are documented and confirm that abnormal responses are not isolated to the auditory system. For example, the visual MMN is significantly reduced in individuals with schizophrenia in an oddball task using letters as stimuli (Neuhaus et al., 2013), visual motion direction (Urban et al., 2008) or horizontal compared to vertical grating patterns as oddballs (Farkas et al., 2015), with a similarly large effect size in group differences. The same pattern of reduced MMN amplitude has been characterized in studies using emotional faces (Csukly et al., 2013; She et al., 2017; Yin et al., 2018), in which the MMN reduction correlates with behavioral performance on an emotion recognition task (Csukly et al., 2013). One recent finding reports that individuals with schizophrenia showed normalvisual MMN elicited by fearful faces but diminished amplitude MMNs elicited by neutral faces (Vogel et al., 2018). A new study has now identified larger visual MMN to happy compared to sad and neutral faces and found that MMN amplitude to the happy faces correlated with schizotypy scores, and interestingly, with measures of autism characteristics, highlighting the difficulty with MMN and specificity of diagnosis (Ford et al., 2022).

Another ERP that is linked to sensory memory is the P1. Visual P1 responses were significantly smaller in schizophrenia in response to low (but not high) spatial frequency gratings that was not related to visual acuity (Farkas et al., 2015). Although the P1 amplitudes did not correlate with symptoms, they were related to cognitive performance, as well as education level, duration of illness, and measures of global functioning (Farkas et al., 2015). Visual MMN has also been elicited in response to changes in the direction of moving dots and is significantly reduced in schizophrenia (Urban et al., 2008). Visual MMN reduction was again associated with global functioning, general symptoms, and medication dosage (Urban et al., 2008), highlighting the parallels between visual and auditory MMN. Despite these similarities across the modalities, we found no findings describing the visual MMN in other SSD or high schizotypy participants.

In summary, despite notable visual and oculomotor involvement in SSD, and a wealth of data demonstrating *auditory* SM deficits, little attention has been paid to *visual* SM specifically and no evidence of atypical iconic memory in SSD. Expanding visual SM research in the SSD population is needed to complement the auditory SM work and to deepen our understanding of visual SM contributions to other aspects of cognition.

# Working Memory Deficits in Schizophrenia Spectrum Disorders

Working memory (WM) deficits are a canonical deficit in people with schizophrenia (for reviews, (Forbes et al., 2009; Lett et al., 2014; Reichenberg, 2010), those with high schizotypy (Siddi et al., 2017), and those at high-risk of psychosis (Millman et al., 2022). WM impairments are broad and include verbal (reviewed in (Seabury & Cannon, 2020), visuospatial, and executive functioning aspects of WM (Barch & Ceaser, 2012; Forbes et al., 2009). It is worth noting that poor WM performance in people with schizophrenia is associated with a variety of neural differences including reduced gray matter volume (Du et al., 2022; Kochunov et al., 2022), abnormal (hypo- and hyper-) connectivity patterns (Ding et al., 2019; Du et al., 2022; Fryer et al., 2015; Hashimoto et al., 2010; Schutte et al., 2021; Seabury & Cannon, 2020; Unschuld et al., 2014), hypoactive clusters (Seabury & Cannon, 2020), and abnormal oscillations (Reilly et al., 2018). At a lower level, one account of the WM deficit in schizophrenia suggests that PFC *inhibition* is disrupted, leading to impaired WM (Meiron et al., 2022). PFC inhibition is thought to be driven by abnormal interneuron function, which in turn reduces gamma oscillation amplitude (Reilly et al., 2018); see also (Toader et al., 2020). A challenge in specifying underlying neural mechanisms of the WM deficit is that there is variability across samples with different patterns emerging in distinguishable subgroups (Rodriguez et al., 2019). Advances in neuroimaging provide important future paths for identifying biomarkers of working memory that reflect schizophrenia symptoms, and for producing therapies to mitigate symptoms. In the paragraphs below, we specify the findings specific to auditory and visual WM as a parallel to the sections on auditory and visual SM above.

## Auditory Working Memory

Auditory WM is impaired across SSD. Deficits are known in schizophrenia, their first-degree relatives (Seidman et al., 2012; Seidman, Pousada-Casal, et al., 2016), and in the high-risk population (Higuchi et al., 2013; Rutschmann et al., 1980), as is verbal WM (Seabury & Cannon, 2020). Importantly, this deficit is apparent even in early-course schizophrenia (Gonzalez-Blanch et al., 2006) and is associated with abnormal neural responses (Leicht et al., 2015; Papageorgiou et al., 2001). Importantly, auditory WM performance predicts the presence of auditory hallucinations (Gisselgard et al., 2014; Jenkins et al., 2018). Therefore, similar to auditory SM, WM deficits may be related to *symptoms* rather than onset of psychosis.

Potential WM biomarkers are associated with altered neural function and patterns of neural connectivity. The neural correlates of WM deficits include the PFC (for a review, see (Hashimoto et al., 2010), and relevant sensory cortices (e.g., (Javitt & Sweet, 2015; Menon et al., 2001; Stevens et al., 1998). However, when investigating the neural correlates associated with abnormal WM functioning in schizophrenia, there is inconsistency. Several studies reported PFC *hyperactivity*, whereas others reported the opposite (for a review see (Leitman et al., 2005). Other brain areas implicated in WM retrieval include parietal regions (for a review, see (Funahashi, 2017; Hamilton et al., 2018; Olson & Berryhill, 2009). Parietal function is also abnormal in schizophrenia particularly in those who hallucinate (Hashimoto et al., 2010; Wible et al., 2009) and hypoactive temporal lobe activity (Wible et al., 2009) correlates with schizophrenia symptom severity (Menon et al., 2001). More recent analyses investigating altered connectivity extend these regions-of-interest findings by identifying network dysfunction. For example, increased dorsolateral and medial PFC connectivity in those with schizophrenia and their nonclinical first-degree relatives, compared to controls (Unschuld et al., 2014), and within left superior frontal gyrus (Ding et al., 2019). Several distinct patterns of hyper- and hypo- connectivity are associated with deficits in verbal WM in people with schizophrenia – including altered salience network (SN), default mode network (DMN), and frontoparietal network (FPN) interactions (Rodriguez et al., 2019). Together, there is a network-level deficit in schizophrenia that impacts auditory WM.

Some evidence suggests auditory WM deficits are associated with other auditory-related symptoms such as auditory hallucinations (Geng et al., 2020; Gisselgard et al., 2014; Jenkins et al., 2018), and auditory processing deficits (Moschopoulos et al., 2021). For example, deficits in MMN to pitch deviants were related to deficits in prosody processing (non-verbal communication of emotion) in individuals with schizophrenia (Leitman et al., 2005) and P50 suppression in the auditory ERP correlated with WM performance (Hamilton et al., 2018). Network connectivity also identifies altered DMN activity associated with auditory hallucinations (Geng et al., 2020). Finally, there is an intriguing observation that the visual word form area engages during auditory perception and WM in people with schizophrenia (Herman et al., 2020), suggests that broadly altered structure and function underlies auditory WM function.

Worse WM performance is implicated in schizotypy (Park & McTigue, 1997; Siddi et al., 2017), particularly in those who displayed more positive and negative symptoms (Schmidt-Hansen & Honey, 2009), and was associated with worse communication abilities (Kerns & Becker, 2008). It has also been suggested that poorer working memory performance may be a biomarker for increased risk of developing psychosis due to the large effect sizes shown in schizotypy (Siddi et al., 2017). However, there is debate as to the robustness of the WM deficit (Lenzenweger & Gold, 2000) and whether the effect is due to impaired attentional ability, with some evidence indicating dissociable deficits (Marsh et al., 2017). In summary, abnormalities in auditory WM in schizophrenia are seen in diminished form in individuals with high schizotypy. This supports the idea that traits associated with schizophrenia are on a spectrum. Notably, compared to the auditory SM research, there is remarkably little research conducted on biomarkers of auditory WM deficits across the SSD.

## Visual Working Memory in Schizophrenia Spectrum Disorders

A broad literature investigates visual WM deficits in people with schizophrenia and related disorders. Deficits in object, spatial and verbal WM are well-documented in review articles (e.g., (Barch, 2005; Haenschel & Linden, 2011; Piskulic et al., 2007). Briefly, across measures of accuracy, reaction time, and confidence, individuals with schizophrenia broadly perform less accurately, more slowly, and less confidently than neurotypicals. Impaired WM extends to the at-risk and schizotypy populations. In the at-risk population, two recent meta-analyses point to WM performance as reflecting symptom severity. Overall cognition, attention, processing speed, and WM were found to be significantly worse in those who were high-risk compared to healthy controls using the MATRICS Consensus Cognitive Battery (MCCB; (Zheng et al., 2018). Including both visual and auditory measures of working memory and symptoms using the Schedules for Clinical Assessment in Neuropsychiatry was able to distinguish between individuals diagnosed with schizophrenia, their unaffected first-degree relatives, and non-clinical individuals (Kent et al., 2004). Importantly, neural accounts for the visual WM impairment note the contributing factor of abnormal SM and atypical prefrontal function (e.g., (Haenschel & Linden, 2011).

A few biomarkers are associated with visual WM in SSD. There is a lower amplitude P100 (P1) responses over electrode site Oz in individuals with schizophrenia (Haenschel et al., 2009), and in those with high schizotypy (Koychev et al., 2010) to a visual delayed discrimination task, highlighting the contribution of early sensory processing on later cognition. Others report that schizophrenia participants have abnormal theta power during n-back performance, and reduced P100 in response to TMS pulses (Hoy et al., 2021). Importantly, atypical gamma oscillations can be observed in first-episode psychosis (Missonnier et al., 2020). Additional effects likely arise from gamma-band oscillatory activity during WM tasks that fails to increase with increased WM load in those with schizophrenia (Basar-Eroglu et al., 2007; Cho et al., 2006). A second biomarker of encoding-related WM deficit is the N2pc, which is also reduced in those with schizophrenia (Mayer et al., 2020). Broadly atypical frontal lobe activations in at risk youth (van Gool et al., 2022) and connectivity patterns are known across schizophrenia spectrum disorders (Briend et al., 2020; Schmidt et al., 2014), and at risk samples (Schutte et al., 2021).

# What Does This Mean for Biomarkers of Schizophrenia-Spectrum Disorders?

In surveying the SSD literature, a patchy pattern emerges: biomarkers associated with *auditory* SM (particularly the MMN) and *visual* WM in individuals with schizophrenia have been examined in detail and appear to be related to symptom severity. In contrast, the complement, visual SM and auditory WM, receive less attention. Progress is also hampered because there is little work conducted in SSD apart from those with a schizophrenia diagnosis. Diagnosed individuals often have comorbid diagnoses and require medications which can obscure the relationship between symptoms and biomarkers. To gain a foothold in the untapped areas (see Table 1), we argue that filling in the missing cells (visual SM, auditory WM) across SSD will accelerate the pace of data collection and expedite our ability to distinguish between competing theoretical perspectives. A final deficiency is that apart from the auditory MMN, there is little consistency in methodology, making studies difficult to generalize across. The standardization of auditory MMN procedures may be one of the reasons why the auditory MMN is one of the more compelling biomarkers of schizophrenia.

Our goal is to make clear that countering these oversights may improve the identification and characterization of useful biomarkers to better assess and mitigate SSD. For many researchers, including ourselves, gaining access to substantial clinical populations is improbable. Testing special populations, especially those with psychosis, impose ethical and practical difficulties with regards to care and consent. In contrast, evaluating subclinical populations provides a useful option in investigating SSD. It is also a conservative approach because those with high schizotypy are expected to show smaller, more modest effects than those with psychiatric diagnoses. As noted, perceptually, the auditory MMN is the best studied biomarker associated with schizophrenia and abnormalities persist across SSD, including in the subclinical population. In visual WM, the range of analyses is more varied without commitment to a particular biomarker of SSD. The absence of clear biomarkers associated with the complementary areas of visual SM and auditory WM make for a puzzling circumstance. Assuming a similar pattern of sensory and working memory abnormalities in schizotypy to those found in diagnosed schizophrenia, focusing on schizotypy can help identify causal mechanisms that are linked to symptom severity. In short, we encourage ERP research in auditory and visual SM, and auditory and visual WM, preferably in the same clinical individuals as much as possible, and across SSD and subclinical populations.

## How Could Biomarkers of Symptom Severity Improve Clinical Outcomes?

One goal of documenting behavioral deficits and associated biomarkers of symptom severity in schizotypy is to improve clinical outcomes, potentially by identifying appropriate treatments related to the underlying mechanisms. Biomarkers could also document a patient’s progress, such as improvements of deterioration over time, and potentially identify the mechanism of improvement. For example, there is growing evidence that sensory training methods can improve cognition in schizophrenia. Auditory training methods that require learning to discriminate between different frequency modulated sweeps improve auditory processing speed (Biagianti et al., 2016) and executive functioning (Dale et al., 2016). Extending this observation, multisensory (visual and audio) training on three simultaneity judgment tasks reduced the temporal binding window in a neurotypical group, with effects lasting seven days. The multisensory versions had bigger effects than the unisensory versions. Training duration had little effect on improvements in the temporal binding window (Zerr et al., 2019). A review of sensory-targeted cognitive training methods found consistent improvements in multiple areas of cognition (Donde, Mondino, et al., 2019), including WM (Hubacher et al., 2013; Lawlor-Savage & Goghari, 2014). If the early auditory signal is improved, this would benefit later auditory processing. However, benefits were short-lived and time-intensive. One theorized mechanism of sensory training is that it induces neural plasticity, providing a window to normalize sensory and cognitive functioning. Indeed, Donde et al. (2019) reviewed biomarkers of schizophrenia that normalized after sensory training. They highlighted that training imposes *no known side effects* and it may provide a more holistic approach to reducing schizophrenia symptomatology. Others note that ignoring sensory issues creates an informational “bottleneck” that hampers patients' ability to improve cognitive functioning (Genevsky et al., 2010). Training in processing speed showed greater improvements across cognitive abilities compared to narrow effects after WM training (Cassetta et al., 2019). Improving low-level sensory processing benefits upstream processing, but more work is essential to determine which approach best alleviates symptoms. Focusing on schizotypy may allow for some of the mechanisms to be disentangled through more detailed investigation without clinical complications such as medications and contact time in the clinic.

Other treatments, including noninvasive stimulation approaches (e.g., transcranial direct current stimulation: tDCS; transcranial magnetic stimulation: TMS), report improvements in sensory and cognitive markers, and reduce symptoms (reviewed in (Berryhill & Martin, 2018). For example, anodal tDCS improved pitch MMN amplitude and 2-back WM performance in people with schizophrenia (Impey et al., 2017). A separate study reported improved auditory P50 ERP response and reduced auditory hallucinations after anodal tDCS (Kim et al., 2018). In treatment resistant individuals with schizophrenia, a TMS protocol (intermittent theta burst stimulation) targeting left PFC improved visual WM ability and reduced symptoms (Wang et al., 2022). However, a recent meta-analysis evaluating findings from TMS and tDCS approaches reports no collective benefit across 22 studies (Sloan et al., 2021). Early-stage neurostimulation findings show promise, in terms of behavior and normalized biomarkers. However, small sample sizes, brief protocols, and a variable clinical population contribute to heterogeneous outcomes and a need for optimization. It is also unclear how stimulation methods would impact a less symptomatic population, particularly if the biomarkers in schizotypy are less pronounced than they are in diagnosed individuals.

## Can Biomarkers be Used to Predict Schizophrenia Onset?

When assessing biomarkers of symptom severity, there is a burgeoning question as to whether some of the biomarkers already discussed here can be used to predict the onset of schizophrenia. This assumes that biomarkers related to symptom severity worsen in some way immediately before the onset of schizophrenia. However, even if this is not the case, it could be that a different combination of symptom-related biomarkers is helpful in predicting conversion.

Ideally a biomarker could be used to identify single individuals as approaching schizophrenia with enough confidence to justify beginning treatment before an official diagnosis. Although biomarkers need to have a high degree of predictive accuracy, there is also the issue of specificity. Individuals with bipolar disorder may experience psychosis, but unlike schizophrenia, it is not necessary for diagnosis. This overlap in psychosis means that a successful biomarker needs to be sufficiently *sensitive* to predict onset of psychosis *and* be *specific* to SSD (Insel et al., 2011).

As previously discussed, biomarkers of sensory memory, particularly the auditory MMN, have been singled out for being reliably smaller in chronic schizophrenia compared to neurotypical individuals (Naatanen et al., 2015; Umbricht & Krljes, 2005). A recent large-sample study found typical MMN in unaffected siblings of people with schizophrenia, leading them to propose that reduced MMN may be closely linked with the onset of psychosis (Donaldson et al., 2021). However, efforts to date have found that the auditory MMN is only slightly smaller in early course schizophrenia (Lho et al., 2020; Salisbury et al., 2007) and is not reliably reduced in individuals at their first-episode of schizophrenia (Erickson et al., 2016; Haigh, Coffman, et al., 2017), or in first-degree unaffected relatives (Magno et al., 2008). Furthermore, longitudinal studies following individuals who are at-risk of developing schizophrenia have yet to detect robust reductions in auditory MMN that provide any confidence of impending conversion to psychosis (Atkinson et al., 2017; Erickson et al., 2016; Koshiyama et al., 2017).

However, the emphasis on a *single* biomarker to predict psychosis onset may be the issue. In the at-risk population, two recent meta-analyses point to WM performance as a predictor of developing psychosis. For instance, re-evaluating findings relying on the MATRICS Consensus Cognitive Battery identified the factors of overall cognition, attention, processing speed, and WM as *significantly* predictive of developing psychosis (Zheng et al., 2018). This is broadly consistent with an earlier meta-analysis that selected WM and visual learning to identify individuals likely to develop psychosis (De Herdt et al., 2013). In individuals with schizotypy, meta-analysis again pointed to a general deficit across verbal and visuospatial WM as a significant predictor of developing psychosis (Siddi et al., 2017). Multifactorial analyses of multiple biomarkers have improved specificity of predicting who is at-risk of developing schizophrenia later in life (Seidman, Shapiro, et al., 2016) and with improvements in machine learning, more sophisticated tools are available to detect even subtle patterns of functional abnormalities in SSD (Tai et al., 2019).

# Concluding Thoughts

To address the public health challenge associated with schizophrenia, we should accelerate our understanding of SSD through identification of useful biomarkers of SSD. This includes conducting research in overlooked sensory modalities (e.g., chemical senses, somatosensation). We need to be more cognizant of gaps in our understanding of sensory processing and their contributions to later cognitive processing. One way to do this is to measure responses from different parts of the sensory processing stream simultaneously. Advancements in technology, reduction in the price of certain electrophysiology equipment, and improvements in methodology have made this approach a reality without having to build equipment from scratch.

SM and WM deficits in those with high schizotypy and individuals with diagnosed schizophrenia, highlight that schizophrenia really is a spectrum of symptoms. Focusing on schizotypy requires a larger sample size is needed to detect the smaller effects (compared to schizophrenia) but offers a straightforward method for assessing relationships between symptom severity and functioning along the schizophrenia spectrum. Due to the lack of studies focusing on multisensory and multidomain functioning in schizotypy, we are forced to assume that the benefits of multivariate biomarkers will be as helpful in characterizing those with high schizotypy as it should be in the diagnosed population.

The deficits in SM and WM along the schizophrenia spectrum offer insight into how these previously disparate abnormalities interact and accrue downstream effects. Furthermore, they highlight the value of investigating the use of early-stage sensory interventions for treatment. There is already some evidence of *training* in SM or WM to alleviate deficits. The effectiveness of sensory training on symptoms has implications for other conditions associated with sensory abnormalities. Assessing biomarkers along the sensory pathway and treating early sensory symptoms is being used in autism (Dinstein et al., 2015; Haigh, 2018; Whitaker et al., 2016)) and in bipolar depression (for a review, see (Sit & Haigh, 2019)). However, it will be important to demonstrate whether these mechanisms are (1) causal, and (2) sensory modality specific.

Comparing SM and WM has the added benefit of identifying biomarkers and neural mechanisms that are unique to schizophrenia symptomatology. For example, schizophrenia is indistinguishable from autism on neuropsychological tests, including WM (Eack et al., 2013). Whereas the schizophrenia population differs from autism in their sensory responses, namely that individuals with schizophrenia typically under-respond whereas individuals with autism tend to over-respond (Haigh et al., 2016; Haigh et al., 2022). Identifying multiple biomarkers associated with schizophrenia and SSD more broadly can help with diagnostic specificity with greater accuracy than single markers.

To improve outcomes associated with schizophrenia, new approaches are needed. We advocate for collecting multiple biomarkers from available, subclinical populations that can collectively monitor symptoms and reveal the mechanisms underlying the symptoms. Alternatively, they may identify treatment successes. With a more complete mechanistic understanding of schizophrenia, such a suite of biomarkers could identify appropriate courses of treatment that will improve long-term outcomes.

# Acknowledgments

This material is based upon work supported by NIMH R15MH122935 to SH, MB and MD. The content is solely the responsibility of the authors and does not represent the official views of any funding agency.

**Conflicts of interest statement:** the authors have no conflicts of interest to declare.

**Author contributions:** SMH and MEB contributed equally to the manuscript. AKG assisted with the literature search and summary. MD contributed to the writing of the manuscript. All authors approved the final submission.

**Graphical Abstract:**

**Graphical text:** Degraded processing of the incoming sensory signal can impact the ability to manipulate sensory information in working memory and, similarly, poor working memory can degrade the processing on incoming sensory signal. This interaction is being explored in individuals with schizophrenia and along the schizophrenia spectrum.

**Figure Legends:**

***Figure 1.*** *Model of the relationship between poor sensory memory and poor working memory: low quality incoming signal (sensory memory) can impact the ability to manipulate sensory information in working memory. Similarly, poor ability to manipulate information can degrade the processing of the incoming signal.*

***Figure 2.*** *Schizophrenia-related symptoms across the spectrum, organized in order of symptom severity.*

***Figure 3.*** *Illustration of differences in mismatch negativity where individuals with schizophrenia show the smallest MMN (grey), followed by high schizotypy / subclinical individuals (grey, dark line), and low schizotypy showing the largest MMN (black).*

**Table Legend:**

***Table 1.*** *Number of papers identified when search terms were entered into PubMed\*. Auditory and visual modalities were added to search terms for schizophrenia and schizotypy separately. The percent of total search terms that focused on auditory or visual processing are shown. Owing to the mismatch negativity dominating the sensory memory literature, mismatch negativity (MMN) was searched for separately. Highlighted cells show the modality dominance: over twice the number of papers are published in one modality over the other.*

# 

# 

# 

# References

Atkinson, R. J., Fulham, W. R., Michie, P. T., Ward, P. B., Todd, J., Stain, H., Langdon, R., Thienel, R., Paulik, G., Cooper, G., Min, T. C., & Schall, U. (2017). Electrophysiological, cognitive and clinical profiles of at-risk mental state: The longitudinal Minds in Transition (MinT) study. *PLoS One*, *12*(2), e0171657. <https://doi.org/10.1371/journal.pone.0171657>

Baldeweg, T., Wong, D., & Stephan, K. E. (2006). Nicotinic modulation of human auditory sensory memory: Evidence from mismatch negativity potentials. *Int J Psychophysiol*, *59*(1), 49-58. <https://doi.org/10.1016/j.ijpsycho.2005.07.014>

Barch, D. M. (2005). The cognitive neuroscience of schizophrenia. *Annu Rev Clin Psychol*, *1*, 321-353. <https://doi.org/10.1146/annurev.clinpsy.1.102803.143959>

Barch, D. M., & Ceaser, A. (2012). Cognition in schizophrenia: core psychological and neural mechanisms. *Trends Cogn Sci*, *16*(1), 27-34. <https://doi.org/10.1016/j.tics.2011.11.015>

Basar-Eroglu, C., Brand, A., Hildebrandt, H., Karolina Kedzior, K., Mathes, B., & Schmiedt, C. (2007). Working memory related gamma oscillations in schizophrenia patients. *Int J Psychophysiol*, *64*(1), 39-45. <https://doi.org/10.1016/j.ijpsycho.2006.07.007>

Bates, T. C. (2005). The panmodal sensory imprecision hypothesis of schizophrenia: reduced auditory precision in schizotypy. *Personality and Individual Differences*, *38*(2), 437-449. <https://doi.org/10.1016/j.paid.2004.04.021>

Bernardin, F., Schwan, R., Lalanne, L., Ligier, F., Angioi-Duprez, K., Schwitzer, T., & Laprevote, V. (2017). The role of the retina in visual hallucinations: A review of the literature and implications for psychosis. *Neuropsychologia*, *99*, 128-138. <https://doi.org/10.1016/j.neuropsychologia.2017.03.002>

Berryhill, M. E., & Martin, D. (2018). Cognitive Effects of Transcranial Direct Current Stimulation in Healthy and Clinical Populations: An Overview. *J ECT*, *34*(3), e25-e35. <https://doi.org/10.1097/YCT.0000000000000534>

Biagianti, B., Fisher, M., Neilands, T. B., Loewy, R., & Vinogradov, S. (2016). Engagement with the auditory processing system during targeted auditory cognitive training mediates changes in cognitive outcomes in individuals with schizophrenia. *Neuropsychology*, *30*(8), 998-1008. <https://doi.org/10.1037/neu0000311>

Briend, F., Armstrong, W. P., Kraguljac, N. V., Keilhloz, S. D., & Lahti, A. C. (2020). Aberrant static and dynamic functional patterns of frontoparietal control network in antipsychotic-naive first-episode psychosis subjects. *Hum Brain Mapp*, *41*(11), 2999-3008. <https://doi.org/10.1002/hbm.24992>

Broyd, S. J., Michie, P. T., Bruggemann, J., van Hell, H. H., Greenwood, L. M., Croft, R. J., Todd, J., Lenroot, R., & Solowij, N. (2016). Schizotypy and auditory mismatch negativity in a non-clinical sample of young adults. *Psychiatry Res Neuroimaging*, *254*, 83-91. <https://doi.org/10.1016/j.pscychresns.2016.06.011>

Cannon, T. D., Yu, C., Addington, J., Bearden, C. E., Cadenhead, K. S., Cornblatt, B. A., Heinssen, R., Jeffries, C. D., Mathalon, D. H., McGlashan, T. H., Perkins, D. O., Seidman, L. J., Tsuang, M. T., Walker, E. F., Woods, S. W., & Kattan, M. W. (2016). An Individualized Risk Calculator for Research in Prodromal Psychosis. *Am J Psychiatry*, *173*(10), 980-988. <https://doi.org/10.1176/appi.ajp.2016.15070890>

Cassetta, B. D., Tomfohr-Madsen, L. M., & Goghari, V. M. (2019). A randomized controlled trial of working memory and processing speed training in schizophrenia. *Psychol Med*, *49*(12), 2009-2019. <https://doi.org/10.1017/S0033291718002775>

Catts, S. V., Shelley, A. M., Ward, P. B., Liebert, B., McConaghy, N., Andrews, S., & Michie, P. T. (1995). Brain potential evidence for an auditory sensory memory deficit in schizophrenia. *Am J Psychiatry*, *152*(2), 213-219. <https://doi.org/10.1176/ajp.152.2.213>

Cavanaugh, J., Berman, R. A., Joiner, W. M., & Wurtz, R. H. (2016). Saccadic Corollary Discharge Underlies Stable Visual Perception. *J Neurosci*, *36*(1), 31-42. <https://doi.org/10.1523/JNEUROSCI.2054-15.2016>

Cho, R. Y., Konecky, R. O., & Carter, C. S. (2006). Impairments in frontal cortical gamma synchrony and cognitive control in schizophrenia. *Proc Natl Acad Sci U S A*, *103*(52), 19878-19883. <https://doi.org/10.1073/pnas.0609440103>

Claridge, G., & Beech, T. (1995). Fully and quasi-dimensional constructions of schizotypy. *Schizotypal Personal.*, *29*, 192-216.

Coffman, B. A., Haigh, S. M., Murphy, T. K., Leiter-Mcbeth, J., & Salisbury, D. F. (2018). Reduced auditory segmentation potentials in first-episode schizophrenia. *Schizophr Res*, *195*, 421-427. <https://doi.org/10.1016/j.schres.2017.10.011>

Coffman, B. A., Haigh, S. M., Murphy, T. K., & Salisbury, D. F. (2017). Impairment in Mismatch Negativity but not Repetition Suppression in Schizophrenia. *Brain Topogr*, *30*(4), 521-530. <https://doi.org/10.1007/s10548-017-0571-1>

Creutzfeldt, O., Ojemann, G., & Lettich, E. (1989). Neuronal activity in the human lateral temporal lobe. II. Responses to the subjects own voice. *Exp Brain Res*, *77*(3), 476-489. <https://doi.org/10.1007/BF00249601>

Csukly, G., Stefanics, G., Komlosi, S., Czigler, I., & Czobor, P. (2013). Emotion-related visual mismatch responses in schizophrenia: impairments and correlations with emotion recognition. *PLoS One*, *8*(10), e75444. <https://doi.org/10.1371/journal.pone.0075444>

Dale, C. L., Brown, E. G., Fisher, M., Herman, A. B., Dowling, A. F., Hinkley, L. B., Subramaniam, K., Nagarajan, S. S., & Vinogradov, S. (2016). Auditory Cortical Plasticity Drives Training-Induced Cognitive Changes in Schizophrenia. *Schizophr Bull*, *42*(1), 220-228. <https://doi.org/10.1093/schbul/sbv087>

Dale, C. L., Findlay, A. M., Adcock, R. A., Vertinski, M., Fisher, M., Genevsky, A., Aldebot, S., Subramaniam, K., Luks, T. L., Simpson, G. V., Nagarajan, S. S., & Vinogradov, S. (2010). Timing is everything: neural response dynamics during syllable processing and its relation to higher-order cognition in schizophrenia and healthy comparison subjects. *Int J Psychophysiol*, *75*(2), 183-193. <https://doi.org/10.1016/j.ijpsycho.2009.10.009>

De Herdt, A., Wampers, M., Vancampfort, D., De Hert, M., Vanhees, L., Demunter, H., Van Bouwel, L., Brunner, E., & Probst, M. (2013). Neurocognition in clinical high risk young adults who did or did not convert to a first schizophrenic psychosis: a meta-analysis. *Schizophr Res*, *149*(1-3), 48-55. <https://doi.org/10.1016/j.schres.2013.06.017>

Devrim-Ucok, M., Keskin-Ergen, Y., & Ucok, A. (2016). Lack of progressive reduction in P3 amplitude after the first-episode of schizophrenia: A 6-year follow-up study. *Psychiatry Res*, *243*, 303-311. <https://doi.org/10.1016/j.psychres.2016.02.065>

Ding, Y., Ou, Y., Su, Q., Pan, P., Shan, X., Chen, J., Liu, F., Zhang, Z., Zhao, J., & Guo, W. (2019). Enhanced Global-Brain Functional Connectivity in the Left Superior Frontal Gyrus as a Possible Endophenotype for Schizophrenia. *Front Neurosci*, *13*, 145. <https://doi.org/10.3389/fnins.2019.00145>

Dinstein, I., Heeger, D. J., & Behrmann, M. (2015). Neural variability: friend or foe? *Trends Cogn Sci*, *19*(6), 322-328. <https://doi.org/10.1016/j.tics.2015.04.005>

Donaldson, K. R., Larsen, E. M., Jonas, K., Tramazzo, S., Perlman, G., Foti, D., Mohanty, A., & Kotov, R. (2021). Mismatch negativity amplitude in first-degree relatives of individuals with psychotic disorders: Links with cognition and schizotypy. *Schizophr Res*, *238*, 161-169. <https://doi.org/10.1016/j.schres.2021.10.006>

Donde, C., Brunelin, J., & Haesebaert, F. (2020). Duration, pitch and intensity features reveal different magnitudes of tone-matching deficit in schizophrenia. *Schizophr Res*, *215*, 460-462. <https://doi.org/10.1016/j.schres.2019.10.003>

Donde, C., Martinez, A., Kantrowitz, J. T., Silipo, G., Dias, E. C., Patel, G. H., Sanchez-Pena, J., Corcoran, C. M., Medalia, A., Saperstein, A., Vail, B., & Javitt, D. C. (2019). Bimodal distribution of tone-matching deficits indicates discrete pathophysiological entities within the syndrome of schizophrenia. *Transl Psychiatry*, *9*(1), 221. <https://doi.org/10.1038/s41398-019-0557-8>

Donde, C., Mondino, M., Brunelin, J., & Haesebaert, F. (2019). Sensory-targeted cognitive training for schizophrenia. *Expert Rev Neurother*, *19*(3), 211-225. <https://doi.org/10.1080/14737175.2019.1581609>

Du, Y., He, X., Kochunov, P., Pearlson, G., Hong, L. E., van Erp, T. G. M., Belger, A., & Calhoun, V. D. (2022). A new multimodality fusion classification approach to explore the uniqueness of schizophrenia and autism spectrum disorder. *Hum Brain Mapp*. <https://doi.org/10.1002/hbm.25890>

Erickson, M. A., Ruffle, A., & Gold, J. M. (2016). A Meta-Analysis of Mismatch Negativity in Schizophrenia: From Clinical Risk to Disease Specificity and Progression. *Biol Psychiatry*, *79*(12), 980-987. <https://doi.org/10.1016/j.biopsych.2015.08.025>

Escera, C., & Malmierca, M. S. (2014). The auditory novelty system: an attempt to integrate human and animal research. *Psychophysiology*, *51*(2), 111-123. <https://doi.org/10.1111/psyp.12156>

Ettinger, U., Meyhofer, I., Steffens, M., Wagner, M., & Koutsouleris, N. (2014). Genetics, cognition, and neurobiology of schizotypal personality: a review of the overlap with schizophrenia. *Front Psychiatry*, *5*, 18. <https://doi.org/10.3389/fpsyt.2014.00018>

Farkas, K., Stefanics, G., Marosi, C., & Csukly, G. (2015). Elementary sensory deficits in schizophrenia indexed by impaired visual mismatch negativity. *Schizophr Res*, *166*(1-3), 164-170. <https://doi.org/10.1016/j.schres.2015.05.011>

Feinberg, I. (1978). Efference copy and corollary discharge: implications for thinking and its disorders. *Schizophr Bull*, *4*(4), 636-640. <https://doi.org/10.1093/schbul/4.4.636>

Fernandes, T. M. P., Silverstein, S. M., Butler, P. D., Keri, S., Santos, L. G., Nogueira, R. L., & Santos, N. A. (2019). Color vision impairments in schizophrenia and the role of antipsychotic medication type. *Schizophr Res*, *204*, 162-170. <https://doi.org/10.1016/j.schres.2018.09.002>

Fisher, D. J., Rudolph, E. D., Ells, E. M. L., Knott, V. J., Labelle, A., & Tibbo, P. G. (2019). Mismatch negativity-indexed auditory change detection of speech sounds in early and chronic schizophrenia. *Psychiatry Res Neuroimaging*, *287*, 1-9. <https://doi.org/10.1016/j.pscychresns.2019.03.010>

Forbes, N. F., Carrick, L. A., McIntosh, A. M., & Lawrie, S. M. (2009). Working memory in schizophrenia: a meta-analysis. *Psychol Med*, *39*(6), 889-905. <https://doi.org/10.1017/S0033291708004558>

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

Ford, T. C., Hugrass, L. E., & Jack, B. N. (2022). The Relationship Between Affective Visual Mismatch Negativity and Interpersonal Difficulties Across Autism and Schizotypal Traits. *Front Hum Neurosci*, *16*, 846961. <https://doi.org/10.3389/fnhum.2022.846961>

Friedman, T., Sehatpour, P., Dias, E., Perrin, M., & Javitt, D. C. (2012). Differential relationships of mismatch negativity and visual p1 deficits to premorbid characteristics and functional outcome in schizophrenia. *Biol Psychiatry*, *71*(6), 521-529. <https://doi.org/10.1016/j.biopsych.2011.10.037>

Fryer, S. L., Roach, B. J., Ford, J. M., Turner, J. A., van Erp, T. G., Voyvodic, J., Preda, A., Belger, A., Bustillo, J., O'Leary, D., Mueller, B. A., Lim, K. O., McEwen, S. C., Calhoun, V. D., Diaz, M., Glover, G., Greve, D., Wible, C. G., Vaidya, J., . . . Mathalon, D. H. (2015). Relating Intrinsic Low-Frequency BOLD Cortical Oscillations to Cognition in Schizophrenia. *Neuropsychopharmacology*, *40*(12), 2705-2714. <https://doi.org/10.1038/npp.2015.119>

Funahashi, S. (2017). Working Memory in the Prefrontal Cortex. *Brain Sci*, *7*(5). <https://doi.org/10.3390/brainsci7050049>

Genevsky, A., Garrett, C. T., Alexander, P. P., & Vinogradov, S. (2010). Cognitive training in schizophrenia: a neuroscience-based approach. *Dialogues Clin Neurosci*, *12*(3), 416-421. <https://www.ncbi.nlm.nih.gov/pubmed/20954435>

Geng, H., Xu, P., Sommer, I. E., Luo, Y. J., Aleman, A., & Curcic-Blake, B. (2020). Abnormal dynamic resting-state brain network organization in auditory verbal hallucination. *Brain Struct Funct*, *225*(8), 2315-2330. <https://doi.org/10.1007/s00429-020-02119-1>

Gisselgard, J., Anda, L. G., Bronnick, K., Langeveld, J., Ten Velden Hegelstad, W., Joa, I., Johannessen, J. O., & Larsen, T. K. (2014). Verbal working memory deficits predict levels of auditory hallucination in first-episode psychosis. *Schizophr Res*, *153*(1-3), 38-41. <https://doi.org/10.1016/j.schres.2013.12.018>

Gonzalez-Blanch, C., Alvarez-Jimenez, M., Rodriguez-Sanchez, J. M., Perez-Iglesias, R., Vazquez-Barquero, J. L., & Crespo-Facorro, B. (2006). Cognitive functioning in the early course of first-episode schizophrenia spectrum disorders: timing and patterns. *Eur Arch Psychiatry Clin Neurosci*, *256*(6), 364-371. <https://doi.org/10.1007/s00406-006-0646-6>

Grant, P., Green, M. J., & Mason, O. J. (2018). Models of Schizotypy: The Importance of Conceptual Clarity. *Schizophr Bull*, *44*(suppl\_2), S556-S563. <https://doi.org/10.1093/schbul/sby012>

Haenschel, C., Bittner, R. A., Waltz, J., Haertling, F., Wibral, M., Singer, W., Linden, D. E., & Rodriguez, E. (2009). Cortical oscillatory activity is critical for working memory as revealed by deficits in early-onset schizophrenia. *J Neurosci*, *29*(30), 9481-9489. <https://doi.org/10.1523/JNEUROSCI.1428-09.2009>

Haenschel, C., & Linden, D. (2011). Exploring intermediate phenotypes with EEG: working memory dysfunction in schizophrenia. *Behav Brain Res*, *216*(2), 481-495. <https://doi.org/10.1016/j.bbr.2010.08.045>

Hahn, B., Kappenman, E. S., Robinson, B. M., Fuller, R. L., Luck, S. J., & Gold, J. M. (2011). Iconic decay in schizophrenia. *Schizophr Bull*, *37*(5), 950-957. <https://doi.org/10.1093/schbul/sbp164>

Haigh, S. M. (2018). Variable sensory perception in autism. *Eur J Neurosci*, *47*(6), 602-609. <https://doi.org/10.1111/ejn.13601>

Haigh, S. M., Coffman, B. A., Murphy, T. K., Butera, C. D., Leiter-McBeth, J. R., & Salisbury, D. F. (2019). Reduced late mismatch negativity and auditory sustained potential to rule-based patterns in schizophrenia. *Eur J Neurosci*, *49*(2), 275-289. <https://doi.org/10.1111/ejn.14274>

Haigh, S. M., Coffman, B. A., Murphy, T. K., Butera, C. D., & Salisbury, D. F. (2016). Abnormal auditory pattern perception in schizophrenia. *Schizophr Res*, *176*(2-3), 473-479. <https://doi.org/10.1016/j.schres.2016.07.007>

Haigh, S. M., Coffman, B. A., & Salisbury, D. F. (2017). Mismatch Negativity in First-Episode Schizophrenia: A Meta-Analysis. *Clin EEG Neurosci*, *48*(1), 3-10. <https://doi.org/10.1177/1550059416645980>

Haigh, S. M., Matteis, M., Coffman, B. A., Murphy, T. K., Butera, C. D., Ward, K. L., Leiter-McBeth, J. R., & Salisbury, D. F. (2017). Mismatch negativity to pitch pattern deviants in schizophrenia. *Eur J Neurosci*, *46*(6), 2229-2239. <https://doi.org/10.1111/ejn.13660>

Haigh, S. M., Van Key, L., Brosseau, P., Eack, S. M., Leitman, D. I., Salisbury, D.F., & Behrmann, M. (2022). Assessing Trial-to-Trial Variability in Auditory ERPs in Autism and Schizophrenia. *J Autism Dev Disord.* <https://doi.org/10.1007/s10803-022-05771-0>

Hamilton, H. K., Williams, T. J., Ventura, J., Jasperse, L. J., Owens, E. M., Miller, G. A., Subotnik, K. L., Nuechterlein, K. H., & Yee, C. M. (2018). Clinical and Cognitive Significance of Auditory Sensory Processing Deficits in Schizophrenia. *Am J Psychiatry*, *175*(3), 275-283. <https://doi.org/10.1176/appi.ajp.2017.16111203>

Harper, L., Spencer, E., Davidson, C., & Hutchinson, C. V. (2020). Selectively reduced contrast sensitivity in high schizotypy. *Exp Brain Res*, *238*(1), 51-62. <https://doi.org/10.1007/s00221-019-05695-9>

Hashimoto, R., Lee, K., Preus, A., McCarley, R. W., & Wible, C. G. (2010). An fMRI study of functional abnormalities in the verbal working memory system and the relationship to clinical symptoms in chronic schizophrenia. *Cereb Cortex*, *20*(1), 46-60. <https://doi.org/10.1093/cercor/bhp079>

Herman, A. B., Brown, E. G., Dale, C. L., Hinkley, L. B., Subramaniam, K., Houde, J. F., Fisher, M., Vinogradov, S., & Nagarajan, S. S. (2020). The Visual Word Form Area compensates for auditory working memory dysfunction in schizophrenia. *Sci Rep*, *10*(1), 8881. <https://doi.org/10.1038/s41598-020-63962-0>

Higuchi, Y., Sumiyoshi, T., Seo, T., Miyanishi, T., Kawasaki, Y., & Suzuki, M. (2013). Mismatch negativity and cognitive performance for the prediction of psychosis in subjects with at-risk mental state. *PLoS One*, *8*(1), e54080. <https://doi.org/10.1371/journal.pone.0054080>

Holliday, W. B., Gurnsey, K., Sweet, R. A., & Teichert, T. (2018). A putative electrophysiological biomarker of auditory sensory memory encoding is sensitive to pharmacological alterations of excitatory/inhibitory balance in male macaque monkeys. *J Psychiatry Neurosci*, *43*(3), 182-193. <https://www.ncbi.nlm.nih.gov/pubmed/29688874>

Hoy, K. E., Coyle, H., Gainsford, K., Hill, A. T., Bailey, N. W., & Fitzgerald, P. B. (2021). Investigating neurophysiological markers of impaired cognition in schizophrenia. *Schizophr Res*, *233*, 34-43. <https://doi.org/10.1016/j.schres.2021.06.025>

Hsieh, M. H., Lin, Y. T., Chien, Y. L., Hwang, T. J., Hwu, H. G., Liu, C. M., & Liu, C. C. (2019). Auditory Event-Related Potentials in Antipsychotic-Free Subjects With Ultra-High-Risk State and First-Episode Psychosis. *Front Psychiatry*, *10*, 223. <https://doi.org/10.3389/fpsyt.2019.00223>

Hubacher, M., Weiland, M., Calabrese, P., Stoppe, G., Stocklin, M., Fischer-Barnicol, D., Opwis, K., & Penner, I. K. (2013). Working memory training in patients with chronic schizophrenia: a pilot study. *Psychiatry J*, *2013*, 154867. <https://doi.org/10.1155/2013/154867>

Impey, D., Baddeley, A., Nelson, R., Labelle, A., & Knott, V. (2017). Effects of transcranial direct current stimulation on the auditory mismatch negativity response and working memory performance in schizophrenia: a pilot study. *J Neural Transm (Vienna)*, *124*(11), 1489-1501. <https://doi.org/10.1007/s00702-017-1783-y>

Insel, T. R., Morris, S. E., & Heinssen, R. K. (2011). Standardization, integration, and sharing-leveraging research investments. *Biol Psychiatry*, *70*(1), 5-6. <https://doi.org/10.1016/j.biopsych.2011.05.004>

Javitt, D. C. (2009). When doors of perception close: bottom-up models of disrupted cognition in schizophrenia. *Annu Rev Clin Psychol*, *5*, 249-275. <https://doi.org/10.1146/annurev.clinpsy.032408.153502>

Javitt, D. C., Doneshka, P., Grochowski, S., & Ritter, W. (1995). Impaired mismatch negativity generation reflects widespread dysfunction of working memory in schizophrenia. *Arch Gen Psychiatry*, *52*(7), 550-558. <https://doi.org/10.1001/archpsyc.1995.03950190032005>

Javitt, D. C., Shelley, A. M., Silipo, G., & Lieberman, J. A. (2000). Deficits in auditory and visual context-dependent processing in schizophrenia: defining the pattern. *Arch Gen Psychiatry*, *57*(12), 1131-1137. <https://doi.org/10.1001/archpsyc.57.12.1131>

Javitt, D. C., & Sweet, R. A. (2015). Auditory dysfunction in schizophrenia: integrating clinical and basic features. *Nat Rev Neurosci*, *16*(9), 535-550. <https://doi.org/10.1038/nrn4002>

Jenkins, L. M., Bodapati, A. S., Sharma, R. P., & Rosen, C. (2018). Working memory predicts presence of auditory verbal hallucinations in schizophrenia and bipolar disorder with psychosis. *J Clin Exp Neuropsychol*, *40*(1), 84-94. <https://doi.org/10.1080/13803395.2017.1321106>

Kalmady, S. V., Paul, A. K., Greiner, R., Agrawal, R., Amaresha, A. C., Shivakumar, V., Narayanaswamy, J. C., Greenshaw, A. J., Dursun, S. M., & Venkatasubramanian, G. (2020). Extending schizophrenia diagnostic model to predict schizotypy in first-degree relatives. *NPJ Schizophr*, *6*(1), 30. <https://doi.org/10.1038/s41537-020-00119-y>

Kantrowitz, J. T., Epstein, M. L., Lee, M., Lehrfeld, N., Nolan, K. A., Shope, C., Petkova, E., Silipo, G., & Javitt, D. C. (2018). Improvement in mismatch negativity generation during d-serine treatment in schizophrenia: Correlation with symptoms. *Schizophr Res*, *191*, 70-79. <https://doi.org/10.1016/j.schres.2017.02.027>

Keane, B. P., Cruz, L. N., Paterno, D., & Silverstein, S. M. (2018). Self-Reported Visual Perceptual Abnormalities Are Strongly Associated with Core Clinical Features in Psychotic Disorders. *Front Psychiatry*, *9*, 69. <https://doi.org/10.3389/fpsyt.2018.00069>

Keane, B. P., Erlikhman, G., Kastner, S., Paterno, D., & Silverstein, S. M. (2014). Multiple forms of contour grouping deficits in schizophrenia: what is the role of spatial frequency? *Neuropsychologia*, *65*, 221-233. <https://doi.org/10.1016/j.neuropsychologia.2014.10.031>

Keane, B. P., Peng, Y., Demmin, D., Silverstein, S. M., & Lu, H. (2018). Intact perception of coherent motion, dynamic rigid form, and biological motion in chronic schizophrenia. *Psychiatry Res*, *268*, 53-59. <https://doi.org/10.1016/j.psychres.2018.06.052>

Kent, A. R., Fox, A. M., Michie, P. T., & Jablensky, A. V. (2004). Differential impairment of working memory performance in first-degree relatives of individuals with schizophrenia. *Acta Neuropsychiatr*, *16*(3), 149-153. <https://doi.org/10.1111/j.0924-2708.2004.00070.x>

Kerns, J. G., & Becker, T. M. (2008). Communication disturbances, working memory, and emotion in people with elevated disorganized schizotypy. *Schizophr Res*, *100*(1-3), 172-180. <https://doi.org/10.1016/j.schres.2007.11.005>

Kim, J. S., Kwon, Y. J., Lee, H. Y., Lee, H. S., Kim, S., & Shim, S. H. (2020). Mismatch Negativity Indices as a Prognostic Factor for Remission in Schizophrenia. *Clin Psychopharmacol Neurosci*, *18*(1), 127-135. <https://doi.org/10.9758/cpn.2020.18.1.127>

Kim, M., Yoon, Y. B., Lee, T. H., Lee, T. Y., & Kwon, J. S. (2018). The effect of tDCS on auditory hallucination and P50 sensory gating in patients with schizophrenia: A pilot study. *Schizophr Res*, *192*, 469-470. <https://doi.org/10.1016/j.schres.2017.04.023>

Kircher, T. T., Rapp, A., Grodd, W., Buchkremer, G., Weiskopf, N., Lutzenberger, W., Ackermann, H., & Mathiak, K. (2004). Mismatch negativity responses in schizophrenia: a combined fMRI and whole-head MEG study. *Am J Psychiatry*, *161*(2), 294-304. <https://doi.org/10.1176/appi.ajp.161.2.294>

Kirihara, K., Tada, M., Koshiyama, D., Fujioka, M., Usui, K., Araki, T., & Kasai, K. (2020). A Predictive Coding Perspective on Mismatch Negativity Impairment in Schizophrenia. *Front Psychiatry*, *11*, 660. <https://doi.org/10.3389/fpsyt.2020.00660>

Knight, R., Sherer, M., Putchat, C., & Carter, G. (1978). A picture integration task for measuring iconic memory in schizophrenics. *J Abnorm Psychol*, *87*(3), 314-321. <https://www.ncbi.nlm.nih.gov/pubmed/681602>

Knott, V., Impey, D., Philippe, T., Smith, D., Choueiry, J., de la Salle, S., & Dort, H. (2014). Modulation of auditory deviance detection by acute nicotine is baseline and deviant dependent in healthy nonsmokers: a mismatch negativity study. *Hum Psychopharmacol*, *29*(5), 446-458. <https://doi.org/10.1002/hup.2418>

Kochunov, P., Fan, F., Ryan, M. C., Hatch, K. S., Tan, S., Jahanshad, N., Thompson, P. M., van Erp, T. G. M., Turner, J. A., Chen, S., Du, X., Adhikari, B., Bruce, H., Hare, S., Goldwaser, E., Kvarta, M., Huang, J., Tong, J., Cui, Y., . . . Hong, L. E. (2022). Translating ENIGMA schizophrenia findings using the regional vulnerability index: Association with cognition, symptoms, and disease trajectory. *Hum Brain Mapp*, *43*(1), 566-575. <https://doi.org/10.1002/hbm.25045>

Koshiyama, D., Kirihara, K., Tada, M., Nagai, T., Koike, S., Suga, M., Araki, T., & Kasai, K. (2017). Duration and frequency mismatch negativity shows no progressive reduction in early stages of psychosis. *Schizophr Res*, *190*, 32-38. <https://doi.org/10.1016/j.schres.2017.03.015>

Koychev, I., El-Deredy, W., Haenschel, C., & Deakin, J. F. (2010). Visual information processing deficits as biomarkers of vulnerability to schizophrenia: an event-related potential study in schizotypy. *Neuropsychologia*, *48*(7), 2205-2214. <https://doi.org/10.1016/j.neuropsychologia.2010.04.014>

Lawlor-Savage, L., & Goghari, V. M. (2014). Working memory training in schizophrenia and healthy populations. *Behav Sci (Basel)*, *4*(3), 301-319. <https://doi.org/10.3390/bs4030301>

Le, L., Kaur, R., Meiser, B., & Green, M. J. (2020). Risk of schizophrenia in relatives of individuals affected by schizophrenia: A meta-analysis. *Psychiatry Res*, *286*, 112852. <https://doi.org/10.1016/j.psychres.2020.112852>

Leicht, G., Andreou, C., Polomac, N., Lanig, C., Schottle, D., Lambert, M., & Mulert, C. (2015). Reduced auditory evoked gamma band response and cognitive processing deficits in first episode schizophrenia. *World J Biol Psychiatry*, *16*(6), 387-397. <https://doi.org/10.3109/15622975.2015.1017605>

Leitman, D. I., Foxe, J. J., Butler, P. D., Saperstein, A., Revheim, N., & Javitt, D. C. (2005). Sensory contributions to impaired prosodic processing in schizophrenia. *Biol Psychiatry*, *58*(1), 56-61. <https://doi.org/10.1016/j.biopsych.2005.02.034>

Lenzenweger, M. F., & Gold, J. M. (2000). Auditory working memory and verbal recall memory in schizotypy. *Schizophr Res*, *42*(2), 101-110. <https://doi.org/10.1016/s0920-9964(99)00121-8>

Lett, T. A., Voineskos, A. N., Kennedy, J. L., Levine, B., & Daskalakis, Z. J. (2014). Treating working memory deficits in schizophrenia: a review of the neurobiology. *Biol Psychiatry*, *75*(5), 361-370. <https://doi.org/10.1016/j.biopsych.2013.07.026>

Levy, D. L., Sereno, A. B., Gooding, D. C., & O'Driscoll, G. A. (2010). Eye tracking dysfunction in schizophrenia: characterization and pathophysiology. *Curr Top Behav Neurosci*, *4*, 311-347. <https://doi.org/10.1007/7854_2010_60>

Lho, S. K., Kim, M., Park, J., Hwang, W. J., Moon, S. Y., Oh, S., & Kwon, J. S. (2020). Progressive Impairment of Mismatch Negativity Is Reflective of Underlying Pathophysiological Changes in Patients With First-Episode Psychosis. *Front Psychiatry*, *11*, 587. <https://doi.org/10.3389/fpsyt.2020.00587>

Lieberman, J. A., Small, S. A., & Girgis, R. R. (2019). Early Detection and Preventive Intervention in Schizophrenia: From Fantasy to Reality. *Am J Psychiatry*, *176*(10), 794-810. <https://doi.org/10.1176/appi.ajp.2019.19080865>

Light, G. A., & Braff, D. L. (2005). Mismatch negativity deficits are associated with poor functioning in schizophrenia patients. *Arch Gen Psychiatry*, *62*(2), 127-136. <https://doi.org/10.1001/archpsyc.62.2.127>

Light, G. A., Hsu, J. L., Hsieh, M. H., Meyer-Gomes, K., Sprock, J., Swerdlow, N. R., & Braff, D. L. (2006). Gamma band oscillations reveal neural network cortical coherence dysfunction in schizophrenia patients. *Biol Psychiatry*, *60*(11), 1231-1240. <https://doi.org/10.1016/j.biopsych.2006.03.055>

Ludwig, S., Spitzer, B., Jacobs, A. M., Sekutowicz, M., Sterzer, P., & Blankenburg, F. (2016). Spectral EEG abnormalities during vibrotactile encoding and quantitative working memory processing in schizophrenia. *Neuroimage Clin*, *11*, 578-587. <https://doi.org/10.1016/j.nicl.2016.04.004>

Magno, E., Yeap, S., Thakore, J. H., Garavan, H., De Sanctis, P., & Foxe, J. J. (2008). Are auditory-evoked frequency and duration mismatch negativity deficits endophenotypic for schizophrenia? High-density electrical mapping in clinically unaffected first-degree relatives and first-episode and chronic schizophrenia. *Biol Psychiatry*, *64*(5), 385-391. <https://doi.org/10.1016/j.biopsych.2008.03.019>

March, L., Cienfuegos, A., Goldbloom, L., Ritter, W., Cowan, N., & Javitt, D. C. (1999). Normal time course of auditory recognition in schizophrenia, despite impaired precision of the auditory sensory ("echoic") memory code. *J Abnorm Psychol*, *108*(1), 69-75. <https://doi.org/10.1037//0021-843x.108.1.69>

Marsh, J. E., Vachon, F., & Sorqvist, P. (2017). Increased distractibility in schizotypy: Independent of individual differences in working memory capacity? *Q J Exp Psychol (Hove)*, *70*(3), 565-578. <https://doi.org/10.1080/17470218.2016.1172094>

May, P. J., & Tiitinen, H. (2010). Mismatch negativity (MMN), the deviance-elicited auditory deflection, explained. *Psychophysiology*, *47*(1), 66-122. <https://doi.org/10.1111/j.1469-8986.2009.00856.x>

May, P. J. C., & Tiitinen, H. (2007). The role of adaptation-based memory in auditory cortex. *International Congress Series*, *1300*, 53-56.

Mayer, J. S., Korinth, S., Peters, B., & Fiebach, C. J. (2020). An Electrophysiological Dissociation of Encoding vs. Maintenance Failures in Visual-Spatial Working Memory. *Front Psychol*, *11*, 522. <https://doi.org/10.3389/fpsyg.2020.00522>

McCleery, A., Lee, J., Joshi, A., Wynn, J. K., Hellemann, G. S., & Green, M. F. (2015). Meta-analysis of face processing event-related potentials in schizophrenia. *Biol Psychiatry*, *77*(2), 116-126. <https://doi.org/10.1016/j.biopsych.2014.04.015>

McCleery, A., Wynn, J. K., Mathalon, D. H., Roach, B. J., & Green, M. F. (2018). Hallucinations, neuroplasticity, and prediction errors in schizophrenia. *Scand J Psychol*, *59*(1), 41-48. <https://doi.org/10.1111/sjop.12413>

McLachlan, N. M., Phillips, D. S., Rossell, S. L., & Wilson, S. J. (2013). Auditory processing and hallucinations in schizophrenia. *Schizophr Res*, *150*(2-3), 380-385. <https://doi.org/10.1016/j.schres.2013.08.039>

Meiron, O., David, J., & Yaniv, A. (2022). Early Auditory Processing Predicts Efficient Working Memory Functioning in Schizophrenia. *Brain Sci*, *12*(2). <https://doi.org/10.3390/brainsci12020212>

Menon, V., Anagnoson, R. T., Mathalon, D. H., Glover, G. H., & Pfefferbaum, A. (2001). Functional neuroanatomy of auditory working memory in schizophrenia: relation to positive and negative symptoms. *Neuroimage*, *13*(3), 433-446. <https://doi.org/10.1006/nimg.2000.0699>

Millman, Z. B., Roemer, C., Vargas, T., Schiffman, J., Mittal, V. A., & Gold, J. M. (2022). Neuropsychological Performance Among Individuals at Clinical High-Risk for Psychosis vs Putatively Low-Risk Peers With Other Psychopathology: A Systematic Review and Meta-Analysis. *Schizophr Bull*. <https://doi.org/10.1093/schbul/sbac031>

Missonnier, P., Prevot, A., Herrmann, F. R., Ventura, J., Padee, A., & Merlo, M. C. G. (2020). Disruption of gamma-delta relationship related to working memory deficits in first-episode psychosis. *J Neural Transm (Vienna)*, *127*(1), 103-115. <https://doi.org/10.1007/s00702-019-02126-5>

Moberg, P. J., Arnold, S. E., Doty, R. L., Kohler, C., Kanes, S., Seigel, S., Gur, R. E., & Turetsky, B. I. (2003). Impairment of odor hedonics in men with schizophrenia. *Am J Psychiatry*, *160*(10), 1784-1789. <https://doi.org/10.1176/appi.ajp.160.10.1784>

Mohn-Haugen, C. R., Mohn, C., Laroi, F., Teigset, C. M., Oie, M. G., & Rund, B. R. (2022). A systematic review of premorbid cognitive functioning and its timing of onset in schizophrenia spectrum disorders. *Schizophr Res Cogn*, *28*, 100246. <https://doi.org/10.1016/j.scog.2022.100246>

Moschopoulos, N., Nimatoudis, I., Kaprinis, S., Boutsikos, K., Sidiras, C., & Iliadou, V. (2021). Implications for Early Diagnosis and Treatment in Schizophrenia Due to Correlation between Auditory Perceptual Deficits and Cognitive Impairment. *J Clin Med*, *10*(19). <https://doi.org/10.3390/jcm10194557>

Muller-Preuss, P., & Ploog, D. (1981). Inhibition of auditory cortical neurons during phonation. *Brain Res*, *215*(1-2), 61-76. <https://doi.org/10.1016/0006-8993(81)90491-1>

Murphy, T. K., Haigh, S. M., Coffman, B. A., & Salisbury, D. F. (2020). Mismatch Negativity and Impaired Social Functioning in Long-Term and in First Episode Schizophrenia Spectrum Psychosis. *Front Psychiatry*, *11*, 544. <https://doi.org/10.3389/fpsyt.2020.00544>

Naatanen, R. (1985). Selective attention and stimulus processing: Reflections in event-related potentials, magnetoencephalogram and regional cerebral blood flow. . *Attention & Performance*, *11*, 355-373.

Naatanen, R., Shiga, T., Asano, S., & Yabe, H. (2015). Mismatch negativity (MMN) deficiency: a break-through biomarker in predicting psychosis onset. *Int J Psychophysiol*, *95*(3), 338-344. <https://doi.org/10.1016/j.ijpsycho.2014.12.012>

Naatanen, R., Simpson, M., & Loveless, N. E. (1982). Stimulus deviance and evoked potentials. *Biol Psychol*, *14*(1-2).

Naatanen, R., Todd, J., & Schall, U. (2016). Mismatch negativity (MMN) as biomarker predicting psychosis in clinically at-risk individuals. *Biol Psychol*, *116*, 36-40. <https://doi.org/10.1016/j.biopsycho.2015.10.010>

Neuhaus, A. H., Brandt, E. S., Goldberg, T. E., Bates, J. A., & Malhotra, A. K. (2013). Evidence for impaired visual prediction error in schizophrenia. *Schizophr Res*, *147*(2-3), 326-330. <https://doi.org/10.1016/j.schres.2013.04.004>

Niznikiewicz, M. A., Spencer, K. M., Dickey, C., Voglmaier, M., Seidman, L. J., Shenton, M. E., & McCarley, R. W. (2009). Abnormal pitch mismatch negativity in individuals with schizotypal personality disorder. *Schizophr Res*, *110*(1-3), 188-193. <https://doi.org/10.1016/j.schres.2008.10.017>

O'Driscoll, G. A., & Callahan, B. L. (2008). Smooth pursuit in schizophrenia: a meta-analytic review of research since 1993. *Brain Cogn*, *68*(3), 359-370. <https://doi.org/10.1016/j.bandc.2008.08.023>

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. *Clin EEG Neurosci*, *47*(1), 3-10. <https://doi.org/10.1177/1550059415581708>

Okruszek, L., & Pilecka, I. (2017). Biological motion processing in schizophrenia - Systematic review and meta-analysis. *Schizophr Res*, *190*, 3-10. <https://doi.org/10.1016/j.schres.2017.03.013>

Olson, I. R., & Berryhill, M. (2009). Some surprising findings on the involvement of the parietal lobe in human memory. *Neurobiol Learn Mem*, *91*(2), 155-165. <https://doi.org/10.1016/j.nlm.2008.09.006>

Papageorgiou, C., Kontaxakis, V. P., Havaki-Kontaxaki, B. J., Stamouli, S., Vasios, C., Asvestas, P., Matsopoulos, G. K., Kontopantelis, E., Rabavilas, A., Uzunoglu, N., & Christodoulou, G. N. (2001). Impaired P600 in neuroleptic naive patients with first-episode schizophrenia. *Neuroreport*, *12*(13), 2801-2806. <https://doi.org/10.1097/00001756-200109170-00010>

Park, S., & McTigue, K. (1997). Working memory and the syndromes of schizotypal personality. *Schizophr Res*, *26*(2-3), 213-220. <https://doi.org/10.1016/s0920-9964(97)00051-0>

Patel, G. H., Arkin, S. C., Ruiz-Betancourt, D. R., DeBaun, H. M., Strauss, N. E., Bartel, L. P., Grinband, J., Martinez, A., Berman, R. A., Leopold, D. A., & Javitt, D. C. (2020). What you see is what you get: visual scanning failures of naturalistic social scenes in schizophrenia. *Psychol Med*, 1-10. <https://doi.org/10.1017/S0033291720001646>

Phillipson, O. T., & Harris, J. P. (1985). Perceptual changes in schizophrenia: a questionnaire survey. *Psychol Med*, *15*(4), 859-866. <https://doi.org/10.1017/s0033291700005092>

Piskulic, D., Olver, J. S., Norman, T. R., & Maruff, P. (2007). Behavioural studies of spatial working memory dysfunction in schizophrenia: a quantitative literature review. *Psychiatry Res*, *150*(2), 111-121. <https://doi.org/10.1016/j.psychres.2006.03.018>

Price, G. W., Michie, P. T., Johnston, J., Innes-Brown, H., Kent, A., Clissa, P., & Jablensky, A. V. (2006). A multivariate electrophysiological endophenotype, from a unitary cohort, shows greater research utility than any single feature in the Western Australian family study of schizophrenia. *Biol Psychiatry*, *60*(1), 1-10. <https://doi.org/10.1016/j.biopsych.2005.09.010>

Pritchard, W. S., Brandt, M. E., O'Dell, T. J., Shappell, S. A., & Barratt, E. S. (1985). Individual differences in visual event-related potentials: P300 cognitive augmenting/reducing parallels N100 sensory augmenting/reducing. *Int J Psychophysiol*, *3*(1), 49-56. <https://doi.org/10.1016/0167-8760(85)90019-4>

Rabinowicz, E. F., Silipo, G., Goldman, R., & Javitt, D. C. (2000). Auditory sensory dysfunction in schizophrenia: imprecision or distractibility? *Arch Gen Psychiatry*, *57*(12), 1149-1155. <https://doi.org/10.1001/archpsyc.57.12.1149>

Ramage, E. M., Weintraub, D. M., Allen, D. N., & Snyder, J. S. (2012). Evidence for stimulus-general impairments on auditory stream segregation tasks in schizophrenia. *J Psychiatr Res*, *46*(12), 1540-1545. <https://doi.org/10.1016/j.jpsychires.2012.08.028>

Ranlund, S., Calafato, S., Thygesen, J. H., Lin, K., Cahn, W., Crespo-Facorro, B., de Zwarte, S. M. C., Diez, A., Di Forti, M., Group, Iyegbe, C., Jablensky, A., Jones, R., Hall, M. H., Kahn, R., Kalaydjieva, L., Kravariti, E., McDonald, C., McIntosh, A. M., . . . Bramon, E. (2018). A polygenic risk score analysis of psychosis endophenotypes across brain functional, structural, and cognitive domains. *Am J Med Genet B Neuropsychiatr Genet*, *177*(1), 21-34. <https://doi.org/10.1002/ajmg.b.32581>

Reichenberg, A. (2010). The assessment of neuropsychological functioning in schizophrenia. *Dialogues Clin Neurosci*, *12*(3), 383-392. <https://www.ncbi.nlm.nih.gov/pubmed/20954432>

Reilly, T. J., Nottage, J. F., Studerus, E., Rutigliano, G., Micheli, A. I., Fusar-Poli, P., & McGuire, P. (2018). Gamma band oscillations in the early phase of psychosis: A systematic review. *Neurosci Biobehav Rev*, *90*, 381-399. <https://doi.org/10.1016/j.neubiorev.2018.04.006>

Restuccia, D., Zanini, S., Cazzagon, M., Del Piero, I., Martucci, L., & Della Marca, G. (2009). Somatosensory mismatch negativity in healthy children. *Dev Med Child Neurol*, *51*(12), 991-998. <https://doi.org/10.1111/j.1469-8749.2009.03367.x>

Rodriguez, M., Zaytseva, Y., Cvrckova, A., Dvoracek, B., Dorazilova, A., Jonas, J., Sustova, P., Vorackova, V., Hajkova, M., Kratochvilova, Z., Spaniel, F., & Mohr, P. (2019). Cognitive Profiles and Functional Connectivity in First-Episode Schizophrenia Spectrum Disorders - Linking Behavioral and Neuronal Data. *Front Psychol*, *10*, 689. <https://doi.org/10.3389/fpsyg.2019.00689>

Rosburg, T., Boutros, N. N., & Ford, J. M. (2008). Reduced auditory evoked potential component N100 in schizophrenia--a critical review. *Psychiatry Res*, *161*(3), 259-274. <https://doi.org/10.1016/j.psychres.2008.03.017>

Rutschmann, J., Cornblatt, B., & Erlenmeyer-Kimling, L. (1980). Auditory recognition memory in adolescents at risk for schizophrenia: report on a verbal continuous recognition task. *Psychiatry Res*, *3*(2), 151-161. <https://doi.org/10.1016/0165-1781(80)90032-3>

Saint-Amour, D., De Sanctis, P., Molholm, S., Ritter, W., & Foxe, J. J. (2007). Seeing voices: High-density electrical mapping and source-analysis of the multisensory mismatch negativity evoked during the McGurk illusion. *Neuropsychologia*, *45*(3), 587-597. <https://doi.org/10.1016/j.neuropsychologia.2006.03.036>

Salisbury, D. (2010). N400 to lexical ambiguity and semantic incongruity in schizophrenia. *Int J Psychophysiol*, *75*(2), 127-132. <https://doi.org/10.1016/j.ijpsycho.2009.10.002>

Salisbury, D. F., Kohler, J., Shenton, M. E., & McCarley, R. W. (2020). Deficit Effect Sizes and Correlations of Auditory Event-Related Potentials at First Hospitalization in the Schizophrenia Spectrum. *Clin EEG Neurosci*, *51*(4), 198-206. <https://doi.org/10.1177/1550059419868115>

Salisbury, D. F., Kuroki, N., Kasai, K., Shenton, M. E., & McCarley, R. W. (2007). Progressive and interrelated functional and structural evidence of post-onset brain reduction in schizophrenia. *Arch Gen Psychiatry*, *64*(5), 521-529. <https://doi.org/10.1001/archpsyc.64.5.521>

Salisbury, D. F., McCathern, A. G., Coffman, B. A., Murphy, T. K., & Haigh, S. M. (2018). Complex mismatch negativity to tone pair deviants in long-term schizophrenia and in the first-episode schizophrenia spectrum. *Schizophr Res*, *191*, 18-24. <https://doi.org/10.1016/j.schres.2017.04.044>

Schmidt, A., Diwadkar, V. A., Smieskova, R., Harrisberger, F., Lang, U. E., McGuire, P., Fusar-Poli, P., & Borgwardt, S. (2014). Approaching a network connectivity-driven classification of the psychosis continuum: a selective review and suggestions for future research. *Front Hum Neurosci*, *8*, 1047. <https://doi.org/10.3389/fnhum.2014.01047>

Schmidt-Hansen, M., & Honey, R. C. (2009). Working memory and multidimensional schizotypy: dissociable influences of the different dimensions. *Cogn Neuropsychol*, *26*(7), 655-670. <https://doi.org/10.1080/02643291003644501>

Schnakenberg Martin, A. M., Bartolomeo, L., Howell, J., Hetrick, W. P., Bolbecker, A. R., Breier, A., Kidd, G., & O'Donnell, B. F. (2018). Auditory feature perception and auditory hallucinatory experiences in schizophrenia spectrum disorder. *Eur Arch Psychiatry Clin Neurosci*, *268*(7), 653-661. <https://doi.org/10.1007/s00406-017-0839-1>

Schutte, M. J. L., Bohlken, M. M., Collin, G., Abramovic, L., Boks, M. P. M., Cahn, W., Dauwan, M., van Dellen, E., van Haren, N. E. M., Hugdahl, K., Koops, S., Mandl, R. C. W., & Sommer, I. E. C. (2021). Functional connectome differences in individuals with hallucinations across the psychosis continuum. *Sci Rep*, *11*(1), 1108. <https://doi.org/10.1038/s41598-020-80657-8>

Seabury, R. D., & Cannon, T. D. (2020). Memory Impairments and Psychosis Prediction: A Scoping Review and Theoretical Overview. *Neuropsychol Rev*, *30*(4), 521-545. <https://doi.org/10.1007/s11065-020-09464-2>

Sehatpour, P., Javitt, D. C., De Baun, H. M., Carlson, M., Beloborodova, A., Margolin, D. H., Carlton, M. B. L., Brice, N. L., & Kantrowitz, J. T. (2022). Mismatch negativity as an index of target engagement for excitation/inhibition-based treatment development: a double-blind, placebo-controlled, randomized, single-dose cross-over study of the serotonin type-3 receptor antagonist CVN058. *Neuropsychopharmacology*, *47*(3), 711-718. <https://doi.org/10.1038/s41386-021-01170-8>

Seidman, L. J., Meyer, E. C., Giuliano, A. J., Breiter, H. C., Goldstein, J. M., Kremen, W. S., Thermenos, H. W., Toomey, R., Stone, W. S., Tsuang, M. T., & Faraone, S. V. (2012). Auditory working memory impairments in individuals at familial high risk for schizophrenia. *Neuropsychology*, *26*(3), 288-303. <https://doi.org/10.1037/a0027970>

Seidman, L. J., Pousada-Casal, A., Scala, S., Meyer, E. C., Stone, W. S., Thermenos, H. W., Molokotos, E., Agnew-Blais, J., Tsuang, M. T., & Faraone, S. V. (2016). Auditory Vigilance and Working Memory in Youth at Familial Risk for Schizophrenia or Affective Psychosis in the Harvard Adolescent Family High Risk Study. *J Int Neuropsychol Soc*, *22*(10), 1026-1037. <https://doi.org/10.1017/S1355617716000242>

Seidman, L. J., Shapiro, D. I., Stone, W. S., Woodberry, K. A., Ronzio, A., Cornblatt, B. A., Addington, J., Bearden, C. E., Cadenhead, K. S., Cannon, T. D., Mathalon, D. H., McGlashan, T. H., Perkins, D. O., Tsuang, M. T., Walker, E. F., & Woods, S. W. (2016). Association of Neurocognition With Transition to Psychosis: Baseline Functioning in the Second Phase of the North American Prodrome Longitudinal Study. *JAMA Psychiatry*, *73*(12), 1239-1248. <https://doi.org/10.1001/jamapsychiatry.2016.2479>

She, S., Li, H., Ning, Y., Ren, J., Wu, Z., Huang, R., Zhao, J., Wang, Q., & Zheng, Y. (2017). Revealing the Dysfunction of Schematic Facial-Expression Processing in Schizophrenia: A Comparative Study of Different References. *Front Neurosci*, *11*, 314. <https://doi.org/10.3389/fnins.2017.00314>

Shelley, A. M., Silipo, G., & Javitt, D. C. (1999). Diminished responsiveness of ERPs in schizophrenic subjects to changes in auditory stimulation parameters: implications for theories of cortical dysfunction. *Schizophr Res*, *37*(1), 65-79. <https://doi.org/10.1016/s0920-9964(98)00138-8>

Shin, K. S., Kim, J. S., Kim, S. N., Koh, Y., Jang, J. H., An, S. K., O'Donnell, B. F., Chung, C. K., & Kwon, J. S. (2012). Aberrant auditory processing in schizophrenia and in subjects at ultra-high-risk for psychosis. *Schizophr Bull*, *38*(6), 1258-1267. <https://doi.org/10.1093/schbul/sbr138>

Siddi, S., Petretto, D. R., & Preti, A. (2017). Neuropsychological correlates of schizotypy: a systematic review and meta-analysis of cross-sectional studies. *Cogn Neuropsychiatry*, *22*(3), 186-212. <https://doi.org/10.1080/13546805.2017.1299702>

Silverstein, S. M., Fradkin, S. I., & Demmin, D. L. (2020). Schizophrenia and the retina: Towards a 2020 perspective. *Schizophr Res*, *219*, 84-94. <https://doi.org/10.1016/j.schres.2019.09.016>

Silverstein, S. M., & Lai, A. (2021). The Phenomenology and Neurobiology of Visual Distortions and Hallucinations in Schizophrenia: An Update. *Front Psychiatry*, *12*, 684720. <https://doi.org/10.3389/fpsyt.2021.684720>

Sit, D., & Haigh, S. (2019). Use of "Lights" for Bipolar Depression. *Curr Psychiatry Rep*, *21*(6), 45. <https://doi.org/10.1007/s11920-019-1025-0>

Sloan, N. P., Byrne, L. K., Enticott, P. G., & Lum, J. A. G. (2021). Non-Invasive Brain Stimulation Does Not Improve Working Memory in Schizophrenia: A Meta-Analysis of Randomised Controlled Trials. *Neuropsychol Rev*, *31*(1), 115-138. <https://doi.org/10.1007/s11065-020-09454-4>

Sperling, G. (1960). Negative Afterimage without Prior Positive Image. *Science*, *131*(3413), 1613-1614. <https://doi.org/10.1126/science.131.3413.1613>

Sperry, R. W. (1950). Neural basis of the spontaneous optokinetic response produced by visual inversion. *J Comp Physiol Psychol*, *43*(6), 482-489. <https://doi.org/10.1037/h0055479>

Stevens, A. A., Goldman-Rakic, P. S., Gore, J. C., Fulbright, R. K., & Wexler, B. E. (1998). Cortical dysfunction in schizophrenia during auditory word and tone working memory demonstrated by functional magnetic resonance imaging. *Arch Gen Psychiatry*, *55*(12), 1097-1103. <https://doi.org/10.1001/archpsyc.55.12.1097>

Tai, A. M. Y., Albuquerque, A., Carmona, N. E., Subramanieapillai, M., Cha, D. S., Sheko, M., Lee, Y., Mansur, R., & McIntyre, R. S. (2019). Machine learning and big data: Implications for disease modeling and therapeutic discovery in psychiatry. *Artif Intell Med*, *99*, 101704. <https://doi.org/10.1016/j.artmed.2019.101704>

Taylor, J. A., Matthews, N., Michie, P. T., Rosa, M. J., & Garrido, M. I. (2017). Auditory prediction errors as individual biomarkers of schizophrenia. *Neuroimage Clin*, *15*, 264-273. <https://doi.org/10.1016/j.nicl.2017.04.027>

Thakkar, K. N., Ghermezi, L., Silverstein, S. M., Slate, R., Yao, B., Achtyes, E. D., & Brascamp, J. W. (2021). Stronger tilt aftereffects in persons with schizophrenia. *J Abnorm Psychol*, *130*(2), 186-197. <https://doi.org/10.1037/abn0000653>

Thakkar, K. N., & Rolfs, M. (2019). Disrupted Corollary Discharge in Schizophrenia: Evidence From the Oculomotor System. *Biol Psychiatry Cogn Neurosci Neuroimaging*, *4*(9), 773-781. <https://doi.org/10.1016/j.bpsc.2019.03.009>

Toader, O., von Heimendahl, M., Schuelert, N., Nissen, W., & Rosenbrock, H. (2020). Suppression of Parvalbumin Interneuron Activity in the Prefrontal Cortex Recapitulates Features of Impaired Excitatory/Inhibitory Balance and Sensory Processing in Schizophrenia. *Schizophr Bull*, *46*(4), 981-989. <https://doi.org/10.1093/schbul/sbz123>

Umbricht, D., & Krljes, S. (2005). Mismatch negativity in schizophrenia: a meta-analysis. *Schizophr Res*, *76*(1), 1-23. <https://doi.org/10.1016/j.schres.2004.12.002>

Unschuld, P. G., Buchholz, A. S., Varvaris, M., van Zijl, P. C., Ross, C. A., Pekar, J. J., Hock, C., Sweeney, J. A., Tamminga, C. A., Keshavan, M. S., Pearlson, G. D., Thaker, G. K., & Schretlen, D. J. (2014). Prefrontal brain network connectivity indicates degree of both schizophrenia risk and cognitive dysfunction. *Schizophr Bull*, *40*(3), 653-664. <https://doi.org/10.1093/schbul/sbt077>

Urban, A., Kremlacek, J., Masopust, J., & Libiger, J. (2008). Visual mismatch negativity among patients with schizophrenia. *Schizophr Res*, *102*(1-3), 320-328. <https://doi.org/10.1016/j.schres.2008.03.014>

van Gool, K. C. A., Collin, G., Bauer, C. C. C., Molokotos, E., Mesholam-Gately, R. I., Thermenos, H. W., Seidman, L. J., Gabrieli, J. D. E., Whitfield-Gabrieli, S., & Keshavan, M. S. (2022). Altered working memory-related brain activity in children at familial high risk for psychosis: A preliminary study. *Schizophr Res*, *240*, 186-192. <https://doi.org/10.1016/j.schres.2021.12.030>

Vogel, B. O., Stasch, J., Walter, H., & Neuhaus, A. H. (2018). Emotional context restores cortical prediction error responses in schizophrenia. *Schizophr Res*, *197*, 434-440. <https://doi.org/10.1016/j.schres.2018.02.030>

Wang, L., Li, Q., Wu, Y., Ji, G. J., Wu, X., Xiao, G., Qiu, B., Hu, P., Chen, X., He, K., & Wang, K. (2022). Intermittent theta burst stimulation improved visual-spatial working memory in treatment-resistant schizophrenia: A pilot study. *J Psychiatr Res*, *149*, 44-53. <https://doi.org/10.1016/j.jpsychires.2022.02.019>

Weintraub, D. M., Ramage, E. M., Sutton, G., Ringdahl, E., Boren, A., Pasinski, A. C., Thaler, N., Haderlie, M., Allen, D. N., & Snyder, J. S. (2012). Auditory stream segregation impairments in schizophrenia. *Psychophysiology*, *49*(10), 1372-1383. <https://doi.org/10.1111/j.1469-8986.2012.01457.x>

Whitaker, L., Jones, C. R., Wilkins, A. J., & Roberson, D. (2016). Judging the Intensity of Emotional Expression in Faces: the Effects of Colored Tints on Individuals With Autism Spectrum Disorder. *Autism Res*, *9*(4), 450-459. <https://doi.org/10.1002/aur.1506>

Wible, C. G., Lee, K., Molina, I., Hashimoto, R., Preus, A. P., Roach, B. J., Ford, J. M., Mathalon, D. H., McCarthey, G., Turner, J. A., Potkin, S. G., O'Leary, D., Belger, A., Diaz, M., Voyvodic, J., Brown, G. G., Notestine, R., Greve, D., Lauriello, J., & Fbirn. (2009). fMRI activity correlated with auditory hallucinations during performance of a working memory task: data from the FBIRN consortium study. *Schizophr Bull*, *35*(1), 47-57. <https://doi.org/10.1093/schbul/sbn142>

Winkler, I. (2007). Interpreting the Mismatch Negativity. *J Psychophysiology*, *21*(3-4). <https://doi.org/https://doi.org/10.1027/0269-8803.21.34.147>

Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: predictive regularity representations and perceptual objects. *Trends Cogn Sci*, *13*(12), 532-540. <https://doi.org/10.1016/j.tics.2009.09.003>

Wolf, A., Ueda, K., & Hirano, Y. (2021). Recent updates of eye movement abnormalities in patients with schizophrenia: A scoping review. *Psychiatry Clin Neurosci*, *75*(3), 82-100. <https://doi.org/10.1111/pcn.13188>

Wu, J., Buchsbaum, M. S., Moy, K., Denlea, N., Kesslak, P., Tseng, H., Plosnaj, D., Hetu, M., Potkin, S., Bracha, S., & et al. (1993). Olfactory memory in unmedicated schizophrenics. *Schizophr Res*, *9*(1), 41-47. <https://doi.org/10.1016/0920-9964(93)90008-7>

Yin, G., She, S., Zhao, L., & Zheng, Y. (2018). The dysfunction of processing emotional faces in schizophrenia revealed by expression-related visual mismatch negativity. *Neuroreport*, *29*(10), 814-818. <https://doi.org/10.1097/WNR.0000000000001037>

Zerr, M., Freihorst, C., Schutz, H., Sinke, C., Muller, A., Bleich, S., Munte, T. F., & Szycik, G. R. (2019). Brief Sensory Training Narrows the Temporal Binding Window and Enhances Long-Term Multimodal Speech Perception. *Front Psychol*, *10*, 2489. <https://doi.org/10.3389/fpsyg.2019.02489>

Zheng, W., Zhang, Q. E., Cai, D. B., Ng, C. H., Ungvari, G. S., Ning, Y. P., & Xiang, Y. T. (2018). Neurocognitive dysfunction in subjects at clinical high risk for psychosis: A meta-analysis. *J Psychiatr Res*, *103*, 38-45. <https://doi.org/10.1016/j.jpsychires.2018.05.001>