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Research Paper

Altered theta band and theta/beta ratio in mismatch negativity associate with treatment effect in schizophrenia with auditory hallucinations

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ABSTRACT

Evidence suggests that attenuated mismatch negative (MMN) waves have a close link to auditory verbal hallucinations (AVH) and their clinical outcomes, especially impaired neural oscillations such as θ, β representing attentional control. In current study, thirty patients with schizophrenia and AVH (SZ) and twenty-nine healthy controls (HC) underwent multi-feature MMN paradigm measurements including frequency and duration deviant stimuli (fMMN and dMMN). Clinical symptoms and MMN paradigm were followed up among SZ group after 8-week treatment. Results demonstrated that hallucinating patients exhibited attenuated dMMN amplitudes across Fz ($p = 0.010$), F1 ($p = 0.020$) and F2 ($p = 0.014$) electrodes, which were trendily recovered after treatment. Meanwhile, θ band and TBR at frontal fMMN and right temporal dMMN were significantly reduced in SZs. After treatment, SZs showed reduced scores of Hoffman's Auditory Hallucinations Rating Scale (AHRS), with a remarkable recovery in right temporal TBR of dMMN ($p = 0.042$) and a trending change in frontal TBR of fMMN ($p = 0.090$). The β band was decreased in dMMN ($p = 0.035$) by time. Additionally, P3 scores of Positive and Negative Syndrome Scale (PANSS) were negatively correlated with θ band of fMMN at baseline. Baseline scores of AHRS negatively predicted changes of dMMN amplitude after treatment, and changes of β band in left temporal dMMN predicted the reduction in scores of PANSS negative scale. These findings supported that deficits in θ oscillation and TBR during auditory attention process were crucial to clinical progression of schizophrenia with AVH.

1. Introduction

Schizophrenia is a devastating disease with high recurrence and high disability, resulting in 13.4 million years of disability-related life loss (YLDs) in 2016 (Charlson et al., 2018). Auditory verbal hallucination (AVH), which is one of hallmark symptoms of schizophrenia, has been reported by approximately 60–90 % patients with schizophrenia through their life course of illness (Saha et al., 2005). Although symptom of AVHs can be rapidly alleviate in majority of patients with antipsychotic medication (Sommer et al., 2012), approximately 30 % of patients with schizophrenia may become resistant to medications (Liu et al., 2015). For those who suffer from the disorder, evidence suggests that AVH and attentional deficits show a synergistic relationship (Coffman et al., 2022). Behavioral data of auditory attentional control are inversely correlated with auditory hallucination severity, indicating the failure of frontal attention/behavioral control on perceptual misrepresentations (Hugdahl, 2009). However, the neurological mechanism underling the effects of auditory attention on AVH symptom and its treatment outcomes in schizophrenia are still elusive.

Electroencephalographically derived event-related potential (ERP) is

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a non-invasive technology that provides an exquisitely sensitive measure to sensory and cognitive impairments in schizophrenia. Among them, MMN is one of well-established biomarkers to reflect attentional and perceptual impairment in schizophrenia (Justo-Guillén et al., 2019; Kong et al., 2024). Auditory MMN was elicited by any discriminable change in auditory stimulation regardless of whether one is consciously attending to such deviation. One commonly accepted theory of MMN generation is that it reflects a dis-concordance between the sensory-memory representation of the precedingly regular auditory stimulation and the input of deviant auditory event (Naatanen et al., 2005). Evidence suggests that this process of change-detection firstly occurs in the primary and secondary auditory cortices, and subsequently triggers frontal-cortex processes underlying the involuntary attention switch to stimulus change. Reduced MMN amplitude is consistently found in psychotic disorders (Donaldson et al., 2020; Erickson et al., 2016) and is associated with various psychotic symptoms (Donaldson et al., 2021; Fisher et al., 2011; Kim et al., 2020), including auditory hallucinations (Fisher et al., 2008b, 2012). Given these characteristics, MMN is proposed as a potential "break-through biomarker" for understanding the treatment and prediction of psychotic symptoms over time (Light and Näätänen, 2013; Näätänen et al., 2015).

Previous studies have investigated the relationship between MMN and positive symptoms, but the results are inconsistent (Chen et al., 2016; Donaldson et al., 2020; Erickson et al., 2017). Some evidence indicates that MMN deficits were associated with AVHs symptoms not only in chronic schizophrenia (Fisher et al., 2011, 2014; Perrin et al., 2018) but also in early phase psychosis (Rudolph et al., 2015). Other investigations have compared the MMN generations in schizophrenic patients with and without AVH, and found more diminished MMN amplitudes to duration deviant or pitch deviant in schizophrenic patients with AVH (Francis et al., 2020). However, recent research using free-field tone discrimination as deviant discovered relatively preserved MMN in hallucinating subjects compared to non-hallucinating ones (Perrin et al., 2018). A few longitudinal research have explored MMN as a role of prediction for outcomes of clinical symptoms or functional outcomes (Donaldson et al., 2023; Knott et al., 2020; Light and Näätänen, 2013). These efforts have implicated that the alterations of MMN amplitude might be a valuable predictor of alleviating or worsening auditory hallucinations during both short-term psychological therapy (Knott et al., 2020) or long-term clinical observation (Donaldson et al., 2023). Yet, longitudinal studies of MMN to predict short-term effect of treatment in patients with AVH are sparse and limited. Elucidating which feature of MMN is stable relative to AVH symptom, as well as clarifying the utility of MMN in predicting treatment outcomes of AVH, may be essential to identify reliable neurophysiological markers for better treatment strategies for AVHs.

Nevertheless, with limited information, the standard time-domain ERP measures shed relatively insufficient information on mechanism of neural processing at the circuit level, and there is a trending focus on investigating the characteristics of neuro-oscillatory event-related spectral perturbation (ERSP) among ERP trials in recent years. As reported, frequency bands normally included delta (0.5–4 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (12–24) and gamma (> 24 Hz) frequency ranges. Regarding MMN, previous studies have linked decreased deviant MMN amplitudes mainly to impaired lower frequency oscillations (Hong et al., 2012) in schizophrenia, especially theta band (Choi et al., 2013; Hua et al., 2023; Lee et al., 2017). This is also well-established in rodent studies, showing primary theta frequency band generation during MMN paradigms. The altered MMN may be mediated by the N-Methyl-D-Aspartate Receptor (NMDAR) antagonists or agonists (Lee et al., 2018; Rosburg and Kreitschmann-Andermahr, 2016), which eventually influences theta power of MMN (Kantrowitz et al., 2018). Other frequency bands (e.g. beta, gamma) (Ballesteros et al., 2013) may be also related to MMN activity, especially when recoding with intracranial electrodes in primate's auditory cortex (El Karoui et al., 2015; Suda et al., 2022). Unlike MMN amplitude, which shows maximal signal located at fronto-central sites, neural oscillations of MMN often widely communicate between the temporal, frontal cortices and subcortex (Lakatos et al., 2020; Suda et al., 2022). And both frontal and temporal cortices of demonstrated altered theta or beta band responses to deviant stimuli at cross-sectional studies of schizophrenia (Sauer et al., 2023; Suda et al., 2022). More recently, a cross-band measure, theta/beta ratio, has been widely used as an index of attention in control healthy (Kobayashi et al., 2020) and patients with schizophrenia (Markiewicz and Dobrowolska, 2020). Though evidence is in the altered band of MMN among schizophrenia, its relationship with clinical symptom, such as AVH, is far from being understood. Finally, to our knowledge, seldom research has investigated the changes of band oscillations in MMN after treatment among schizophrenic patients, and rarely no evidence was established in patients with AVH.

The present study aimed to examine the differences of MMN amplitude and spectral perturbation in cases with schizophrenia experiencing AVH and non-patient healthy comparison participants, and to follow up its reliability as clinical predictor after 8-week treatment. Employing the multi-feature MMN paradigm, included both frequency and tone as deviance stimuli, we hypothesized that the amplitude of frontal MMN amplitude and neural oscillation in frontal and temporal cortices will be impaired in schizophrenia patients with AVH. Further, it is expected that alterations of MMN amplitude, theta, beta band oscillation or theta/beta ratio will be recovered to some extent after treatment. Finally, the features of MMN will be associated with severity of AVH and fluctuate with the exacerbation or amelioration of AVH symptom.

2. Method

2.1. Participants

Thirty patients with schizophrenia were recruited at Shanghai Mental Health Center (SMHC). All patients met the diagnostic criteria for schizophrenia or schizophreniform disorder based on the Structured Clinical Interview for DSM-5 Patient Edition and reported auditory hallucinations at the enrollment, seeking medication to relieve clinical symptoms. The positive and negative syndrome scale (PANSS) was used to assess clinical psychopathology for patients. Participants were enrolled only when they have score of P3 item greater than or equal to 2 (Li et al., 2023). And severity of auditory hallucination for each patient was assessed by the Auditory Hallucination Rating Scale (AHRS) (Hoffman et al., 2003). Twenty-nine healthy controls were recruited from local communities in Shanghai, matched with the age and gender of patients. They were assessed using the Structured Clinical Interview for DSM-5 (non-patient version) to exclude the history of any psychiatric disorder. Subjects with a family history of psychosis in their first-degree relatives were also excluded to minimize the influence of genetic risks. Both patients and healthy participants underwent EEG measurement at baseline. After receiving 8-week treatment, 16 patients underwent the same clinical assessments and EEG acquisition as those of baseline.

All participants were between the ages of 15 and 50 years with education levels higher than primary school; were free of substance abuse, suicidal ideation, pregnancy, and unstable medical illness. Written informed consent was obtained from each participant or participant's guardian. The study protocol was approved by the Ethics Committee of SMHC (the ethic approval number: 2017-04) and conducted in accordance with the Declaration of Helsinki.

2.2. MMN paradigm and procedure

Auditory ERPs were obtained using an MMN paradigm as in our previous study (Wu et al., 2022). In brief, 675 standard tones (1000 Hz, 50 ms), 75 frequency deviant tones (1500 Hz, 50 ms), and 75 duration deviant tones (1000 Hz, 100 ms) were presented randomly with a 500 ms stimulus onset asynchrony in the paradigm. Auditory stimuli were

delivered through headphones while participants were instructed to watch a silent cartoon without paying attention to the tone.

During EEG recording, the participants were seated in a quiet electromagnetic-shielded room. The continuous EEG signals were recorded with a 64-channel BrainCap (Brain Products Company, Germany), referenced to the tip of the nose, and filtered online (0.01–200 Hz). Two EOG electrodes were placed below the left eye and above the right eye respectively. The impedance was maintained below 5 kΩ and the sample rate was 1000 Hz.

2.3. Data preprocessing

The offline preprocessing was firstly conducted by BrainVision Analyzer 2.0 (Brain Products Company, Germany). Raw data were bandpass filtered between 0.1 and 40 Hz with a zero-phase-shift IIR Butterworth filter (24 dB/Oct), re-referenced to an average reference, and corrected for ocular movements and other artifacts (Wang et al., 2018). Continuous data were then segmented into epochs from −1000 to 2000 ms relative to the stimulus onset and subsequently baseline corrected (from −200 ms to 0 ms relative to the stimulus onset). Epochs were rejected if the amplitudes exceeded ±100 uV. For 3 regions of interest, 9 electrode sites were selected for subsequent analysis: frontal area (F1, Fz, F2), left temporal area (T7, CP5, TP7), right temporal area (T8, CP6, TP8).

2.4. ERP analysis

The preprocessed data were bandpass filtered at 0.01–15 Hz for ERP analysis. For each participant, average waveform was calculated through all artifact-rejected epochs within time window of -200 ms to 500 ms. MMN was obtained by subtracting waveforms of standard stimuli from the responding waves of frequency deviant tones (fMMN) or duration deviant tones (dMMN), respectively. MMN peak was identified as the most negative wave in specific time window (130 ms–250 ms for fMMN; 150 ms–260 ms for dMMN) at each electrode of interest. Latency was measured as the time from the onset to MMN peak. Mean amplitude was also calculated by averaging the voltage values within the time window.

2.5. ERSP analysis

Complex Morlet wavelet based on EEGLAB (Delorme and Makeig, 2004) and MATLAB scripts (MathWorks, Inc., Natick, MA, USA) was used to decompose the preprocessed single trial (epochs from −1000 to 2000 ms) (Wu et al., 2022) to extract the ERSP for the waveforms in response to the deviant tones (Morales and Bowers, 2022). The number of wavelet cycles varied from 3 at 3 Hz to 7 at 40 Hz. At each time point, the extracted data were baseline corrected (from −100 ms to 0 ms relative to the stimulus onset) into log spectrum for each frequency point: $\text{Log spectrum} = 10 \times log_{10}\frac{\text{Timepoint spectrum}}{\text{Average baseline spectrum}}$. Each time point in the MMN time window (130 ms - 250 ms for fMMN, 150 ms - 260 ms for dMMN) was extracted and averaged at 9 electrode sites of interest. The average log spectrum of each frequency point within the frequency bands of interest (θ: 4–7 Hz, β: 13–30 Hz) was calculated based on MMN time window. For further analysis, θ-β ratio (TBR) was calculated as a marker of attentional control (Hao et al., 2019). Given that the ERSP data were logarithmic corrected, TBR was estimated to be the difference in average log spectrum between θ and β.

2.6. Statistical analysis

Statistical analyses were conducted using R (Version 3.5.2) (Team, 2014). For demographic, clinical and ERP data, independent *t*-tests were used to examine the group differences of continuous data, while Chi-squared tests were used to examine the differences of categorical data at baseline. A repeated measures analysis of variance (ANOVA) was performed for ERSP data within patient group, with False Discovery Rate (FDR) correction for 3 regions of interest. For the MMN indicators with significant between-group differences, two-tailed Spearman correlation analysis was performed to detect the relationship between those indicators and clinical characteristics as explorative analysis. The significant threshold level was set at $p < 0.05$.

3. Results

In this study, 30 patients with schizophrenia (SZ) and 29 healthy controls (HC) were recruited at baseline. 7 patients were first-episode schizophrenia, and other patients experienced relapse of schizophrenia with AVH symptom as their main complaints. Among them, 25 patients received only antipsychotics (described as chlorpromazine equivalents), and 5 patients received modified electroconvulsive therapy (MECT) combined with antipsychotics (Table 1). A total of 16 SZs completed clinical and EEG assessments after treatment. At follow-up, 14 patients underwent monotherapy of antipsychotics, other 2 patient received MECT therapy (Table S1). Demographic and clinical characteristics of SZs and HCs were shown in Tables 1 and S2. There was no significant difference in age or gender between SZs and HCs. The average PANSS score for SZs was 68.43 (SD = 12.65). Changes of clinical characteristics after treatment were shown in Table S1. According to AHRS and PANSS Positive Scores, 56.3 % patients responded (reduction rate ≥ 30 %) to the treatment.

3.1. MMN amplitude and latency

We conducted between-group and follow-up analyses of MMN amplitude and latency at frontal area (Fz, F1, F2). Regarding fMMN, no difference was observed in amplitudes of Fz, F1, and F2 electrodes. Latency of fMMN at Fz was significantly slower in SZ than that in HC group ($T = 2.164$, $p = 0.035$), but not in that of F1 or F2. As for dMMN, SZs showed consistently attenuated dMMN amplitudes across Fz (Mean: $T = -3.174$, $p = 0.002$; Peak: $T = -2.717$, $p = 0.010$), F1 (Mean: $T = -2.950$, $p = 0.005$; Peak: $T = -2.422$, $p = 0.020$) and F2 (Mean: $T = -2.613$, $p = 0.011$; Peak: $T = -2.525$, $p = 0.014$) electrodes, comparing to HCs (Fig. 1 and Table S3). While latency of dMMN demonstrated no difference between two groups. After treatment, repeated measures ANOVA revealed mild but insignificant increase of dMMN mean amplitudes in Fz, F1, and F2 in SZs ($F = 2.379$, $p = 0.144$), and tending recovery in peak amplitudes ($F = 3.280$, $p = 0.090$). No change in fMMN amplitudes was found at follow-up.

Table 1
Demographics and clinical characteristics at baseline.

	SZ (n = 30)	HC (n = 29)	T/Z value	p value
Age	29.73 ± 8.30	26.93 ± 6.99	1.400	0.167
Gender (male/female)	11/19	11/18	0.010	0.920
PANSS Positive score	17.77 ± 3.92			
PANSS Negative score	17.43 ± 4.49			
PANSS General score	33.23 ± 6.43			
PANSS Total score	68.43 ± 12.65			
AHRS score	25.67 ± 5.82			
MECT (treated/untreated)	5/25			
Chlorpromazine equivalents (mg)	543.72 ± 278.13			

Note: SZ = patients with schizophrenia, HC = healthy controls, PANSS = Positive and Negative Syndrome Scale, AHRS = Auditory Hallucination Rating Scale, MECT = Modified Electric Convulsive Therapy.

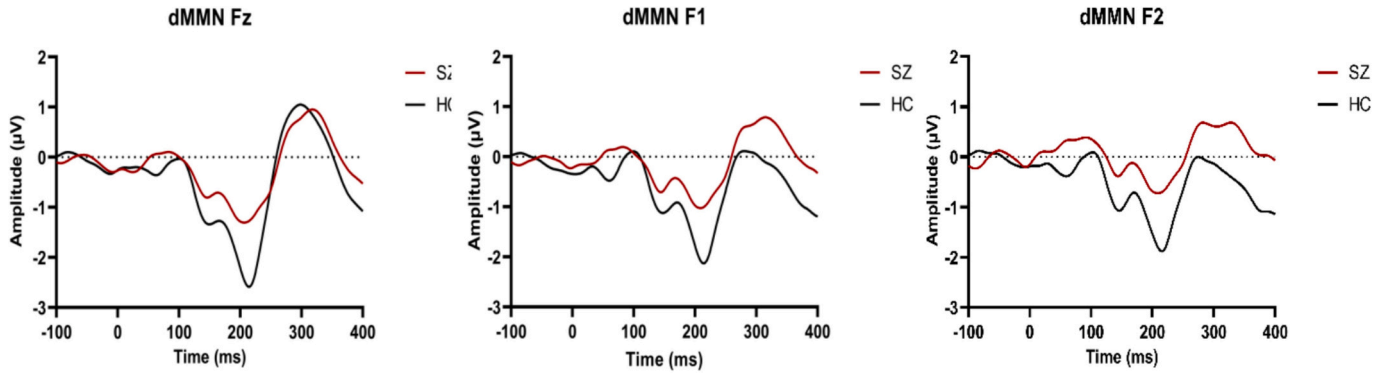

Fig. 1. Grand averaged dMMN waveforms for patients (SZ) and healthy controls (HC) at frontal (Fz, F1, F2) electrode sites.

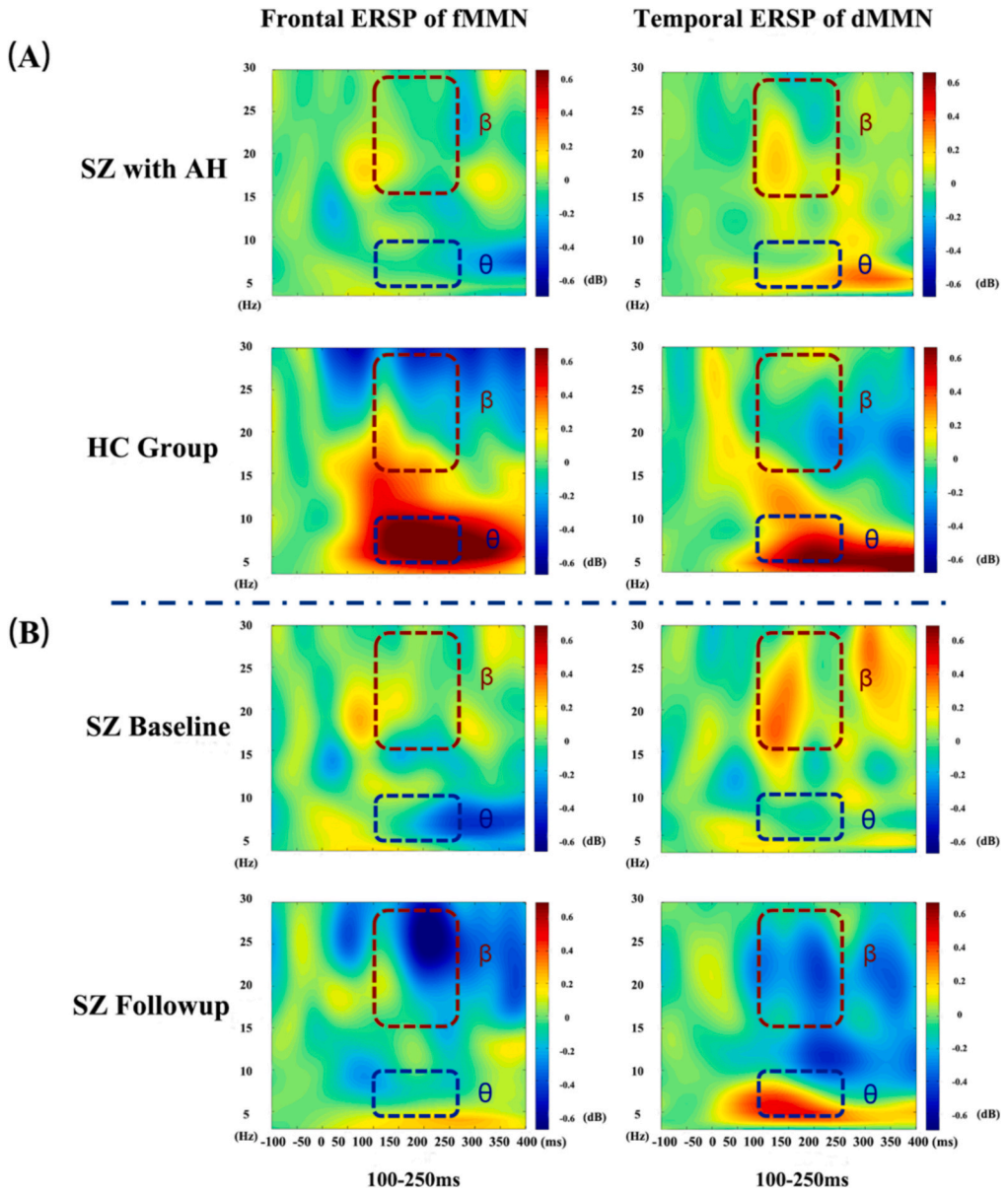

Fig. 2. Oscillations of theta and beta bands related to deviant stimuli within the MMN paradigm. (A). Neuro-oscillatory power of responses to deviant stimuli of fMMN and dMMN at baseline, with significant decrease of activity in the theta (4–7 Hz) frequency range in SZs with AVH. (B). Time-frequency plots showing changes of theta and beta frequency responding to deviants of MMN within SZ group, after 8-week treatment.

3.2. ERSP relative to deviants of MMN

At baseline, SZs showed lower ERSP of θ band ($F = 11.740$, $p = 0.003$, FDR corrected) and reduced TBR ($F = 11.050$, $p = 0.006$, FDR corrected) at frontal area than HCs, relative to fMMN deviants. Besides, relative to dMMN deviants, ERSP of θ band ($F = 6.410$, $p = 0.042$, FDR corrected) and TBR ($F = 7.114$, $p = 0.030$, FDR corrected) were significantly diminished in SZ group at right temporal area. After treatment, SZs demonstrated a significant increase in right temporal TBR ($F = 7.797$, $p = 0.042$, FDR corrected) of dMMN and a trending recovery in frontal TBR ($F = 5.758$, $p = 0.09$, FDR corrected) of fMMN. Further, ERSP of β band were decreased across frontal ($F = 6.059$, $p = 0.035$, FDR corrected) and bilateral temporal area (right: $F = 7.834$, $p = 0.035$; left: $F = 5.376$, $p = 0.035$, FDR corrected) of dMMN by time. Abnormalities of ERSP among SZs with AVH were illustrated in Fig. 2 and Tables S4 and S5, and changes in TBR index after treatment were displayed in Fig. 3.

3.3. Relationship between electrophysiological data and clinical data

Correlation analysis revealed that P3 scores of PANSS were negatively correlated with ERSP relative to fMMN deviants in θ band at the frontal lobe ($r = -0.393$, $p = 0.032$), as well as marginally correlated with frontal TBR of fMMN ($r = -0.351$, $p = 0.057$) among SZs. After 8-week treatment, a positive correlation was observed in changes of β band relative to duration deviants at the left temporal lobe and the reduction of negative scores of PANSS ($r = 0.558$, $p = 0.025$) (Fig. 4). Further, AHRS ($r = -0.517$, $p = 0.040$) and PANSS negative scores ($r = -0.536$, $p = 0.033$) at baseline both negatively predicted changes of mean dMMN amplitude at frontal lobe.

4. Discussion

In this study, we investigated the features of auditory MMN in relation to AVH symptom of schizophrenia and the longitudinal alterations after 8-week treatment. Our primary finding demonstrated a pronounced reduction in dMMN amplitudes at baseline, along with attenuated θ oscillation and TBR index in both fMMN and dMMN deviants in SZ group. After treatment, there was a tending recovery in peak amplitudes of dMMN, and frontal TBR of fMMN and right temporal TBR of dMMN were recovered to some extent. At clinical aspect, frontal θ power in fMMN deviants negatively correlated with P3 scores of PANSS, and changes of left temporal β band of dMMN predicted amelioration of PANSS negative scores. Further, AHRS and PANSS negative scores at baseline both negatively predicted changes of mean dMMN amplitude at frontal lobe. Our results revealed that dysfunction in β and θ oscillations of MMN might be related to symptom of auditory hallucination and reflected possible frontal and temporal circuits underlying abnormal auditory process in schizophrenia.

At time frame level, we found a significant between-group difference in negative amplitudes of dMMN, but not in that of fMMN. Current findings replicated that deficit of dMMN was consistently exhibited in schizophrenia with AVH (Fisher et al., 2014), even when comparing to schizophrenia patients without AVH (Fisher et al., 2008b; Sun et al., 2020). However, some researchers failed to reveal direct correlation between severity of AVH and decreased MMN amplitude at cross-sectional studies (Fisher et al., 2008a, 2008b) Though previous research documented that MMN amplitudes were correlated with both state and trait measures of AVHs (Fisher et al., 2011, 2014), our results might support dMNN amplitude be a trait-related feature of auditory hallucination. Of note, in current study, the amplitudes of fMMN were relatively preserved in patients with AVH. Studies using multi-paradigms of MMN indeed reported dMMN could better discriminate AVH patients from HC and non-AVH patients than fMMN, intensity MMN, etc. (Abalo-Rodriguez et al., 2023). It may follow that encoding of sound duration require more complex processing in the auditory cortex compared to other acoustic features, making the process more vulnerable to auditory cortex defects (Michie et al., 2002) such as auditory hallucination. The sensitivity of duration deviants to auditory hallucination may reflect intricate nature of the cortical computations

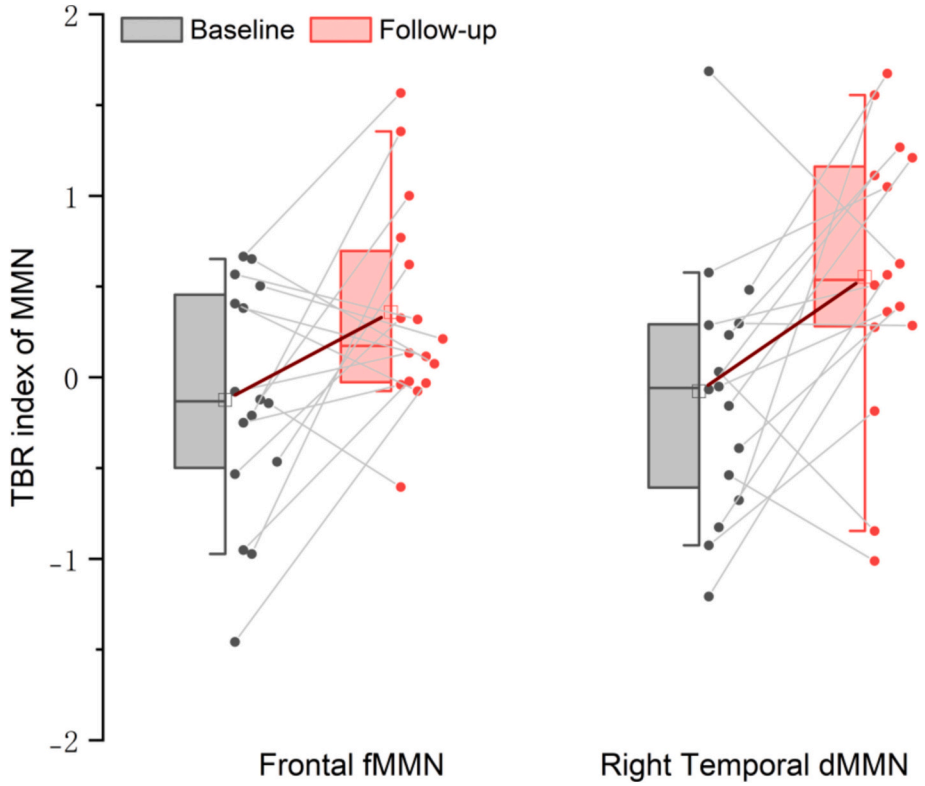

Fig. 3. Changes of TBR index in frontal region of fMMN and right temporal region of dMMN in SZs with AVH, after 8-week treatment.

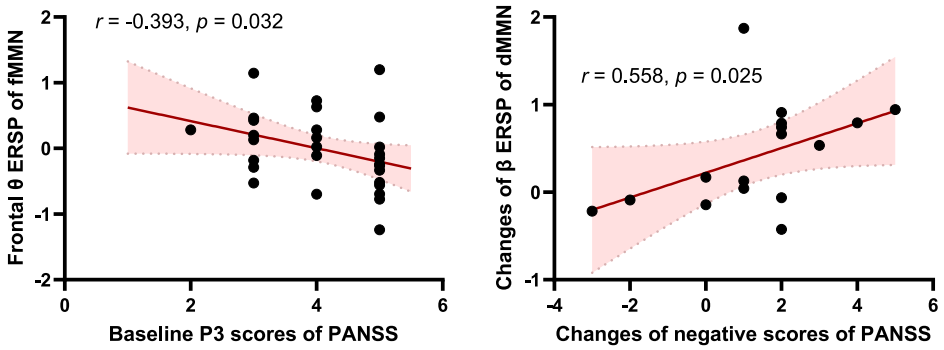

Fig. 4. Correlations between electrophysiological indicators and scores of clinical scales.

involved, which resulted in more defects when more attentional resource is needed for the auditory processing (Fisher et al., 2012). However, it's not necessary to say that auditory hallucination is the only symptom that contributes to deficit of dMMN, but more efforts should be taken on the mechanism of pre-conscious auditory processing related to auditory hallucinations (Fisher et al., 2008b).

Few research has focused on the relationship between specific symptom fluctuation in schizophrenia and changes of MMN by time (Donaldson et al., 2023; Higgins et al., 2021; Lho et al., 2020). Results from long-term observations implicated that reduced MMN amplitude could be a predictor for neural cognition, auditory hallucination, and negative symptoms at follow-up (Donaldson et al., 2023). However, short-term treatments showed inconsistent treatment effects on the changes of MMN (Greenwood et al., 2018; Korostenskaja et al., 2005; Zhou et al., 2013). Our longitudinal comparison revealed a trending recovery of peak dMMN amplitude, but failed to find significant change in mean dMMN amplitude after treatment. Such results may be due to the insufficient treatment time to improve auditory dysfunction or the relatively small sample at follow-up. Though current research included patients only receiving medications and those combined with ECT, the sample is limited for further analysis on which strategy is better for normalize the MMN amplitude. Nevertheless, we found baseline scores of AHRS and PANSS negative subscale both negatively predicted changes of mean dMMN amplitude at frontal lobe, suggesting clinical heterogeneity at baseline may affect response of neuroplasticity to treatment.

In the present study, the most interesting finding is that altered spectral oscillation of MMN among patients with AVH, indicating that the lower θ oscillation and TBR level could be substantial profiles for predicting outcomes of auditory hallucination. A growing literature has indicated MMN deficits in schizophrenia were associated with reduced theta-frequency activity (Hong et al., 2012; Javitt et al., 2000; Kaser et al., 2013), which reflect dysfunction in auditory cortex and somatomotor networks. Moreover, earlier report has confirmed that MMN of varied deviant types (frequency, duration, intensity) occurred primarily in the theta frequency range (Javitt et al., 2018). In our study, we also found both frequency and duration MMN showed diminished θ oscillation to deviant stimuli, with fMMN demonstrating deficits in frontal area and dMMN showing alterations located in right temporal region. Further, attenuated θ band of fMMN showed negative relationship with P3 scores of PANSS in SZs. In addition to deficit of θ oscillation, we also discovered post-treatment effects on β oscillation across frontal and bilateral temporal areas of interest in AVH patients. Though β oscillation was comparable between two groups at baseline, patients showed remarkable decline in β oscillation related to both dMMN and fMMN deviants after treatment. Converging findings suggest that delta-theta band activity may give rise to a response (ERPs) to predicted errors, while the process of predictions is transported by alpha and beta activity (Arnal and Giraud, 2012; Chao et al., 2018). Specifically, anticipating and detecting sensory events usually reset the slow phase, namely delta-theta (2–8 Hz) activity, before the stimulus occurs. Meanwhile, beta activity is suppressed after the presentation of the sound and resynchronizes later when the next expected sound occurs (Arnal and Giraud, 2012). The interact between low and mid-frequency oscillations (delta-theta and beta ranges) during temporal expectations indicates a functional cooperation between these oscillations in predictive timing of sensorimotor systems (Cravo et al., 2011; Saleh et al., 2010). Our spectral results, the decrease in θ band and its association with PANSS P3 score, indicate that AVH patients may be influenced by internal speech and fail to recruit sufficient neural resources to maintain the 'predictive' function of MMN.

As for β oscillation, recent researchers have linked its elevation at motor and temporal lobes to higher frequency of hallucination in case report (Kumar et al., 2014) and cross-species (patients with schizophrenia and rhesus monkeys) EEG study (Ma et al., 2024). Though cognitive process involved top-down modulations of attention and expectation suggest that beta-band activity seems related to the maintenance of the current sensorimotor or cognitive state (Engel and Fries, 2010), our findings may suggest that β oscillation could be related to maintenance of AVH status quo during acoustic MMN processing. And a significant decrease of β-band oscillation after treatment probably indicates the relief of attention to AVH symptom afterwards. The reason we failed to identify the presence of increased β-band oscillation among patients might be that the study was not designed to capture AVH symptoms. Another noteworthy finding of current study is that left temporal β band of dMMN predicts amelioration of PANSS negative scores in schizophrenia. Previous study on large-scale cohorts of schizophrenia outpatients reported reduced MMN amplitude uniquely associated with negative symptoms and lower verbal learning (Koshiyama et al., 2021). Longitudinal research also indicates reduced MMN amplitude could predict not only auditory hallucination, but also cognition and negative symptoms of schizophrenia after 5-year follow-up (Donaldson et al., 2023). Above evidence implicate a potential neuropathway, characterized by altered MMN, might be shared among a cluster of symptoms in schizophrenia, including AVH, cognitive and negative symptoms. The neurobiological connections should be further investigated among above symptoms, especially for those patients with treatment-resistant difficulty.

Prior work in resting-state electroencephalogram indicated that ratio between slow- and fast-wave activity reflected cortical and subcortical neural dynamics related to cognitive and emotional processing (Knyazev, 2007). Among all the ratios of fast and slow frequency spectrum bands, TBR was proved to be correlated with attention control (Angelidis et al., 2016; Putman et al., 2010). In our study, healthy participants exhibited higher level of TBR to deviant stimuli compared to SZs with AVH, and patient group showed significant improvement in TBR after treatment. Their neural responses to deviant stimuli might differ because of varied attentional control processes. Previous study showed resting-state TBR at frontal and parietal regions were inversely associated with distraction tendency in healthy individuals (Kobayashi

et al., 2020). Another MMN research comparing different pitch sequences of deviant stimuli reported that higher TBR was associated with shorter MMN latency in the ascending pitch sequences (Hao et al., 2019). Increased level of TBR might act as a trait in patients with ADHD or anxiety disorders (Arns et al., 2013; Derakshan et al., 2009), suggesting more susceptible to distraction. However, in terms of MMN paradigm, higher TBR could represent more sensitivity to unexpected changes during unattended situations. Therefore, we speculate that lower TBR in SZs with AVH indicates less flexibility in attentional shifts, which is coherent with the theory of imbalanced competition of neural resources between external and internal auditory context during hallucinations. Further, our follow-up results also demonstrated that attentional control of SZs may be ameliorated after a short-term treatment.

5. Limitation

There are several limitations in our study. First, we recruited only schizophrenic participants with AVH in current research. Though evidence suggested that patients without AVH showed relatively intact MMN amplitude compared to hallucinating patients, we still need to validate the contribution of θ, β oscillation in relation to AVH symptoms by comparing above subgroups of SZs in future research. Second, the sample size is relatively small, especially for the follow-up data. There is heterogeneity in the sample, which included both acutely first-episode patients and recurrent patients at chronic stages. Therefore, the conclusion should be carefully considered with the confounding variables such as different disease phases, drug influence, and illness durations, etc. Third, it is acknowledged that EEG signals extracted from electrodes positioned near a particular brain region may not faithfully represent the underlying neuronal activity, which underscores an inherent constraint in electroencephalographic investigations. In the future, we also hope to utilize more spatially sensitive methods, such as magnetic resonance imaging (MRI) and magnetoencephalography (MEG), to conduct further studies. Another limitation is that we did not collect AVH status of patients during MMN measurement, making it impossible to define whether the altered indicators are state-related or trait-related to AVH symptom. Future studies using design of AVH capturing paradigm could further validate and explore our findings. Finally, psychological measurements focusing on attention capacity should be used to further illustrate the neuropsychological mechanism underlying attentional deficits during MMN process among SZs with AVH.

6. Conclusions

Summarizing, our study observed attenuated dMMN amplitudes and reduced θ oscillation, TBR of fMMN and dMMN in SZs with AVH. Baseline θ oscillation and TBR of fMMN negatively predicted AVH severity. Additionally, dMMN amplitudes, as well as the TBR of fMMN and dMMN partially recovered after 8-week treatment. Findings of current research support that deficits in θ oscillation and TBR during auditory attention process were crucial to the clinical progression of AVH symptom in schizophrenia and may act as potential biomarkers for treatment effect.

CRediT authorship contribution statement

Qian Guo: Writing – original draft, Visualization, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Zexin Zhao:** Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Wenzheng Wang:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Xiaonan Hu:** Writing – review & editing. **Hao Hu:** Writing – review & editing, Investigation, Conceptualization. **Yao Hu:** Writing – review & editing, Investigation, Conceptualization. **Lihua Xu:** Writing – review & editing, Investigation, Conceptualization. **Xu Liu:** Writing – review & editing, Investigation, Conceptualization. **Xiaohua Liu:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Guanjun Li:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Zhongying Shi:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Jijun Wang:** Writing – review & editing, Supervision, Project administration, Conceptualization.

Ethical statement

The authors claimed that all procedures during this work were in accordance with the ethical standards of human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Declaration of competing interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scog.2025.100344.

Data availability

The data used in this study are available from the corresponding authors upon reasonable request.

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