

Attenuated post-movement beta rebound reflects psychomotor alterations in major depressive disorder during a simple visuomotor task: a MEG study

Yi Xia

the Affiliated Brain Hospital of Nanjing Medical University

Lingling Hua

the Affiliated Brain Hospital of Nanjing Medical University

Zhongpeng Dai

Southeast University

Yinglin Han

the Affiliated Brain Hospital of Nanjing Medical University

Yishan Du

the Affiliated Brain Hospital of Nanjing Medical University

Shuai Zhao

the Affiliated Brain Hospital of Nanjing Medical University

Hongliang Zhou

the Affiliated Brain Hospital of Nanjing Medical University

Xiaoqin Wang

the Affiliated Brain Hospital of Nanjing Medical University

Rui Yan

the Affiliated Brain Hospital of Nanjing Medical University

Xumiao Wang

the Affiliated Brain Hospital of Nanjing Medical University

HaoWen Zou

Medical School of Nanjing University

Hao Sun

Medical School of Nanjing University

YingHong Huang

Medical School of Nanjing University

ZhiJian Yao (✉ zjyao@nju.edu.cn)

the Affiliated Brain Hospital of Nanjing Medical University

Qing Lu

Southeast University

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Abstract

Background

Psychomotor alterations are a common symptom in patients with major depressive disorder (MDD). The primary motor cortex (M1) plays a vital role in the mechanism of psychomotor alterations. Post-movement beta rebound (PMBR) in the sensorimotor cortex is abnormal in patients with motor abnormalities. However, the changes in M1 beta rebound in patients with MDD remain unclear. This study aimed to primarily explore the relationship between psychomotor alterations and PMBR in MDD.

Methods

132 subjects were enrolled in the study, comprising 65 healthy controls (HCs) and 67 MDD patients. All participants performed a simple right-hand visuomotor task during MEG scanning. PMBR was measured in the left M1 at the source reconstruction level with the time-frequency analysis method. Retardation factor scores and neurocognitive test performance, including the Digit Symbol Substitution Test (DSST), the Making Test Part A (TMT-A), and the Verbal Fluency Test (VFT), were used to measure psychomotor functions. Pearson correlation analyses were used to assess relationships between PMBR and psychomotor alterations in MDD.

Results

The MDD group showed worse neurocognitive performance than the HC group in all three neurocognitive tests. The PMBR was diminished in patients with MDD compared to HCs. In a group of MDD patients, the reduced PMBR was negatively correlated with retardation factor scores. Further, there was a positive correlation between the PMBR and DSST scores. PMBR is negatively associated with the TMT-A scores.

Conclusion

Our findings suggested that the attenuated PMBR in M1 could illustrate the psychomotor disturbance in MDD, possibly contributing to clinical psychomotor symptoms and deficits of cognitive functions.

Background

Major depressive disorder (MDD) is not only characterised by abnormalities in mood and affect but also substantial disturbance in cognition and psychomotor function. Psychomotor alterations have been recognised as one of the crucial features of MDD [1]. The term psychomotor is composed of two parts: "psycho" and "motor," with the former referring to cognitive processes and the latter to motor processes [2]. Psychomotor functions are the foundation of the plan and execution of various behaviour, including speech, facial expression, and cognitive function [3]. Some findings showed that one aspect of

psychomotor alterations, namely psychomotor retardation (PMR), is related to the severity of depression [4] and poor response to antidepressants [5], ultimately contributing to impaired social function and quality of life [6]. However, the neural processes underlying psychomotor alterations in MDD are mainly unknown. Exploring the mechanisms that underpin psychomotor alterations may aid in further understanding the neurological disturbance in MDD.

Psychomotor disturbance has been frequently reported in psychiatric disorders like MDD, bipolar disorder (BD), and schizophrenia (SCH) [2]. Previous work has primarily probed the pathophysiology of psychomotor alterations in mood disorders [7]. It has been suggested that the mania state in mood disorders is associated with psychomotor excitation, while the depressive state is mainly related to psychomotor inhibition [8]. The psychomotor function is modulated by the sensorimotor network [9]. It is suggested that the psychomotor alterations in patients with MDD were correlated with neural circuitry involving the prefrontal cortex, motor-related cortex and basal ganglia [1]. In particular, psychomotor retardation was due to the alteration of cerebellar-thalamo-cortical motor circuitry [10]. Existing functional imaging studies have demonstrated that psychomotor alterations are related to decreased blood flow in the dorsolateral prefrontal cortex, left prefrontal cortex, angular gyrus, and the anterior cingulate [11-13]. Furthermore, structural imaging studies revealed that psychomotor speed is associated with alterations of white matter in motor-related pathways [14-16]. A study discovered that aberrant cerebral blood flow (CBF) in the centre of the motor-regulating circuit might account for the PMR in MDD using a perfusion MRI approach [17]. As Northoff et al. [2] suggested, the motor cortex plays a core role in psychomotor alterations. Therefore, we primarily focused on the role of the primary motor cortex (M1) in the pathophysiology of the psychomotor alterations in MDD.

Given that motor-related areas played a crucial role in movement-related activity [18], we primarily investigated the psychomotor alterations in MDD while performing a simple visuomotor task. A well-characterised neurophysiological feature associated with movement is beta bands (Fig. 1) [19]. It has been reported that a power reduction in beta bands across sensorimotor regions is present before movement onset and during movement execution, i.e., movement-related beta reduction (MRBD) [20]. Following the end of the movement, post-movement beta rebound (PMBR) occurs, defined as an increase in beta amplitude relative to baseline [21]. This response typically occurs 500ms after movement termination and lasts for several seconds (Fig. 1A). Previous studies have suggested that the PMBR originates in the sensorimotor network [19], particularly M1 [22]. There is evidence that MRBD and PMBR may originate from different brain regions. Specifically, MRBD is usually located in the postcentral gyrus, while PMBR locates in the precentral gyrus (Fig. 1B) [22, 23]. Moreover, the two stage-dependent alterations may play different roles in sensorimotor processes. As PMBR is well-accepted to be involved in motor inhibition [24] or sensory feedback [25] in motor cortices, it could regulate cortical sensory processing and motor output [26], which further supports the notion that modulation of sensorimotor could access to investigate control or executive process [18]. PMBR may contribute to visuomotor adaption by maintaining or updating the forward model that guides movement [27]. Research in healthy individuals demonstrated that the decrease of PMBR leads to a slower movement state [28], consistent with reduced movement-related beta activity in Parkinson's disease (PD) [29]. Many physiological or

pathological factors influence beta oscillations, such as healthy ageing [30-39]. Previous studies found that poor motor performance in healthy older adults is related to changes in beta oscillations. Importantly, neuropsychiatric studies also revealed that decreased PMBR in PD [40-42], SCH [43-45], schizotypal personality disorder (SPD) [46], autism spectrum disorder (ASD) [47-49], reflecting impaired sensorimotor integration in psychiatric disease. In general, these findings indicated that the aberrant PMBR impacts motor-related activity and advanced sensorimotor integration. However, the alteration of PMBR and its association with psychomotor disturbance in MDD remains underexplored.

The psychomotor symptoms could be rated by the items related to exhaustion, loss of energy, or attention in the 17-item Hamilton Rating Scale for Depression (HAMD₁₇) [50]. In addition, many neuropsychological tests can objectively measure psychomotor, involving drawing tasks and speech measures. The Trail Making Test Part A (TMT-A), in which participants must link 25 circles with numbers in ascending order, was originally intended to be applied to test processing speed [51]; The Digit Symbol Substitution Test (DSST) measures both cognitive and motor elements of psychomotor since it includes thinking and graphomotor ability [52]; The Verbal Fluency Test (VFT) examines mental processing speed by asking participants to speak certain types of words [53]. These tests can accurately quantify psychomotor changes in MDD patients. In this study, we investigated the PMBR activity in M1 and its potential association with psychomotor alterations in MDD during a visuomotor task using magnetoencephalography (MEG) techniques. Based on previous results in SCH and ASD, we hypothesised that patients with MDD have attenuated PMBR, and this change could reflect a psychomotor slowing in clinical symptoms and cognitive functions.

Methods

Study design

This study adopted a cross-sectional case-control study design. Data, including demographic data, clinical assessments, neurocognitive assessments, MEG scan data and MRI scan data, were collected from patients with MDD and healthy controls (HCs).

Participants

The MDD patients were recruited from the Affiliated Brain Hospital of Nanjing Medical University between 2012 and 2021. All patients met the diagnostic criteria of major depressive disorder using the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition, Text Revision (DSM-IV-TR). The inclusion criteria for this population were: (1) aged 18-50 years; (2) right-handed; (3) native Han Chinese; (4) education level of junior high school or above; (3) Hamilton Rating Scale for Depression 17-item (HAMD₁₇) >17; (4) Young Mania Rating Scale (YMRS) [54] score < 5; (5) no structured psychotherapy or physical therapy within six months. The exclusion criteria included comorbidity with other mental or physical illnesses, history of substance abuse, contraindications for MEG or MRI and pregnancy or lactation.

One hundred thirty-two individuals, comprising 65 healthy controls and 67 MDD patients, were enrolled in this study. Participants were asked to perform a simple visuomotor task during MEG scanning. This work was approved by the Affiliated Brain Hospital of Nanjing Medical University. It was performed in accordance with the Declaration of Helsinki, and all individuals provided written informed permission before participation.

Clinical assessments

We collected general participant information, such as sex, age, education, ethnicity, dominant hand, disease history, marriage and family history. Depression severity was assessed using HAMD₁₇, and manic symptoms were evaluated by YMRS. Retardation factor score was obtained using 1, 7, 8 and 14 items in HAMD₁₇.

Neurocognitive assessments

Based on previous work, psychomotor alterations could be measured by neuropsychological tests [7]. In this study, psychomotor performance was measured by three different neuropsychological tasks: 1) DSST, which assesses psychomotor processing speed by instructing subjects to fill in as many blanks as possible within 90s according to the number symbol correspondence table. Test performance is evaluated by the number of successful completions; 2) TMT-A, a test in which individuals are directed to draw lines through consecutive numbers to determine the time needed to complete the test; and 3) VFT, a task in which participants are asked to name as many animals or vegetables as possible in 60s.

MEG scanning

Subjects were placed in a magnetically shielded room during the entire experiment. Data were collected using a 275-channel CTF system (VSM Med Tech Inc., Port Coquitlam, Canada). The sampling rate is 1,200 Hz. The participants were lying in the MEG machine during a visuomotor task. Three coils, one nasion and two preauricular points were used to check head movements during the recording. Electrocardiography and electrooculography were also recorded. The experiment consists of two blocks, each lasting 5 min. Participants can take a small break between blocks.

Task Paradigm

Participants were asked to perform a visuomotor task during MEG scanning. Participants responded with a right index finger button press to visual stimuli. Fig. 2 shows an illustration of this paradigm. During each trial, grey light was exhibited on a projection screen for 2,500 milliseconds, then a green light lasting 500 milliseconds. Subjects responded with a right index finger button press to visual stimuli. Individuals were asked to concentrate on the projection screen, keep their bodies still, and press the button as quickly as possible. Participants first completed a practice examination to understand the task, followed by a formal exam including 180 trials.

MRI scanning

MRI data were recorded with a Siemens Verio 3T MRI system. The parameters were the same as those used in our earlier articles [55]. Three markers were put in the same location as MEG to facilitate registration between MEG data and structural MRI in the following data analysis.

Data processing

The entire data processing process is illustrated in Fig. 3. The raw data were epoched into a time window (-0.7 to 2.3 s). The 0s represent the emergence of a visual cue or green light. The data was down-sampled to 400 Hz. Then we applied Synthetic third-order gradiometer noise cancellation and removed linear trend and line interference with a 50-Hz band-stop filter. Visual artifact rejection was used to exclude trials and channels with high variation. Furthermore, with a visual examination, an independent component analysis (ICA) was used to eliminate eye artifacts, as well as cardiac and muscular components.

We analysed the MEG data in FieldTrip (version 20210720)[56]. The structural T1 data was imported and segmented using FieldTrip, with 250 anatomical volumes in each direction and “singleshell”.

Beamforming and time course extraction

As PMBR is located in the contralateral M1, we want to extract the beta time series of the M1 in this right finger tap task. Using the linearly constrained minimum variance (LCMV) beamformer [57], we can compute virtual channel time series at the locations of interest. The coordinates of the M1 were defined by the Automated Anatomical Labeling (AAL) template atlas [58]. The spatial filter was computed with the covariance and the forward model for the left M1. Using the spatial filters, virtual channel time series were obtained.

Time-Frequency spectrograms

Using a Hanning taper-based time window of four cycles at each frequency, the source-level time-frequency representation of the time series of the M1 may be obtained (1–100 Hz in steps of 1 Hz). We compared the active energy to the baseline using relative energy percentage. The baseline was defined between the -0.5–0 s window relative to the appearance of green light. The time-frequency spectrograms were averaged across participants in both groups.

PMBR quantification

We averaged mid-beta band (17–25Hz) amplitude from the data baseline adjusted in time windows (1 < t < 2s) acceptable for the PMBR. Eventually, we obtained a value per participant representing the PMBR at the left M1.

Statistical analyses

A two-sample t-test was used to check age and education, whereas chi-square tests were conducted to compare gender between the MDD patients and healthy controls. All statistical analyses were done on SPSS 19.0 software (IBM Corp., Armonk, NY, USA). The differences in neurocognitive test performance

(DSST, TMT-A and VFT) between the MDD and HC groups were compared by a two-sample t-test (two-tailed). The difference in PMBR amplitude between groups was analysed using a two-sample t-test. We then computed correlations between PMBR values and retardation factor, neurocognitive tests performance (DSST, TMT-A and VFT), controlling for age, sex and education. To avoid multiple comparison problems, further FDR correction was conducted using a special MATLAB function.

Results

Demographics and clinical characteristics

Sixty-seven MDD patients and sixty-five HCs were enrolled in the study. There was no significant difference in sex, age or education between the two groups. HAMD₁₇ total score and retardation factor score were recorded for patients only.

Neurocognitive tests performance

The MDD group showed worse neurocognitive test performance than controls in all three tasks (Table 2). Statistical analyses revealed significant differences in DSST ($t = 3.05, p = 0.003$), TMT-A ($t = -3.38, p = 0.001$), and VFT ($t = 3.13, p = 0.003$).

Group differences of PMBR

Fig. 4A shows the time-frequency representations of the left M1 for the MDD and HC groups. We observed that PMBR appeared following MRBD in the HC group, which has been well-established by the work of others. The MDD group experienced a similar but weaker response compared to the HC group. The graph shows that beta amplitude increases around 1s following the visual stimulus. The PMBR value in the MDD group was lower than that in the HC group ($t = 3.70, p < 0.001$) (Table 2, Fig. 4B).

Association between PMBR and clinical assessments

In the MDD group, a two-tailed Pearson's partial correlation was performed to compare the PMBR values with retardation factor scores. Psychomotor alterations are reflective of items relating to exhaustion, energy loss, or lack of attention in HAMD₁₇. So, the retardation factor in HAMD₁₇ can partially represent psychomotor alterations. In MDD patients, we particularly focused on the retardation factor scores, and we found that the retardation scores were inversely correlated with PMBR ($r = -0.365, p = 0.002$). Moreover, given that healthy aging has an impact on beta oscillations[30-39], the correlations remained significant ($r = -0.374, p = 0.006$, FDR corrected) after controlling sex, age and education (Fig. 5A).

Association between PMBR and neurocognitive assessments

In the MDD group, partial correlation analysis revealed a significant positive correlation between the DSST scores and PMBR values ($r = 0.411, p = 0.012$, FDR corrected). Moreover, the TMT-A scores were negatively associated with PMBR ($r = -0.451, p = 0.003$, FDR corrected). The VFT scores were positively

correlated with PMBR ($r = 0.348$, $p = 0.022$). However, this effect is no longer significant after controlling sex, age and education, and performing FDR correction ($r = 0.348$, $p = 0.06$, FDR corrected). Fig. 5B and Fig. 5C visualise the relationship between neurocognitive assessments and PMBR amplitude. There was a significant negative correlation between age and PMBR in HCs ($r = -0.300$, $p = 0.015$). However, age was not significantly correlated with PMBR in MDD patients.

Discussion

In this study, we primarily explored the alteration of motor-related oscillatory activity to assess the psychomotor alterations in patients with MDD by utilising clinical assessments, neurocognitive tests and a simple visuomotor task during MEG recording. More Specifically, MDD patients had slower psychomotor speed and attenuated PMBR activity than HCs. Furthermore, there was an association between psychomotor alterations and PMBR values in the MDD group. This supported our earlier hypothesis that the strength of motor-related cortical oscillations impacts slowing mental and motor processes in MDD. In addition, there was a significant negative correlation between age and PMBR in HCs.

Compared to the previous study, we explored the PMBR activity in M1. In line with earlier studies that MDD patients have diminished sensorimotor cortex activity [2], we found that MDD patients had lower PMBR in M1 at the end of movement than HCs in our visuomotor MEG study. Several explanations have been put forward to interpret PMBR. One of the theories is that PMBR is generated by the peripheral electrical stimulation of afferent pathways or somatosensory stimuli [59, 60], indicating afferent sensory feedback on completion of the movement [25]. The somatosensory system is not only responsible for afferent input information but also actively communicates with the motor cortex during movement [61]. Poor sensory feedback may cause major problems with sensorimotor integration and reduce the accuracy of feedforward predictions[62]. The afferent sensory feedback could be applied to evaluate movement outcome, possibly impacting the following processing speed. The evidence from healthy individuals revealed that a “slowed movement state” displayed weaker sensorimotor beta dynamics [28] compared to fast movement [63]. More evidence has been provided that patients with motor abnormalities or processing speed slowing displayed attenuated PMBR. For example, Robson et al. found aberrant PMBR activity in SCH [44], as well as ASD [47–49] and PD [40–42]. The evidence suggests that the reduced PMBR activity is characteristic of patients with aberrant psychomotor performance. Furthermore, we also found that the PMBR activity is negatively associated with retardation factor scores in HAMD₁₇, indicating that the lower retardation score in MDD displayed more beta activity in M1. The retardation factor in HAMD₁₇ also reflects psychomotor function [3], suggesting that retardation symptoms result from the dysconnectivity of motor-related brain areas in MDD [64, 65]. Northoff et al. aggregated available evidence in healthy subjects, MDD, BD and SCH, proposing three psychomotor mechanisms, including subcortical modulation of motor circuit, cortical modulation of motor circuit and balance between motor cortex activity and global cortical activity [2]. It is worth noting that all mechanisms lead to the motor cortex. Similarly, the reduced PMBR was related to the severity of mental

disorganisation and impoverishment in SPD, which was associated with thought processing speed [46]. Collectively, these results suggested that the reduced PMBR activity in M1 is possibly one of the key nodes to illustrate the psychomotor alterations in MDD.

Moreover, our study also found that PMBR activity was associated with cognitive function in MDD. In our research, MDD patients performed worse on all three neurocognitive tests compared to HCs. Our findings are congruent with a prior meta-analysis, showing that MDD patients report impairments in motor speed, processing speed, executive functioning in tests with time limitations, and verbal fluency [66]. Consistently, the “time to peak” of the PMBR is associated with the DSST performance in multiple sclerosis (MS) patients. One more study observed that PMBR was significantly correlated with DSST scores in patients with schizophrenia [45], providing strong evidence that PMBR activity could reflect information processing speed [67]. During DSST or TMT-A performance, the psychomotor is engaged in visual processing, cognitive processing, motor planning, and movement execution stages. This is in line with the notion that objective measurements of psychomotor speed on neurocognitive tasks could quantify psychomotor alterations [68, 69]. Thus, it is plausible to observe that the PMBR was highly linked to objective measures of psychomotor alterations via cognitive function (DSST and TMT-A). Abnormal PMBR may represent aberrant sensory feedback. Adults with MDD may have less assurance in the feedforward predictions. This may explain the relationship between PMR and aberrant PMBR observed in this study. In addition, a reduced PMBR may reflect poor inhibition of motor cortex after movement termination. This phenomenon could also affect psychomotor performance in MDD patients.

Limitations

Of note, there are some limitations in this study. First, scales to measure psychomotor alterations in MDD should be used in future studies, such as the Motor Agitation and Retardation Scale (MARS), the CORE index of melancholia, and the Salpetriere Retardation Rating Scale (SRRS) [70–72]. Second, PMBR is modulated by task duration [73] and is related to contraction force [74] and movement duration [75]. It will be better to control these factors in future studies. Thirdly, the current study only focused on M1. The impairments of cognitive function and clinical symptoms in MDD are caused not only by motor cortex dysfunction, but also by the coordination of distant brain areas. We will further explore the long-distance communication between brain regions in the next step.

Conclusion

Overall, our present MEG study revealed that PMBR is diminished in patients with MDD and is associated with psychomotor alterations. The current results provided electrophysiological evidence that the reduced PMBR activity contributes to clinical symptoms and cognitive functions in MDD, clarifying the closed link between PMBR and psychomotor alterations. From the electrophysiological perspective, our findings deepen the understanding of the role of M1 in psychomotor alterations in MDD. As the neurochemical mechanism of psychomotor retardation could be due to the alteration of GABA (Gamma-aminobutyric acid) in the motor-related cortex [76–78], cognitive remediation and neuromodulatory techniques such as

TMS (Transcranial magnetic stimulation) could be practical to interfere with the psychomotor alterations in MDD and improve the clinical outcome. Further research should continue to focus on psychomotor symptoms in MDD and the underlying dynamic activities in the brain.

Abbreviations

MDD: Major depressive disorder; PMR: psychomotor retardation; BD: Bipolar disorder; SCH: Schizophrenia; CBF: cerebral blood flow; M1: primary motor cortex ; MRBD: Movement-related beta reduction; PMBR: Post-movement beta rebound; PD: Parkinson's disease; SPD: Schizotypal personality disorder; ASD: Autism spectrum disorder; HAMD₁₇: 17-item Hamilton Rating Scale for Depression; TMT-A: Trail Making Test Part A; DSST: Digit Symbol Substitution Test; VFT: Verbal Fluency Test; MEG: Magnetoencephalography; HCs: Healthy controls; DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders Fourth Edition, Text Revision; YMRS: Young Mania Rating Scale; LCMV: Linearly constrained minimum variance; AAL: Automated Anatomical Labeling; MS: Multiple sclerosis; MARS: Motor Agitation and Retardation Scale; SRRS: Salpetriere Retardation Rating Scale; GABA: Gamma-aminobutyric acid; TMS: Transcranial magnetic stimulation;

Declarations

Ethics approval and consent to participate

This study was approved by the Medical Ethics Committee of the Affiliated Brain Hospital of Nanjing Medical University (No.2011-KY027). All the research procedures involving human participants followed the Declaration of Helsinki in 1964 and its subsequent amendments. Written informed consent was obtained from all participants and a parent.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated during the current study are not publicly available due to the subjects' privacy, but are available from the corresponding author on reasonable request.

Competing interests

The authors declare no competing interests.

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Authors' contributions

ZhiJian Yao and Qing Lu conceived the study, drafted the protocol, and revised the manuscript. Yi Xia and Lingling Hua managed the literature research, analysed the results and wrote the first draft of the manuscript. Yi Xia and Lingling Hua contributed equally to this work. Zhongpeng Dai provided data processing ideas. Yinglin Han, Yishan Du, Shuai Zhao and Hongliang Zhou collected experimental data. Xiaoqin Wang, Rui Yan and HaoWen Zou prepared the tables. Xumiao Wang, Hao Sun and YingHong Huang prepared the figures. All authors reviewed the manuscript and have approved the final manuscript.

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Author details

¹Department of Psychiatry, the Affiliated Brain Hospital of Nanjing Medical University, Nanjing 210029, China. ²School of Biological Sciences & Medical Engineering, Southeast University, Nanjing 210096, China. ³Child Development and Learning Science, Key Laboratory of Ministry of Education, Southeast University, Nanjing 210096, China. ⁴Nanjing Brain Hospital, Medical School of Nanjing University, Nanjing 210093, China.

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Tables

Table 1.

Subjects' demographic and clinical characteristics.

Variables	MDD (n=67)	HC(n=65)	<i>t/χ</i>	<i>p</i>
Sex(M/F)	33/34	34/31	0.123 ^a	0.726 ^a
Age, y	30.19±10.24	29.22±6.48	-0.658 ^b	0.512 ^b
Education, y	13.90±2.79	14.22±2.17	0.737 ^b	0.462 ^b
Handedness (R/L)	67/0	65/0	-	-
HAMD-17 Total score	24.10±5.58	-	-	_ ^c
Retardation	7.73±1.86	-	-	-

Abbreviations: MDD, major depressive disorder; HC, health control; HAMD, Hamilton Depression Rating Scale;

^a The χ/p value was obtained by two-tailed Pearson chi-square test.

^b Data were presented as the range of minimum-maximum (mean ±SD). The t/p value was obtained by two-sample two-tailed t test.

Table 2.

Neurocognitive test performance and PMBR.

	MDD	HC	<i>t</i>	<i>p</i>
DSST	51.91±11.21	58.41±5.60	3.05	0.003
TMT-A	36.40±12.71	26.21±8.60	-3.38	0.001
VFT	18.56±4.99	22.91±5.89	3.13	0.003
PMBR	0.08±0.07	0.14±0.12	3.70	0.000

DSST: Digit Symbol Substitution Test; TMT-A: Trail Making Test A; VFT: Verbal Fluency Test;

Two-sample t-test, two-sided, alpha-level 0.05.

Figures

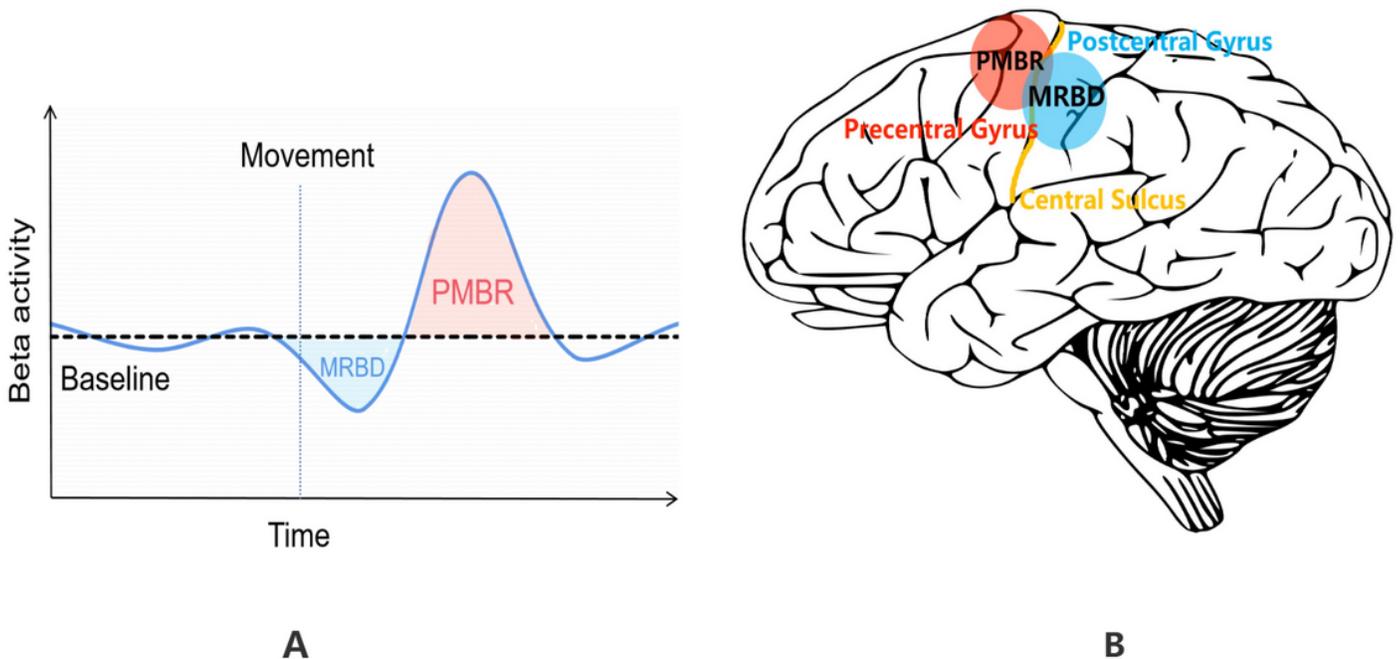


Figure 1

Illustration of MRBD and PMBR. (A) Schematic representation of sensorimotor beta activity. Beta activity reduces (MRBD—blue shaded region) right before and during movement execution. After the end of movement, beta activity rapidly increases (PMBR—red shaded region). Finally, beta activity slowly returns to baseline level. (B) The brain distribution of MRBD and PMBR. MRBD (blue circle) is frequently found in the postcentral gyrus, while PMBR (red circle) is frequently observed in the precentral gyrus.

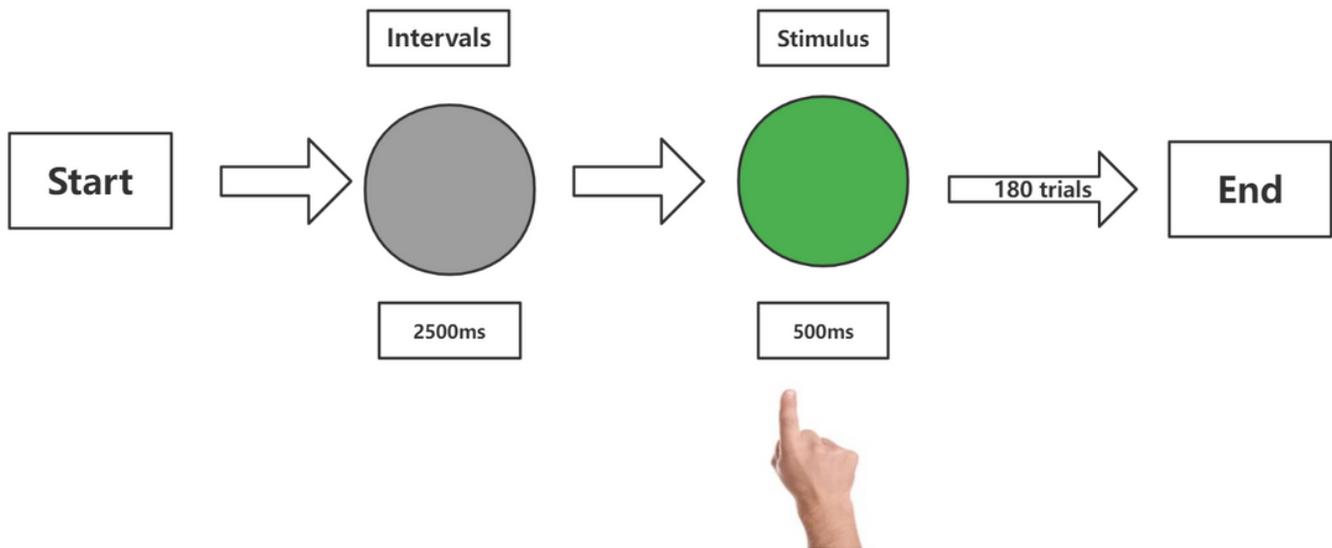


Figure 2

The procedure of the visuomotor experiment. A green light was presented duration of 0.5s. Once the green light appeared, participants were asked to press the button with the right index finger. Green light was followed by an interval of 2.5 s (grey light).

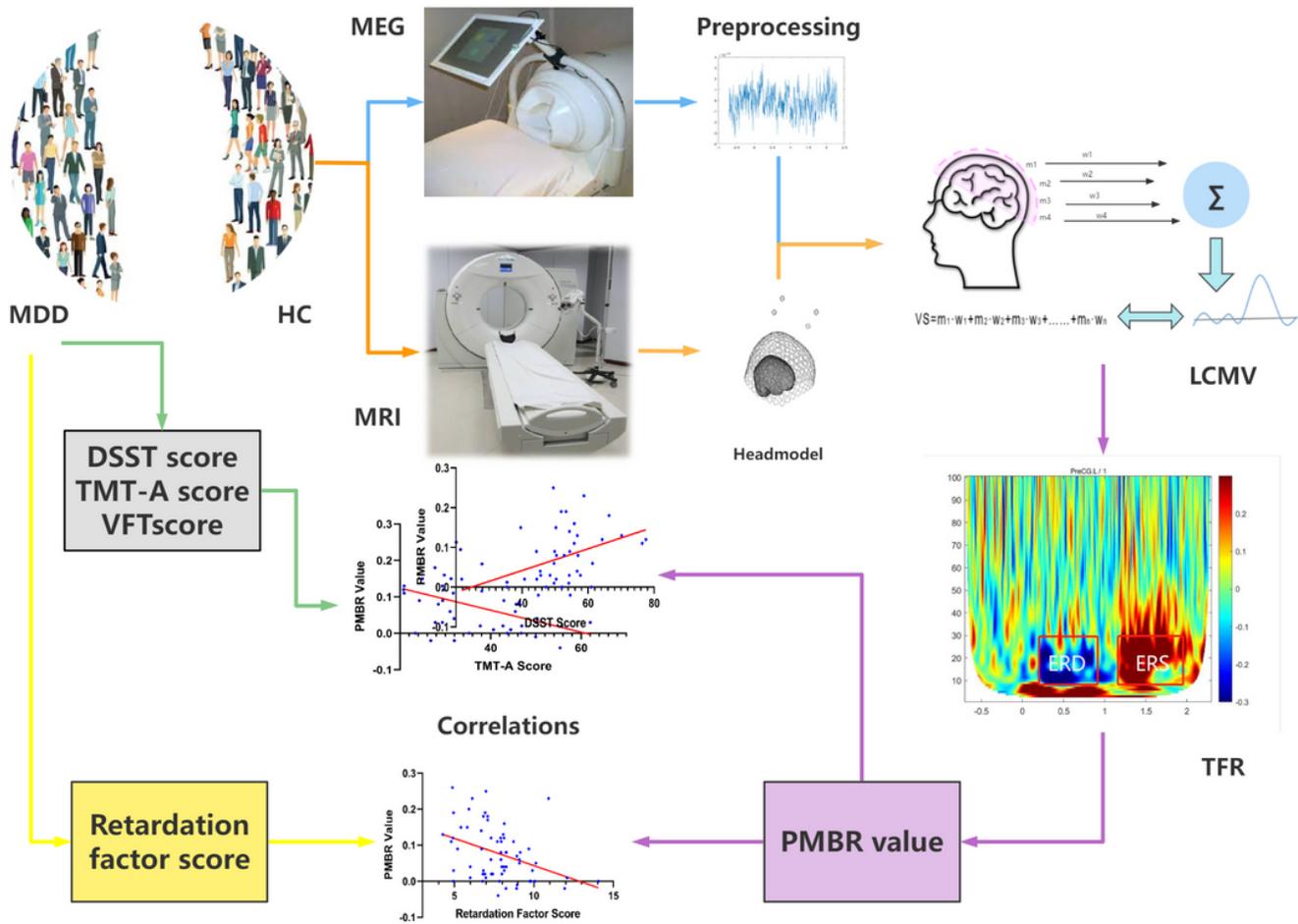


Figure 3

Diagram of the processing flow. For each person, we obtained three types of data, including MRI, MEG, and neurocognitive tests. DSST, TMT-A and VFT are included. For patients with MDD, retardation factor scores were also extracted. MEG data preprocessing contained segmentation, down-sampling, visual artifact rejection and independent component analysis. The headmodels of each participant were obtained by an anatomical MRI scan. Then Using the LCMV beamformer, we then extracted the time course of the left M1 defined via the AAL template atlas. We subsequently calculated the source-level TFR of this virtual sensor timeseries. By averaging the relative power of beta within a time window of interest, a single value was computed to represent the amplitude of PMBR for each participant. Finally, we assessed the relationship between PMBR and retardation factor scores, and neurocognitive task performance.

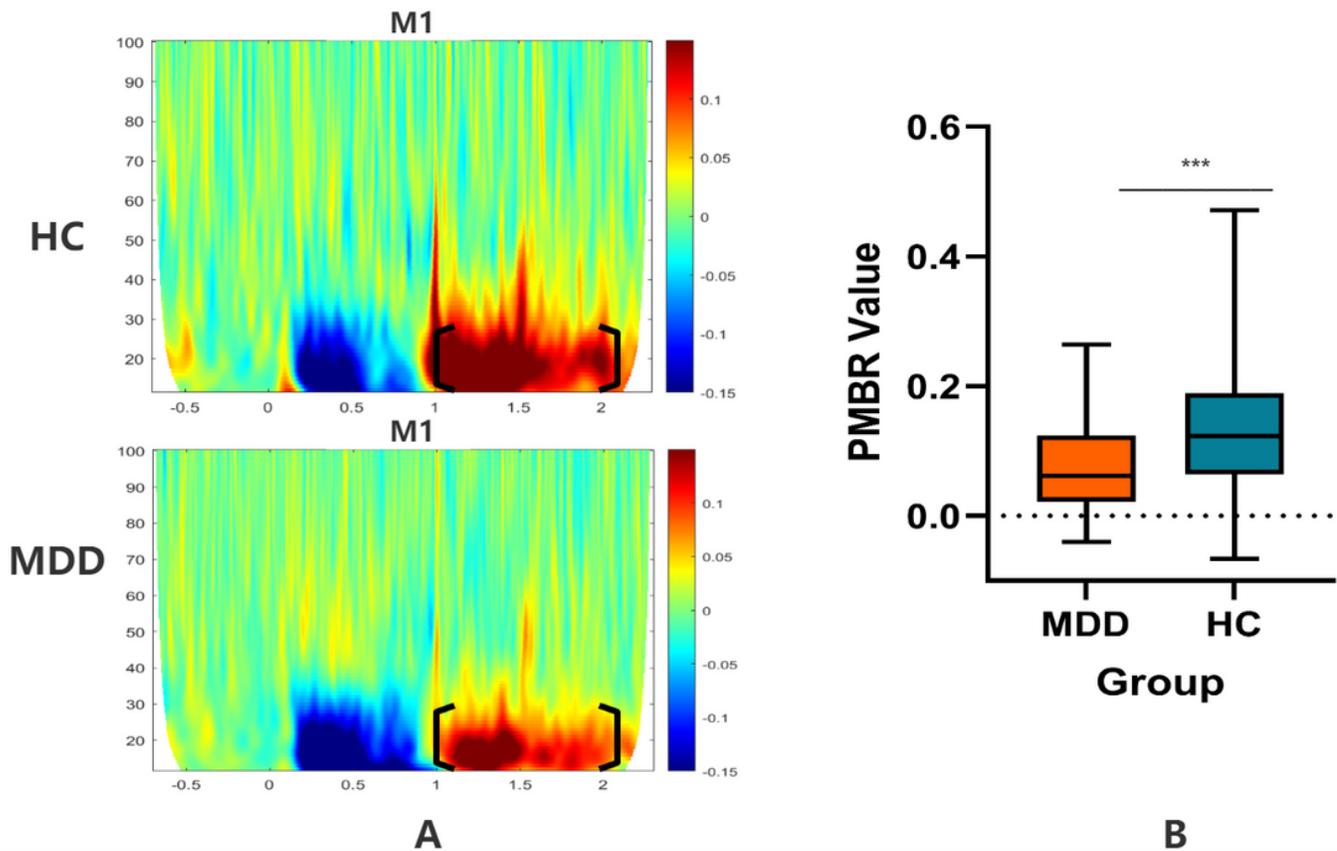
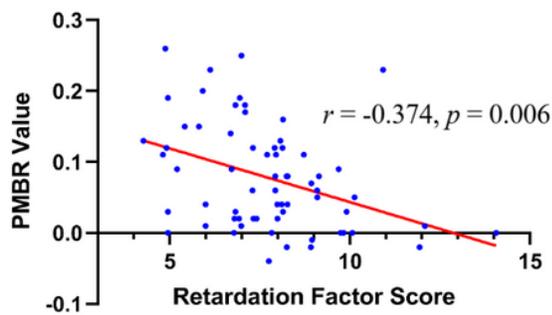
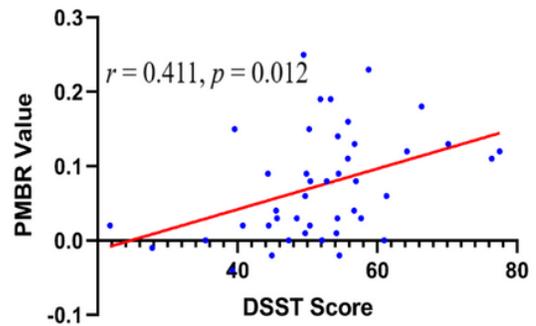


Figure 4

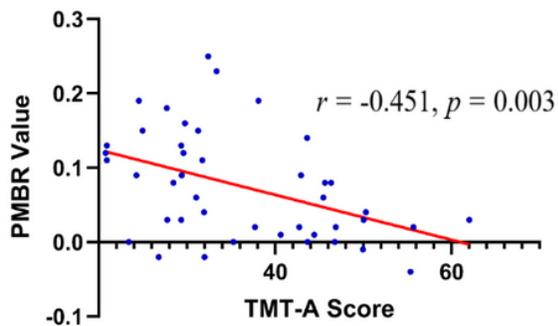
Differences of PMBR. (A) The time–frequency plots of the left M1 for the MDD and HC group. Both figures illustrate that oscillatory activity in the beta band (13–30 Hz) decreases before movement and subsequently rises after movement cessation. The MDD group had a similar but weaker response compare with HCs. According to the graphs, beta energy reduces approximately 0.2s after visual cues and increases around 1s following visual stimuli. The colour scale depicts relative energy changes in comparison to the baseline level (blue colour suggests a drop, while red colour suggests an increase). (B) The PMBR value in MDD and HC group. *** $p < 0.001$



A



B



C

Figure 5

Correlations between PMBR and psychomotor alterations. (A) The PMBR of the left M1 negatively correlated with retardation factor scores. (B) The PMBR of the left M1 had a positive relationship with DSST scores. (C) The PMBR of the left M1 is negatively associated with TMT-A scores.