

Exploring Alpha and Theta Activity in Depression: A Combined Surface EEG and LORETA Study of Cortical and Subcortical Networks

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Abstract

Introduction. Depression is a common mental health condition characterized by disrupted neural activity in cortical and subcortical networks involved in emotion and memory. While alpha and theta oscillations have been linked to depression, their specific roles in symptom domains remain unclear. This study examines these relationships using quantitative EEG (qEEG) and low-resolution electromagnetic tomography analysis (LORETA). **Methods.** Fifty-eight adults with depression underwent resting-state, eyes-closed qEEG. Absolute power and coherence of alpha (8–12 Hz) and theta (4–8 Hz) bands were analyzed across 19 scalp electrodes and hippocampal and amygdala regions using LORETA. Depressive symptom severity was assessed using the Beck Depression Inventory-II (BDI-II). Statistical analyses evaluated associations between EEG parameters and symptom scores. **Results.** Alpha coherence between the left hippocampus and amygdala negatively correlated with somatic symptoms ($r = -0.298$, $p = .027$), explaining 26% of variance in total BDI-II scores. Increased theta coherence in the right frontotemporal network was associated with reductions in affective and somatic symptoms. **Conclusions.** The findings identify neural oscillatory patterns within hippocampal-amygdala and frontotemporal networks as potential biomarkers for depressive symptoms, providing insights into novel therapeutic targets.

Keywords: depression; qEEG; alpha and theta oscillations; hippocampus-amygdala network; frontotemporal area

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Introduction

Depression is a widespread mood disorder that affects over 350 million people globally, significantly contributing to the global disease burden. It is the leading cause of disability worldwide, with a lifetime prevalence of 4.4% in the general population (Friedrich, 2017; Garcia-Batista et al., 2018). Major depressive disorder (MDD) is characterized by symptoms such as persistent low mood, anhedonia, appetite and sleep changes, fatigue, restlessness or slowed movement, feelings of guilt or worthlessness, difficulty concentrating, and suicidal thoughts. According to the DSM-5-TR (American Psychiatric Association, 2022), a diagnosis of MDD requires the

presence of at least five of these symptoms for most of the day, nearly every day, for a minimum of 2 weeks (Cui et al., 2024). Neuroimaging studies, including magnetic resonance imaging (MRI), functional MRI (fMRI), and electroencephalography (EEG), have demonstrated that individuals with depression exhibit both structural abnormalities and functional imbalances within brain networks. These networks are crucial for processes such as emotion regulation, involving regions like the amygdala, subgenual anterior cingulate, caudate, putamen, and pallidum (Siegle et al., 2007), as well as memory, encompassing the hippocampus (HPC), parahippocampal cortex, and other related areas (Dev et al., 2022; Yang et al., 2017). The amygdala

is integral to processing salient stimuli and serves as a central hub within the affective network. Neuroimaging findings indicate increased amygdala connectivity and activation in patients with MDD, alongside reduced overall and subregional resting-state connectivity (Damborská et al., 2020). These abnormalities in the affective network likely contribute to emotional dysregulation (Tang et al., 2018). Another area that has emerged as a critical integrator of emotion and cognition is HPC. Studies have shown reductions in HPC volume across various mood disorders, particularly in MDD (Lorenzetti et al., 2009). HPC plays a crucial role in memory retention and controlling the production of cortisol, a hormone secreted in response to stress. When a person is depressed, his body releases excessive amounts of cortisol, leading to hippocampal atrophy and a reduction in neurogenesis. (Dev et al., 2022). Alongside structural alterations, abnormal HPC functioning has been linked to cognitive impairment and deficits in spatial memory among depressed patients (Gould et al., 2007). Recent functional studies have reported abnormal theta activity in the right anterior HPC and parahippocampal cortices in depressed individuals compared to healthy subjects (Cornwell et al., 2010). Amygdala and HPC are thought to be important for contextual modulation of fear, judgment of emotion, and emotional memory that are critical for remembering motivationally salient stimuli. The coupling between these two regions is predominantly unidirectional, during frequency oscillations; theta and alpha mediate their interregional communication (McGaugh, 2004; Zheng et al., 2017). Abnormal functional connectivity between these two areas, like patterns observed in humans with depression (Gould et al., 2007), has been also documented in a genetic rat model of major depression (Williams et al., 2014). These abnormalities, along with dysfunctions in other regions such as the ventromedial prefrontal cortex, insula, and caudate have been suggested to contribute to the dysregulation of emotional and motivational processes in MDD (Mayberg, 1997).

In resting-state EEG recordings, patients with depression exhibit disrupted connectivity within and between key networks, including the frontotemporal, centroparietal, frontoparietal, and dorsal attention networks, when compared to healthy individuals (16, 10). Elevated beta power in the prefrontal cortex, along with asymmetries in the alpha and theta bands, has been also linked to depressive symptoms (Liu et al., 2024). Machine learning analyses have demonstrated that the right hemisphere exhibits higher accuracy and

performance in detecting depression, and among the various brain wave frequencies, the alpha band has shown the greatest accuracy in the classification of depression (Dev et al., 2022). Frontal alpha asymmetry is a biomarker that measures the balance of alpha wave activity between the left and right hemispheres of the frontal cortex, linked to emotional dysregulation (Tseng et al., 2022). Coherency is another index used in EEG studies to assess functional connectivity between brain regions. It quantifies the phase consistency between two EEG signals over time and frequency. Higher coherency between two regions implies greater functional connectivity, suggesting that these regions are synchronously communicating (Trambaiolli & Biazoli, 2020). In an EEG study on 119 subjects, including 75 healthy subjects and 44 patients with MDD, coherency in the alpha2 band (10–12 Hz) presented significantly positive correlation with symptoms (Trambaiolli & Biazoli, 2020). A machine learning analysis in another study also revealed that patients with MDD exhibited higher functional connectivity compared to controls, particularly in the alpha and beta bands. In the alpha band, connections were linking the frontopolar and DLPFC regions with temporal and parietooccipital areas, while beta band connections were mostly within prefrontal and temporal regions. These connectivity patterns distinguished MDD from bipolar disorder with 81% accuracy (Leuchter et al., 2012). A systematic review of 52 research articles highlighted the significant potential of EEG-based connectivity analysis and brain mapping techniques in identifying biomarkers of depression. The findings consistently identified the frontal cortex and parietal-occipital cortex as critical regions involved in depression detection. The review further emphasized the importance of future research that incorporated larger and more representative sample sizes, along with the application of advanced data analysis methodologies to improve accuracy. It also advocated for the development and use of more precise techniques to localize the brain regions most affected by depression (Dev et al., 2022).

In this study, we tried to address some of these challenges. We examined the alpha and theta absolute power across 19 EEG channels and evaluated their coherence within commonly studied surface networks. To address the limitations of surface EEG, we employed the LORETA technique to estimate these indices—absolute power and coherence—in two critical deep brain regions: the HPC and amygdala. This dual-layered methodology enhances the precision of identifying brain areas implicated in depression. Additionally, our stringent

clinical protocols ensure a high level of sample purity and homogeneity. Participants were carefully selected using well-defined inclusion criteria, thoroughly evaluated by licensed psychologists, and independently verified by registered psychiatrists, addressing a frequent limitation in similar studies. Furthermore, by combining electrophysiological data with questionnaire-based behavioral assessments, we address the shortcomings of traditional behavioral diagnostic approaches, which are often prone to human bias and subjectivity. Analysis of this study was conducted on a cohort of 58 depressed outpatients.

Method

Participants

Our data were collected through convenience sampling from patients at Elumind Psychiatric Clinic in Vancouver, Canada. This approach resulted in a heterogeneous participant pool with variability in age and gender. To address this variability, we stratified the sample into three age groups: young adults (16–24 years), middle-aged adults (25–54 years), and older adults (55 years and above). Participants were also categorized into three groups: those using prescription medication (medicated), those not using prescription medication (nonmedicated), and those who consume alcohol or use marijuana (addicted group). This stratification allowed for a more nuanced understanding of the factors influencing the outcomes of the study. All participants presented to the clinic with depression as their primary complaint and had no history of other psychiatric disorders, intellectual disabilities, or neurological deficits. The final sample consisted of 22 males (mean age: 37.3 ± 14.07 years) and 36 females (mean age: 39.8 ± 16.90 years). Each participant provided written informed consent, completed the Beck Depression Inventory-II (BDI-II) questionnaire, and consented to undergo EEG recordings as part of the study following their therapeutic assessment. The research adhered to the ethical principles outlined in the Declaration of Helsinki (World Medical Association, 1996), including respect for individual autonomy, protection of privacy and confidentiality, maintenance of scientific integrity, and poststudy considerations, such as ensuring participants have access to any beneficial findings arising from the study. The sample size was determined based on previous studies (Bokhan et al., 2023; Yamada et al., 1995).

Beck Depression Inventory (BDI)

After welcoming the participant, informed consent was obtained, and any questions or concerns

regarding data collection, EEG recording, or other procedures were addressed. Participants then sat in a quiet room and completed the Beck Depression Inventory (BDI) questionnaire according to the provided instructions. In terms of assessing severity of symptoms, the BDI-II is a widely used 21-item self-report tool designed for adolescents and adults (Wang & Gorenstein, 2013). It demonstrates strong criterion-based sensitivity and specificity for detecting depression, reinforcing its clinical utility as a diagnostic aid (Wang & Gorenstein, 2013). Since depression symptoms can respond differently to treatment, relying solely on a global score to evaluate treatment response is insufficient. Therefore, a bifactor model of the BDI-II was developed for statistical and clinical purposes, consisting of a general depression factor and three specific factors (cognitive, affective, and somatic), which provided the best fit for the data. This model indicated that BDI-II items could be summed to generate an overall score that accounts for most of the variance, while the specific factors contributed unique variance (García-Batista et al., 2018).

EEG to Quantitative EEG (qEEG) Recording

EEG recordings were conducted in a soundproof, dimly lit chamber with minimum sources of electromagnetic and cellular interference. Participants were seated in a comfortable armchair and instructed to relax and minimize movements to reduce artifacts. EEG data were recorded using a 19-channel WinEEG system (version 202, Mitsar Inc., Russia) during a 5-min, eyes-closed session. The sampling rate was 256 Hz, with electrodes positioned according to the international 10–20 system and impedance maintained below 5 k Ω across electrode sites. Low- and high-pass filters were set at 0.1 Hz and 45 Hz, respectively, with a 55–65 Hz notch filter applied. EEG data were recorded in a monopolar montage with signals referenced to linked ears. Independent component analysis (ICA) was performed to isolate and remove artifacts related to eye movements, muscle activity, and cardiac noise. Two EEG experts then visually inspected and manually corrected the data. Finally, 90 s of artifact-free EEG recordings were selected and imported into NeuroGuide software (version 3.2.8) to measure qEEG. Fourier transform (FFT) was used for quantitative analysis, and various band measures were calculated, considering age and gender.

Regions of Interest (ROIs)

Our primary focus was on the absolute power of theta (4–8 Hz) and alpha (8–12 Hz) bands across 19 electrodes: FP1, FP2, F3, F4, Fz, F7, F8, C3, C4,

Cz, Pz, P3, P4, T3, T4, T5, T6, O1, O2. Additionally, FFT coherence of theta and alpha bands was measured between electrode pairs in the following regions: bi-frontal (FP1–FP2, F3–F4, F7–F8), frontocentral (Fz–Cz), centroparietal (Cz–Pz), frontoparietal (F3–P3, F4–P4, Fz–Pz), and frontotemporal (F3–T3, F3–T5, F4–T4, F4–T6, F7–T3, F7–T5, F8–T4, F8–T6). To assess HPC and amygdala activity and connectivity, we calculated LORETA absolute power (LAP) and LORETA coherence (LC) in the alpha and theta bands for both hemispheres. Default settings of the NeuroGuide software were used, with an epoch duration of 4 s. Electrodes were treated as independent variables in the analysis.

Statistics

To examine the effects of age and gender on the BDI scores and its subscales, we performed a multivariate analysis of variance (MANOVA). Additionally, Pearson's correlation coefficient (r) and Spearman's rank correlation (ρ) were calculated to assess relationships between BDI scores (including subscales) and EEG data, as well as LORETA findings. The choice between these correlation methods was determined based on the normality of the data. Furthermore, to control potential confounding effects of age, drug consumption, and gender, partial correlations were conducted by statistically adjusting for these variables. We used JASP (Jeffreys's Amazing Statistical Program) that is a free, friendly, and open-source software for statistical analysis.

Results

Descriptive data of our participants' BDI scores and its subscales in terms of age group and gender is shown in Table 1. Results revealed significant effect of age on cognitive, $F(2, 50) = 3.61, P = .034, \eta^2 = 0.126$. The pairwise comparison showed that old group reported less scores of cognitive scales in comparison to the middle age and the young group ($p = .005, p = .004$). Medication as a cofactor, significantly affected BDI, $F(2, 50) = 4.33, P = .018, \eta^2 = 0.148$; cognitive, $F(2, 50) = 3.61, P = .034, \eta^2 = 0.126$; and somatic scores, $F(2, 50) = 3.62, P = .034, \eta^2 = 0.127$. Pairwise analysis showed that in all three above scales, addicted group reported higher scores than medicated group (for BDI, $p = .001$, cognitive, $p = .001$, and somatic, $p = .014$).

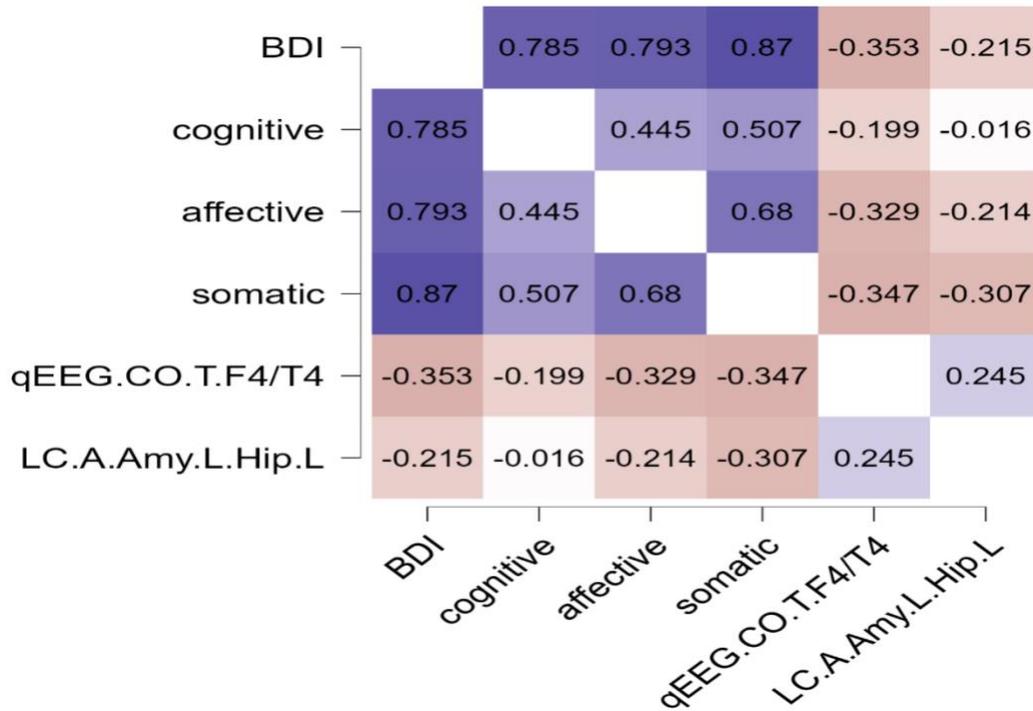
A negative correlation was observed between LORETA alpha coherency of left HPC and left amygdala and somatic scores (Pearson's $r = -0.298, p = .027$). EEG theta coherency of F4–T4 was also negatively correlated with BDI (Spearman's $\rho = -0.353, p = .014$), affective (Spearman's $\rho = -0.329, p = .008$) and somatic scores (Spearman's $\rho = -0.347, p = .010$; Table 2). Further, linear regression showed that LORETA alpha coherency of left HPC and left amygdala could explain 26% of BDI scores variance meaningfully ($R^2 = 0.49, \text{adjusted } R^2 = 0.26, P = .024$; Figure 1).

Table 1

Descriptive Table of Participants, Including Sample Size, Age, Total BDI Score, and Scores for the Cognitive, Affective, and Somatic Components

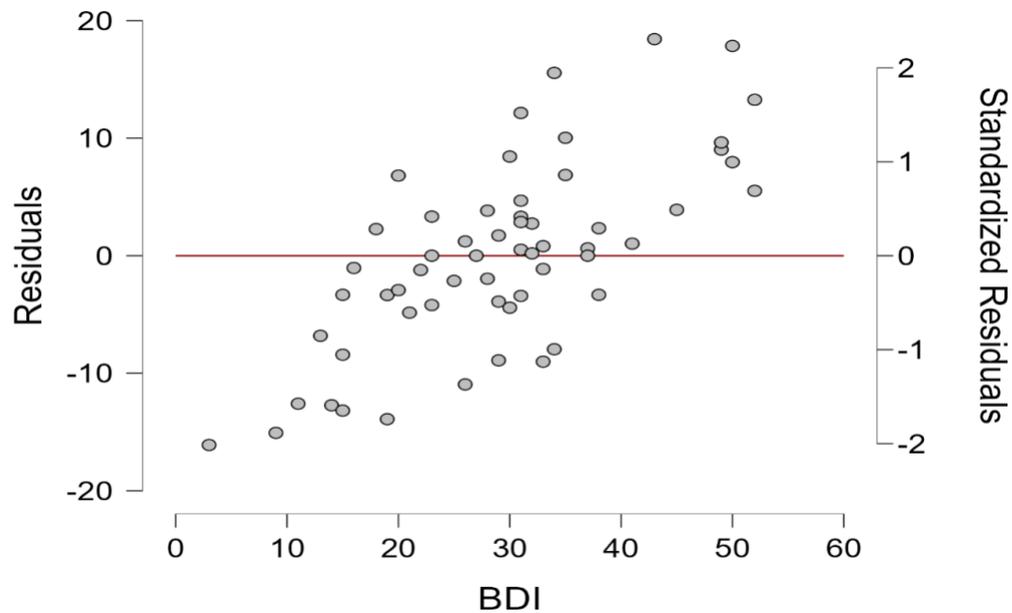
Group	Male			Female		
	Young	Middle age	Old	Young	Middle age	Old
Sample size	6	14	2	10	17	9
Age	22.5 ± 1.9	38.7 ± 5.3	72 ± 2.8	19.7 ± 2.8	39.8 ± 8.3	62.2 ± 5.5
BDI score	27.8 ± 7.52	28.8 ± 12.65	18.5 ± 0.72	36.4 ± 9.57	30.2 ± 12.3	23.1 ± 7.88
Cognitive score	9.5 ± 4.4	10.35 ± 4.41	6 ± 1.41	12.2 ± 3.93	11.17 ± 5.0	6.5 ± 3.43
Affective score	8.6 ± 1.5	7028 ± 3.14	5.5 ± 7	9.1 ± 3.47	6.8 ± 3.3	5.7 ± 2.1
Somatic score	9.6 ± 4.3	11.2 ± 6.1	7 ± 0.00	15.1 ± 4.01	12.2 ± 5.3	12.6 ± 4.7

Table 2. Partial Spearman's Rho Heatmap of Correlation Between BDI and Its Subscale Scores With EEG and LORETA Findings.



Note. BDI = Beck Depression Inventory; LC.A. Amy.L.Hip.L = LORETA coherency of alpha between left amygdala and left hippocampus; qEEG.co.T.F4/T4 = qEEG coherency of theta in F4–T4.

Figure 1. Residuals vs. Dependent Plot LORETA Alpha Coherency of Left HPC/Amygdala and BDI Scores Among MDD Participants.



Note. BDI = Beck Depression Inventory; MDD = major depressive disorder.

Discussion

This study provided a comprehensive investigation into the neurophysiological underpinnings of depression, with a particular focus on alpha and theta brainwave activity. By integrating surface EEG and LORETA methodologies, the research explored cortical and subcortical networks and their relationships with depressive symptomatology, including cognitive, affective, and somatic components.

Behavioral Findings

The findings revealed that elderly participants reported lower scores on cognitive scales compared to middle-aged and young participants. This aligns with prior research suggesting a negative correlation between age and BDI scores, with older adults potentially underreporting depressive symptoms due to factors spanning neurobiological, psychological, and social domains. These factors may obscure self-ratings of depressed mood in the elderly (Lyness et al., 1995). However, this result contrasts with a study of 556 adults and older adults, which found that the elderly scored higher on the somatic and performance subscales, but not on cognitive and affective subscales, compared to adults (Trentini et al., 2005). This disparity may be attributed to differences in sampling methods between our study and theirs. Notably, the studies have differed in terms of participant nationality. Furthermore, our study exclusively included individuals seeking therapy, while their sample may have included individuals who were not actively seeking therapeutic interventions. Another finding was significant effect of addiction on BDI, cognitive, and somatic score. These findings are consistent with previous research on 108 drug abusers, which demonstrated positive correlations between BDI-II subscales (cognitive, affective, and somatic) and the severity of alcohol and drug use (Dum et al., 2008). Similarly, another study on 42 adolescent and young adult marijuana users reported increased depressive symptoms, diminished fun-seeking, and reduced reward responsiveness associated with marijuana use (Wright et al., 2016). It was said that frontolimbic white matter integrity deficits in adolescent users probably contributed to apathy, ultimately exacerbated depressive symptoms.

Electrophysiological Findings

The analysis of LORETA data revealed a significant negative correlation between alpha coherency in the left HPC and left amygdala and somatic scores. Furthermore, this coherency accounted for 26% of the variance in BDI scores, indicating a meaningful

contribution to depressive symptomatology. Supporting these findings, a prominent study on 123 individuals with MDD and 81 matched controls identified significant differences in local networks, particularly in subregions of the left amygdala and the hippocampal tail (Zhang et al., 2022). Patients with MDD demonstrated reduced characteristic path length and modularity in these regions compared to controls. The decreased characteristic path length may reflect increased global information transmission within the hippocampus-amygdala network. This enhanced interaction may underlie the emotional facilitation of memory formation and the persistence of a bias toward sad memories in MDD patients. Reduced modularity indicates that the hippocampus-amygdala network may be less distinctly organized into discrete functional communities, reflecting impaired functional segregation. Such a less modular structure could signify disruptions in feedback and feedforward communication between the HPC and amygdala, potentially contributing to dysregulated emotional memory processes in MDD. Our finding aligns with the broader explanation of these findings. It suggests that promoting regulated, synchronized communication between left HPC and left amygdala via increased alpha coherence—that probably adjust feedback and feedforward communication—might help reduce certain depressive symptoms, particularly somatic ones. Overall, these results underscore the role of neuroanatomical alterations and biased functional interactions within the hippocampus-amygdala network in the pathophysiology of depression.

EEG data analysis revealed a negative correlation between theta coherency in the F4–T4 region and BDI scores, particularly in the affective and somatic components. As theta coherence between the right frontal and right temporal regions increased, depressive symptoms, as measured by these scales, decreased. It is hypothesized that lower brain frequencies, such as theta, reflect subcortical processing in regions like the entorhinal neurons of the medial temporal lobe, driven primarily by mass synchronized neural firing. They enable the synchronization of neural populations across large-scale networks, such as frontal and temporal regions, which play a pivotal role in memory performance and serve as a bridge between self-perception and affective states. (LaVarco et al., 2022; Takahashi et al., 2007). These networks, predominantly mediated by right-lateralized structures, significantly influence self-awareness and mood (Devinsky, 2000; Platek et al., 2004). Theta activity also plays a crucial role in emotional

processing, particularly in response to salient and arousing stimuli. Studies have demonstrated that theta power is greater for emotional stimuli compared to neutral stimuli and is sensitive to affective content irrespective of valence. Furthermore, theta activity is modulated by personal distress, highlighting its role in empathy-related and emotional regulation processes (Romeo & Spironelli, 2024). In the context of our study, the observed increase in theta wave synchronization between the right frontal and temporal cortices likely reflects enhanced functional connectivity within these neural networks. This increased synchronization may facilitate organized cognition and emotional regulation, thereby contributing to the alleviation of depressive symptoms. In confirmation of our finding, another longitudinal study investigated cognitive and emotional development in 81 healthy children and identified a significant role for frontotemporal functional connectivity, measured via EEG coherence, during an episodic memory encoding task. The findings highlighted the involvement of the right frontotemporal region (F4–T8) in supporting memory processes (Blankenship & Bell, 2015). Further support comes from a clinical trial involving 30 adolescents with conduct disorder and 34 controls (Dong et al., 2019). Resting-state fMRI data showed reduced frontotemporal connectivity in adolescents with conduct disorder, specifically in regions underlying cognitive and affective empathy. The study's authors proposed that frontotemporal communication facilitates the use of external social cues processed in temporal regions to infer emotional states in the medial prefrontal cortex. Reduced connectivity may impair the ability to access social cues, affecting cognitive empathy, leading to depressive symptoms. The improved connectivity may support processes such as emotional regulation, memory, and social understanding, contributing to the observed decreases in affective and somatic BDI scores.

Conclusion

Present findings highlight the critical role of synchronized neural activity in cortical and subcortical regions in regulating mood, providing a deeper insight into the mechanisms underlying depressive symptoms. Enhanced connectivity within key networks, such as the hippocampus-amygdala and frontotemporal regions, may represent a target for interventions aimed at alleviating specific depressive symptoms, particularly those related to somatic and affective dimensions. Overall, this study highlights the critical role of neurophysiological alterations in shaping the pathophysiology of

depression and offers a foundation for future research exploring targeted brain areas. However, further studies, particularly those employing integrated EEG-MRI approaches, are necessary to investigate replication. Cofactors such as unwanted artifacts, the limited spatial resolution of LORETA, and the complex reciprocal connections between regions like the amygdala and HPC may confound the results, making it premature to draw clinical applications from these findings.

Limitation and Implication for Future Research

It is notable that our finding about the role of age deserves careful consideration as other important factors such as race, socioeconomic status, and cultural background that might affect reporting of symptoms were not assessed in our study. Our findings were also influenced by the limited sample size, particularly after stratifying participants into three groups, which increased susceptibility to variability and hindered result consolidation. Future studies should address this by leveraging large, stratified EEG databanks. Training machine learning algorithms on prevalidated EEG patterns with adequately sized datasets could equip health professionals with a versatile, portable, and cost-effective tool for reliably diagnosing depression. We strongly recommend adopting standardized artifact correction protocols, enforcing stringent inclusion and exclusion criteria, and incorporating the visual cortex in future analyses—an area we were unable to explore due to the data volume involved.

Author Declaration

The authors declare no conflicts of interest. The research was conducted independently, adhering to ethical standards, without external funding or financial incentives, and driven solely by the authors' academic and clinical interests. Dr. Kourosh Edalati served as the primary supervisor for this research project. The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to restrictions (e.g., containing information that could compromise the privacy of research participants).

References

- American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). <https://doi.org/10.1176/appi.books.9780890425787>
- Blankenship, T. L., & Bell, M. A. (2015). Frontotemporal coherence and executive functions contribute to episodic memory during middle childhood. *Developmental Neuropsychology*, *40*(7–8), 430–444. <https://doi.org/10.1080/87565641.2016.1153099>

- Bokhan, N. A., Galkin, S. A., & Vasilyeva, S. N. (2023). EEG alpha band characteristics in patients with a depressive episode within recurrent and bipolar depression. *Consortium Psychiatricum*, 4(3), 5–12. <https://doi.org/10.17816/CP6140>
- Cornwell, B. R., Salvatore, G., Colon-Rosario, V., Latov, D. R., Holroyd, T., Carver, F. W., Coppola, R., Manji, H. K., Zarate, C. A., & Grillon, C. (2010). Abnormal hippocampal functioning and impaired spatial navigation in depressed individuals: Evidence from whole-head magnetoencephalography. *American Journal of Psychiatry*, 167(7), 836–844. <https://doi.org/10.1176/appi.ajp.2009.09050614>
- Cui, L., Li, S., Wang, S., Wu, X., Liu, Y., Yu, W., Wang, Y., Tang, Y., Xia, M., & Li, B. (2024). Major depressive disorder: Hypothesis, mechanism, prevention and treatment. *Signal Transduction and Targeted Therapy*, 9(1), Article 30. <https://doi.org/10.1038/s41392-024-01738-y>
- Damborská, A., Honzirková, E., Barteček, R., Hořínková, J., Fedorová, S., Ondruš, Š., Michel, C. M., & Rubega, M. (2020). Altered directed functional connectivity of the right amygdala in depression: High-density EEG study. *Scientific Reports*, 10(1), Article 4398. <https://doi.org/10.1038/s41598-020-61264-z>
- Dev, A., Roy, N., Islam, Md. K., Biswas, C., Ahmed, H. U., Amin, Md. A., Sarker, F., Vaidyanathan, R., & Mamun, K. A. (2022). Exploration of EEG-based depression biomarkers identification techniques and their applications: A systematic review. *IEEE Access*, 10, 16756–16781. <https://doi.org/10.1109/ACCESS.2022.3146711>
- Devinsky, O. (2000). Right cerebral hemisphere dominance for a sense of corporeal and emotional self. *Epilepsy & Behavior*, 1(1), 60–73. <https://doi.org/10.1006/ebeh.2000.0025>
- Dong, D., Jiang, Y., Gao, Y., Ming, Q., Wang, X., & Yao, S. (2019). Atypical frontotemporal connectivity of cognitive empathy in male adolescents with conduct disorder. *Frontiers in Psychology*, 9, Article 2778. <https://doi.org/10.3389/fpsyg.2018.02778>
- Dum, M., Pickren, J., Sobell, L. C., & Sobell, M. B. (2008). Comparing the BDI-II and the PHQ-9 with outpatient substance abusers. *Addictive Behaviors*, 33(2), 381–387. <https://doi.org/10.1016/j.addbeh.2007.09.017>
- Friedrich, M. J. (2017). Depression is the leading cause of disability around the world. *JAMA*, 317(15), Article 1517. <https://doi.org/10.1001/jama.2017.3826>
- García-Batista, Z. E., Guerra-Peña, K., Cano-Vindel, A., Herrera-Martínez, S. X., & Medrano, L. A. (2018). Validity and reliability of the Beck Depression Inventory (BDI-II) in general and hospital population of Dominican Republic. *PLoS ONE*, 13(6), Article e0199750. <https://doi.org/10.1371/journal.pone.0199750>
- Gould, N. F., Holmes, M. K., Fantie, B. D., Luckenbaugh, D. A., Pine, D. S., Gould, T. D., Burgess, N., Manji, H. K., & Zarate, C. A. (2007). Performance on a virtual reality spatial memory navigation task in depressed patients. *The American Journal of Psychiatry*, 164(3), 516–519. <https://doi.org/10.1176/ajp.2007.164.3.516>
- LaVarco, A., Ahmad, N., Archer, Q., Pardillo, M., Nunez Castaneda, R., Minervini, A., & Keenan, J. P. (2022). Self-conscious emotions and the right fronto-temporal and right temporal parietal junction. *Brain Sciences*, 12(2), Article 138. <https://doi.org/10.3390/brainsci12020138>
- Leuchter, A. F., Cook, I. A., Hunter, A. M., Cai, C., & Horvath, S. (2012). Resting-state quantitative electroencephalography reveals increased neurophysiologic connectivity in depression. *PLoS ONE*, 7(2), Article e32508. <https://doi.org/10.1371/journal.pone.0032508>
- Liu, X., Zhang, H., Cui, Y., Zhao, T., Wang, B., Xie, X., Liang, S., Sha, S., Yan, Y., Zhao, X., & Zhang, L. (2024). EEG-based major depressive disorder recognition by neural oscillation and asymmetry. *Frontiers in Neuroscience*, 18, Article 1362111. <https://doi.org/10.3389/fnins.2024.1362111>
- Lorenzetti, V., Allen, N. B., Fornito, A., & Yücel, M. (2009). Structural brain abnormalities in major depressive disorder: A selective review of recent MRI studies. *Journal of Affective Disorders*, 117(1–2), 1–17. <https://doi.org/10.1016/j.jad.2008.11.021>
- Lyness, J. M., Cox, C., Curry, J., Conwell, Y., King, D. A., & Caine, E. D. (1995). Older age and the underreporting of depressive symptoms. *Journal of the American Geriatrics Society*, 43(3), 216–221. <https://doi.org/10.1111/j.1532-5415.1995.tb07325.x>
- Mayberg, H. (1997). Limbic-cortical dysregulation: A proposed model of depression. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 9(3), 471–481. <https://doi.org/10.1176/jnp.9.3.471>
- McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience*, 27(1), 1–28. <https://doi.org/10.1146/annurev.neuro.27.070203.144157>
- Platek, S. M., Keenan, J. P., Gallup, G. G., & Mohamed, F. B. (2004). Where am I? The neurological correlates of self and other. *Cognitive Brain Research*, 19(2), 114–122. <https://doi.org/10.1016/j.cogbrainres.2003.11.014>
- Romeo, Z., & Spironelli, C. (2024). Theta oscillations underlie the interplay between emotional processing and empathy. *Heliyon*, 10(14), Article e34581. <https://doi.org/10.1016/j.heliyon.2024.e34581>
- Siegle, G. J., Thompson, W., Carter, C. S., Steinhauer, S. R., & Thase, M. E. (2007). Increased amygdala and decreased dorsolateral prefrontal BOLD responses in unipolar depression: Related and independent features. *Biological Psychiatry*, 61(2), 198–209. <https://doi.org/10.1016/j.biopsych.2006.05.048>
- Takahashi, E., Ohki, K., & Kim, D.-S. (2007). Diffusion tensor studies dissociated two fronto-temporal pathways in the human memory system. *NeuroImage*, 34(2), 827–838. <https://doi.org/10.1016/j.neuroimage.2006.10.009>
- Tang, S., Lu, L., Zhang, L., Hu, X., Bu, X., Li, H., Hu, X., Gao, Y., Zeng, Z., Gong, Q., & Huang, X. (2018). Abnormal amygdala resting-state functional connectivity in adults and adolescents with major depressive disorder: A comparative meta-analysis. *EBioMedicine*, 36, 436–445. <https://doi.org/10.1016/j.ebiom.2018.09.010>
- Trambaiolli, L. R., & Biazoli, C. E. (2020). Resting-state global EEG connectivity predicts depression and anxiety severity. *2020 42nd Annual International Conference of the IEEE Engineering in Medicine & Biology Society (EMBC)*, pp. 3707–3710. Montreal, Canada. <https://doi.org/10.1109/EMBC44109.2020.9176161>
- Trentini, C. M., Xavier, F. M. D. F., Chachamovich, E., Rocha, N. S. D., Hirakata, V. N., & Fleck, M. P. D. A. (2005). The influence of somatic symptoms on the performance of elders in the Beck Depression Inventory (BDI). *Revista Brasileira de Psiquiatria*, 27(2), 119–123. <https://doi.org/10.1590/S1516-44462005000200009>
- Tseng, H.-J., Lu, C.-F., Jeng, J.-S., Cheng, C.-M., Chu, J.-W., Chen, M.-H., Bai, Y.-M., Tsai, S.-J., Su, T.-P., & Li, C.-T. (2022). Frontal asymmetry as a core feature of major depression: A functional near-infrared spectroscopy study. *Journal of Psychiatry and Neuroscience*, 47(3), E186–E193. <https://doi.org/10.1503/jpn.210131>
- Wang, Y.-P., & Gorenstein, C. (2013). Psychometric properties of the Beck Depression Inventory-II: A comprehensive review. *Revista Brasileira de Psiquiatria*, 35(4), 416–431. <https://doi.org/10.1590/1516-4446-2012-1048>
- Williams, K. A., Mehta, N. S., Redei, E. E., Wang, L., & Prociassi, D. (2014). Aberrant resting-state functional connectivity in a genetic rat model of depression. *Psychiatry Research: Neuroimaging*, 222(1–2), 111–113. <https://doi.org/10.1016/j.psychresns.2014.02.001>

- Wright, N. E., Scerpella, D., & Lisdahl, K. M. (2016). Marijuana use is associated with behavioral approach and depressive symptoms in adolescents and emerging adults. *PLoS ONE*, *11*(11), Article e0166005. <https://doi.org/10.1371/journal.pone.0166005>
- Yamada, M., Kimura, M., Mori, T., & Endo, S. (1995). EEG power and coherence in presenile and senile depression. Characteristic findings related to differences between anxiety type and retardation type. *Journal of Nippon Medical School*, *62*(2), 176–185. <https://doi.org/10.1272/jnms1923.62.176>
- Yang, J., Yin, Y., Svob, C., Long, J., He, X., Zhang, Y., Xu, Z., Li, L., Liu, J., Dong, J., Zhang, Z., Wang, Z., & Yuan, Y. (2017). Amygdala atrophy and its functional disconnection with the cortico-striatal-pallidal-thalamic circuit in major depressive disorder in females. *PLoS ONE*, *12*(1), Article e0168239. <https://doi.org/10.1371/journal.pone.0168239>
- Zhang, L., Hu, X., Hu, Y., Tang, M., Qiu, H., Zhu, Z., Gao, Y., Li, H., Kuang, W., & Ji, W. (2022). Structural covariance network of the hippocampus–amygdala complex in medication-naïve patients with first-episode major depressive disorder. *Psychoradiology*, *2*(4), 190–198. <https://doi.org/10.1093/psyrad/kkac023>
- Zheng, J., Anderson, K. L., Leal, S. L., Shestyuk, A., Gulsen, G., Mnatsakanyan, L., Vadera, S., Hsu, F. P. K., Yassa, M. A., Knight, R. T., & Lin, J. J. (2017). Amygdala-hippocampal dynamics during salient information processing. *Nature Communications*, *8*(1), Article 14413. <https://doi.org/10.1038/ncomms14413>

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