JBSS
Journal of Biomedical & Space Sciences

LIASTRA INSTITUTE

Review

Neuroimaging in Anxiety and Depression: An Integrative Review of fMRI, EEG, and Neural Biomarkers

Marina Corrêa Freitas[1,*], Fabrício Veloso[1]

[1]ICBS – International Center for Biomedical & Space Sciences, LIASTRA Institute, Rio de Janeiro, Brazil
*Correspondence: marinafreitas@liastra.com, marinacorreafreitas27@gmail.com | ORCID 0000-0003-1723-4113

SUMMARY

Anxiety and depressive disorders are highly prevalent psychiatric conditions associated with significant disability worldwide. Neuroimaging has advanced understanding of their neural substrates, particularly through functional magnetic resonance imaging (fMRI) and electroencephalography (EEG). This integrative review synthesized studies published between 2013 and 2025 retrieved from PubMed, Scopus, Web of Science, and PsycINFO, including adult clinical samples diagnosed with anxiety and/or depressive disorders. Evidence consistently indicates dysregulation within fronto-limbic circuitry and large-scale brain networks, notably involving the amygdala, prefrontal cortex, anterior cingulate cortex, and default mode network. fMRI findings highlight altered functional connectivity and impaired emotional regulation, whereas EEG studies reveal abnormalities in oscillatory activity and event-related potentials linked to attentional bias and cognitive control. Although convergent neural markers emerge across modalities, methodological heterogeneity limits reproducibility and clinical application. Multimodal integration of spatial and temporal neural measures may enhance biomarker reliability and support the development of precision psychiatry approaches.

KEYWORDS

Neuroimaging; Anxiety Disorders; Depressive Disorders; fMRI; EEG; Neural Biomarkers.

INTRODUCTION

Anxiety and depressive disorders represent complex, heterogeneous psychiatric syndromes characterized by pervasive disturbances in affective regulation, cognitive processing, and stress responsivity. Collectively, they account for a substantial proportion of global disability-adjusted life years (DALYs), imposing profound socioeconomic and public health burdens (World Health Organization, 2023). Major depressive disorder (MDD) is typified by persistent low mood, anhedonia, and cognitive dysfunction, whereas anxiety disorders involve excessive fear, hypervigilance, and heightened physiological arousal. Despite their distinct clinical phenotypes, these conditions frequently co-occur, suggesting shared neurobiological substrates (Etkin & Wager, 2007).

Traditional diagnostic frameworks, including DSM-5 and ICD-11 classifications, rely primarily on symptom clusters rather than objective biological markers. This limitation has motivated the search for neurobiological correlates capable of refining diagnosis, stratifying risk, and predicting treatment response. The National Institute of Mental Health's Research Domain Criteria (RDoC) initiative has emphasized dimensional constructs and neural circuitry as foundational elements for psychiatric classification (Insel et al., 2010). Within this paradigm, neuroimaging plays a central role.

Functional magnetic resonance imaging (fMRI) enables mapping of task-based and resting-state functional connectivity with high spatial resolution. Studies employing resting-state fMRI have consistently demonstrated alterations in intrinsic connectivity networks, including the Default Mode Network (DMN), Salience Network, and Central Executive Network (Menon, 2011; Kaiser et al., 2015). Task-based paradigms further reveal aberrant activation in emotion-processing circuits, particularly within the amygdala and prefrontal cortex (Etkin & Wager, 2007).

Complementarily, electroencephalography (EEG) offers superior temporal resolution, capturing oscillatory dynamics and event-related potentials associated with cognitive and emotional processes (Luck, 2014). EEG markers such as frontal alpha asymmetry, P300 amplitude, and error-related negativity (ERN) have been implicated in affective dysregulation and attentional bias (Thibodeau et al., 2006; Moser et al., 2013).

Despite the abundance of neuroimaging research, inconsistencies across modalities, analytic techniques, and clinical samples have impeded biomarker validation. Therefore, integrating spatial (fMRI) and temporal (EEG) dimensions is essential for a more comprehensive neurobiological model of affective disorders.

The present integrative review synthesizes multimodal neuroimaging evidence in clinically diagnosed anxiety and depressive disorders, examining convergent neural patterns and evaluating their translational implications.

LITERATURE BACKGROUND AND THEORETICAL FOUNDATIONS

From Regional Activation Models to Network Psychiatry

Neuroimaging research in affective disorders has undergone a profound conceptual transformation over the past three decades. Early investigations primarily focused on identifying localized abnormalities in specific brain regions, particularly limbic structures such as the amygdala and hippocampus. These region-centric approaches provided initial evidence of amygdala hyperreactivity in anxiety disorders and volumetric reductions in hippocampal structures in depression. However, as methodological sophistication increased, it became evident that psychiatric disorders could not be fully explained by isolated regional dysfunction.

The transition from regional activation models to network-based frameworks represents a paradigm shift in psychiatric neuroscience. Large-scale brain networks, including the Default Mode Network (DMN), Salience Network (SN), and Central Executive Network (CEN), have emerged as critical organizing systems underlying cognitive and emotional processes (Menon, 2011). The so-called "triple network model" proposes that psychopathology results from disrupted interactions among these intrinsic connectivity networks. In this context, affective disorders are conceptualized not merely as hyper- or hypoactivation of discrete regions but as dysregulated coordination among distributed neural systems.

Within anxiety and depressive disorders, this network perspective provides a more comprehensive explanatory framework. Hyperactivity of the Salience Network may bias attentional resources toward threat detection in anxiety, whereas hyperconnectivity within the DMN may sustain maladaptive rumination in depression (Hamilton et al., 2015). The CEN, primarily anchored in the dorsolateral prefrontal cortex, plays a regulatory role, and its reduced engagement may impair cognitive reappraisal and executive control (Disner et al., 2011).

Thus, contemporary neuroimaging evidence increasingly supports a dynamic systems model of affective psychopathology.

Fronto-Limbic Dysregulation as a Core Mechanism

Despite the shift toward network psychiatry, the fronto-limbic dysregulation model remains central to understanding affective disorders. This model posits that exaggerated bottom-up signaling from limbic structures, particularly the amygdala, is insufficiently modulated by top-down prefrontal control systems (Disner et al., 2011).

Meta-analytic evidence has consistently demonstrated amygdala hyperactivation in response to negative emotional stimuli in both anxiety and depression (Etkin & Wager, 2007). Concurrently, reduced activity in regulatory regions such as the dlPFC and ventromedial prefrontal cortex has been observed. Importantly, this imbalance is not merely additive but reflects altered functional connectivity between these regions.

The ACC further integrates emotional salience and cognitive conflict processing. Dysregulation in this region may impair adaptive behavioral adjustment, contributing to perseverative cognition and maladaptive threat appraisal.

Therefore, while network models broaden the conceptual framework, the fronto-limbic axis remains a mechanistic cornerstone.

Dimensional Psychiatry and the RDoC Framework

A critical theoretical development influencing contemporary neuroimaging research is the shift from categorical to dimensional models of psychopathology. Traditional diagnostic systems (DSM and ICD) rely on symptom clusters that may obscure underlying biological heterogeneity. The Research Domain Criteria (RDoC) initiative introduced by the National Institute of Mental Health emphasizes neural circuitry and dimensional constructs such as negative valence systems, cognitive control, and arousal regulation (Insel et al., 2010).

Under the RDoC framework, anxiety and depression are not viewed as discrete entities but as overlapping disturbances across shared neural domains. For instance, both disorders involve disruptions in negative valence processing and cognitive control systems. This dimensional perspective aligns closely with network-based neuroimaging findings.

Importantly, this theoretical orientation also justifies multimodal neuroimaging integration. If psychiatric phenomena are distributed across circuits rather than confined to categories, then comprehensive measurement requires both spatial and temporal resolution.

The Biomarker Debate: Reproducibility and Validity

Despite enthusiasm surrounding neuroimaging biomarkers, substantial debate persists regarding their reliability and clinical applicability. Concerns include small sample sizes, inflated effect sizes, analytic flexibility, and lack of cross-site replication (Poldrack et al., 2017).

METHODS

Review Design and Research Framework

This review adhered to PRISMA 2020 (Figure 1) guidelines for systematic reporting (Page et al., 2021). Database searches were conducted across PubMed, Scopus, Web of Science, and PsycINFO between 2013–2025.

Search terms integrated diagnostic and neuroimaging constructs:

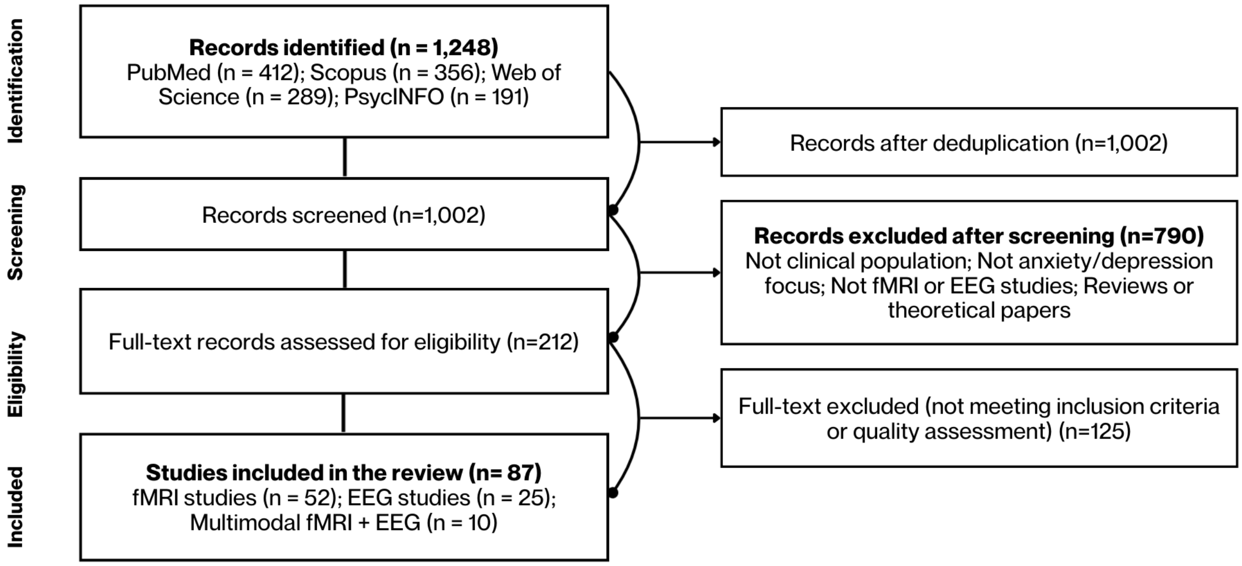

Figure 1. PRISMA 2020 Flow Diagram
PRISMA 2020 flow diagram illustrating the identification, screening, eligibility assessment, and inclusion process of studies investigating fMRI and EEG neural correlates in clinically diagnosed anxiety and depressive disorders (2013–2025).

"major depressive disorder" OR "anxiety disorder") AND ("fMRI" OR "functional connectivity" OR "EEG" OR "oscillatory activity") AND ("biomarker" OR "neural correlate”).

Inclusion criteria required: Adult participants (≥18 years); Clinical diagnosis per DSM-5 or ICD criteria; Use of fMRI and/or EEG; Quantitative empirical design. Studies focusing on animal models, case reports, pediatric populations, or exclusively methodological innovations were excluded.

A total of 1,248 records were identified, 1,002 remained after duplicate removal, and 87 studies were included following eligibility screening.

RESULTS

fMRI Evidence: Network Dysregulation, Emotional

Across the 52 fMRI studies, a highly consistent pattern of fronto-limbic dysregulation emerged in both anxiety and depressive disorders. However, subtle differences in activation patterns and connectivity profiles suggest partially overlapping but distinct neural phenotypes (Table 1).

Amygdala Hyperreactivity and Emotional Salience

Amygdala hyperactivation during exposure to negative emotional stimuli was one of the most robust findings across both disorders (Etkin & Wager, 2007). In anxiety disorders, this hyperactivation was particularly pronounced in response to threat-related stimuli (e.g., fearful faces, anticipation paradigms), supporting models of hypervigilance and exaggerated threat appraisal. In major depressive disorder, amygdala hyperactivation often co-occurred with sustained responses to negative stimuli and blunted responses to positive stimuli, indicating altered valence processing rather than purely threat-specific reactivity.

Importantly, several studies reported altered amygdala-prefrontal connectivity rather than isolated hyperactivity, suggesting that dysregulated communication between limbic and regulatory cortical systems may underlie symptom persistence.

Prefrontal Cortex Hypoactivation and Top-Down Regulation

Consistent hypoactivation of the dorsolateral prefrontal cortex (dlPFC) was observed during cognitive control and emotion regulation tasks (Disner et al., 2011). The dlPFC plays a critical role in working memory, cognitive reappraisal, and inhibitory control. Reduced dlPFC engagement suggests impaired top-down modulation of limbic responses, potentially contributing to rumination in depression and sustained worry in anxiety disorders.

Moreover, reduced ventromedial prefrontal cortex (vmPFC) activity was associated with impaired extinction learning in anxiety and reduced reward valuation in depression. This finding links affective dysregulation to impaired valuation and prediction error signaling.

Table 1. Expanded Summary of fMRI Findings

Brain Region	Anxiety	Depression	Functional Role	Interpretation
Amygdala	Hyperactivation (threat-specific)	Sustained hyperactivation (negative bias)	Emotional salience	Heightened affective reactivity
dlPFC	Hypoactivation	Hypoactivation	Cognitive control	Impaired top-down regulation
vmPFC	Altered activity	Reduced reward valuation	Emotional valuation	Impaired extinction/reward processing
ACC	Hyperactivity (anticipatory)	Reduced activity	Conflict monitoring	Dysregulated performance adjustment
DMN	Network instability	Hyperconnectivity	Self-referential processing	Rumination
Salience Network	Hyperactivity	Altered coupling	Stimulus detection	Hypervigilance
Hippocampus	Connectivity disruption	Volume reduction	Memory encoding	Stress dysregulation

Anterior Cingulate Cortex and Conflict Monitoring

Altered activity in the anterior cingulate cortex (ACC) was frequently reported. The dorsal ACC is implicated in cognitive conflict monitoring, while the ventral ACC contributes to affective integration. Reduced ACC activity in depression has been associated with impaired error processing and diminished adaptive behavioral adjustment (Kaiser et al., 2015). In anxiety disorders, heightened ACC activation during threat anticipation may reflect excessive monitoring of potential errors or negative outcomes.

Default Mode Network (DMN) and Rumination

Resting-state fMRI analyses consistently demonstrated hyperconnectivity within the Default Mode Network in depressive samples (Sheline et al., 2010; Hamilton et al., 2015). DMN hyperconnectivity, particularly between the medial prefrontal cortex and posterior cingulate cortex, correlates strongly with rumination severity and maladaptive self-referential processing.

In anxiety disorders, DMN alterations were less consistently hyperconnected but showed aberrant coupling with the Salience Network, potentially reflecting unstable switching between internal mentation and threat monitoring.

Salience Network and Hypervigilance

The anterior insula and ACC, central nodes of the Salience Network, exhibited altered functional connectivity patterns in anxiety disorders (Menon, 2011). Hyperactivity within this network may amplify detection of emotionally salient stimuli, reinforcing hypervigilant states.

Hippocampal Involvement and Stress Encoding

Emerging studies highlighted hippocampal volume reductions and altered connectivity with the amygdala and prefrontal cortex (Kaiser et al., 2015). Given the hippocampus' role in contextual memory and stress regulation, its dysfunction may contribute to maladaptive encoding of negative experiences and impaired stress recovery.

EEG Evidence: Oscillatory Dysregulation and Event-Related Potentials

EEG studies provided complementary temporal insights into affective dysregulation, revealing abnormalities in oscillatory rhythms and rapid cognitive processing markers (Table 2).

Oscillatory Activity

In anxiety disorders, elevated beta-band activity was frequently reported (Engel & Fries, 2010), consistent with sustained cortical arousal and increased sympathetic activation. Beta oscillations are associated with active cognitive engagement and motor preparation; persistent elevation may reflect chronic hyperarousal.

Depression was more consistently associated with reduced alpha power, particularly in frontal regions (Thibodeau et al., 2006). Frontal alpha asymmetry, characterized by reduced left frontal activity relative to right, has been interpreted as a marker of diminished approach motivation and reward sensitivity.

Theta oscillations in midline frontal regions have also been implicated in error processing and cognitive control, with altered theta power observed in both disorders, though findings remain heterogeneous.

Event-Related Potentials (ERPs)

The P300 component, reflecting attentional allocation and stimulus evaluation, was reduced in amplitude in depressive samples (Polich, 2007), indicating impaired attentional resource allocation. In anxiety disorders, enhanced error-related negativity (ERN) amplitude was consistently observed (Moser et al., 2013), suggesting heightened sensitivity to performance monitoring and internal error detection.

Importantly, ERN hyperactivity may serve as a vulnerability marker for anxiety disorders rather than a state-dependent effect, highlighting its potential prognostic relevance.

JBSS
Journal of Biomedical & Space Sciences
LIASTRA INSTITUTE

Table 2. Expanded Summary of EEG Biomarkers

EEG Marker	Anxiety	Depression	Neural Mechanism	Clinical Implication
Frontal Alpha Asymmetry	Right-dominant	Left hypoactivity	Motivational imbalance	Anhedonia
Beta Power	Elevated	Variable	Hyperarousal	Persistent worry
Theta Power	Altered	Altered	Cognitive control	Executive dysfunction
P300	Reduced	Reduced	Attention allocation	Cognitive slowing
ERN	Increased	Variable	Error monitoring	Perfectionism/anxiety

Multimodal Integration and Neurobiological Subtyping

Multimodal studies demonstrated convergence between spatial and temporal abnormalities. Reduced dlPFC activation observed in fMRI often corresponded with reduced frontal alpha power in EEG, suggesting consistent prefrontal dysregulation across modalities.

Drysdale et al. (2017) identified neurophysiological subtypes of depression using resting-state connectivity patterns, revealing distinct connectivity profiles associated with differential response to transcranial magnetic stimulation. Such findings underscore the promise of multimodal integration in identifying biologically meaningful subgroups.

Integration of EEG and fMRI also allows investigation of oscillation-based connectivity mechanisms underlying large-scale network interactions, bridging spatial and temporal resolution limitations inherent in single-modality approaches.

DISCUSSION

The present integrative synthesis reinforces the conceptualization of anxiety and depressive disorders as network-based pathologies rather than localized structural abnormalities. The repeated observation of fronto-limbic imbalance across modalities supports a unified neurobiological framework centered on dysregulated emotional salience processing and impaired regulatory control.

The amygdala-prefrontal circuitry emerges as a central axis in affective dysregulation. Hyperreactive limbic responses combined with insufficient cortical regulation may generate persistent negative affect, attentional bias toward threat, and maladaptive rumination. Importantly, these patterns are not static; they reflect dynamic network interactions modulated by stress, environmental context, and cognitive appraisal.

Large-scale network dysfunction further refines this model. Hyperconnectivity within the DMN in depression contributes to persistent self-focused rumination, while Salience Network hyperactivity in anxiety may bias attention toward potential threats. The inability to flexibly shift between intrinsic and task-positive networks may underlie cognitive inflexibility observed clinically.

EEG findings enrich this model by revealing rapid temporal abnormalities in attentional allocation and performance monitoring. Enhanced ERN in anxiety suggests exaggerated internal monitoring, potentially contributing to perfectionism and anticipatory worry. Reduced P300 in depression reflects impaired engagement with external stimuli, aligning with cognitive slowing and reduced motivation.

However, several translational barriers remain. Methodological heterogeneity—including variability in acquisition parameters, preprocessing pipelines, and statistical thresholds—complicates replication (Poldrack et al., 2017). Moreover, most studies remain cross-sectional, limiting inference regarding causality and treatment prediction.

Future progress requires large-scale harmonized datasets, multimodal integration, longitudinal tracking, and incorporation of computational modeling approaches. Precision psychiatry frameworks integrating neuroimaging, behavioral, and genetic data may ultimately redefine diagnostic classification and guide individualized interventions.

CONCLUSION

The present integrative review provides convergent evidence that anxiety and depressive disorders are fundamentally characterized by dysregulation of fronto-limbic circuitry and large-scale intrinsic brain networks. Across both fMRI and EEG modalities, a consistent neurobiological pattern emerges: heightened limbic responsivity, particularly within the amygdala; reduced prefrontal regulatory engagement; altered anterior cingulate function; and disrupted coordination among the Default Mode, Salience, and Executive Control Networks. These findings collectively reinforce the conceptualization of affective disorders as dynamic network-based pathologies rather than localized structural abnormalities.

Importantly, the integration of spatially precise measures derived from fMRI with temporally sensitive electrophysiological indices obtained through EEG provides a more comprehensive understanding of the neural architecture underlying emotional dysregulation. While fMRI delineates functional connectivity patterns and network-level organization, EEG reveals rapid oscillatory and event-related processes that reflect cognitive control, attentional allocation, and performance monitoring. The convergence of these modalities strengthens the reliability of candidate neural biomarkers and reduces the interpretative limitations inherent in single-method approaches.

Nevertheless, the path from neuroimaging findings to clinical implementation remains complex. Despite promising neural signatures, substantial methodological heterogeneity persists across acquisition parameters, analytic pipelines, and sample characterization. The predominance of cross-sectional designs further limits causal inference and predictive validity. Consequently, the translation of neuroimaging biomarkers into routine psychiatric diagnostics requires rigorous standardization, harmonized multi-site collaborations, and longitudinal validation studies.

From a translational perspective, multimodal neuroimaging holds considerable promise for advancing precision psychiatry. Biomarker-informed stratification may enable identification of neurobiologically distinct subtypes within traditionally heterogeneous diagnostic categories, potentially guiding personalized pharmacological, psychotherapeutic, or neuromodulatory interventions. Integration with machine learning frameworks and computational modeling may further enhance predictive capacity, moving the field toward individualized risk assessment and treatment optimization.

Beyond clinical implications, these findings also carry epistemological significance. The shift from symptom-based categorical diagnosis toward circuit-based dimensional models represents a paradigm transformation in psychiatry. Neuroimaging does not merely complement existing diagnostic systems; it challenges foundational assumptions about how psychiatric disorders are conceptualized, classified, and treated.

In conclusion, multimodal neuroimaging evidence substantiates the central role of fronto-limbic and large-scale network dysregulation in anxiety and depressive disorders. The integration of fMRI and EEG findings offers a robust framework for understanding the neural mechanisms of affective psychopathology and lays critical groundwork for biomarker-driven precision psychiatry. Continued methodological refinement, large-scale replication, and longitudinal multimodal research will be essential to bridge the gap between neurobiological discovery and tangible clinical impact.

ACKNOWLEDGMENTS

The authors have no acknowledgments to declare.

AUTHOR CONTRIBUTIONS

Conceptualization, F.V.; methodology, M.F.; investigation, M.F.; writing—original draft, M.F.; writing—review & editing, M.F.; funding acquisition, F.V.; resources, E.A.; supervision, M.F.

DECLARATION OF INTERESTS

The authors state no conflict of interest.

REFERENCES

1. Brown, O., & Eremenko, P. (2006). The value proposition for fractionated spacecraft. *Acta Astronautica, 58*(12), 645–657. https://doi.org/10.1016/j.actaastro.2006.02.002
2. Disner, S. G., Beevers, C. G., Haigh, E. A. P., & Beck, A. T. (2011). Neural mechanisms of the cognitive model of depression. Nature Reviews Neuroscience, 12(8), 467–477. https://doi.org/10.1038/nrn3027
3. Drysdale, A. T., Grosenick, L., Downar, J., Dunlop, K., Mansouri, F., Meng, Y., … Liston, C. (2017). Resting-state connectivity biomarkers define neurophysiological subtypes of depression. Nature Medicine, 23(1), 28–38. https://doi.org/10.1038/nm.4246
4. Engel, A. K., & Fries, P. (2010). Beta-band oscillations—signalling the status quo? Current Opinion in Neurobiology, 20(2), 156–165. https://doi.org/10.1016/j.conb.2010.02.015
5. Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. American Journal of Psychiatry, 164(10), 1476–1488. https://doi.org/10.1176/appi.ajp.2007.07030504
6. Fox, M. D., & Raichle, M. E. (2007). Spontaneous fluctuations in brain activity observed with functional magnetic resonance imaging. Nature Reviews Neuroscience, 8(9), 700–711. https://doi.org/10.1038/nrn2201
7. Hamilton, J. P., Farmer, M., Fogelman, P., & Gotlib, I. H. (2015). Depressive rumination, the default-mode network, and the dark matter of clinical neuroscience. Biological Psychiatry, 78(4), 224–230. https://doi.org/10.1016/j.biopsych.2015.02.020
8. Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D., Quinn, K., … Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. American Journal of Psychiatry, 167(7), 748–751. https://doi.org/10.1176/appi.ajp.2010.09091379
9. Kaiser, R. H., Andrews-Hanna, J. R., Wager, T. D., & Pizzagalli, D. A. (2015). Large-scale network dysfunction in major depressive disorder: A meta-analysis of resting-state functional connectivity. JAMA Psychiatry, 72(6), 603–611. https://doi.org/10.1001/jamapsychiatry.2015.0071
10. Luck, S. J. (2014). An introduction to the event-related potential technique (2nd ed.). MIT Press.
11. Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. Trends in Cognitive Sciences, 15(10), 483–506. https://doi.org/10.1016/j.tics.2011.08.003
12. Moser, J. S., Moran, T. P., Schroder, H. S., Donnellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. Biological Psychology, 93(1), 1–10. https://doi.org/10.1016/j.biopsycho.2012.12.001
13. Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., … Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ, 372, n71. https://doi.org/10.1136/bmj.n71

14. Poldrack, R. A., Baker, C. I., Durnez, J., Gorgolewski, K. J., Matthews, P. M., Munafo, M. R., … Yarkoni, T. (2017). Scanning the horizon: Towards transparent and reproducible neuroimaging research. Nature Reviews Neuroscience, 18(2), 115–126. https://doi.org/10.1038/nrn.2016.167

15. Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. Clinical Neurophysiology, 118(10), 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019

16. Sheline, Y. I., Price, J. L., Yan, Z., & Mintun, M. A. (2010). Resting-state functional MRI in depression unmasks increased connectivity between networks via the dorsal nexus. Proceedings of the National Academy of Sciences, 107(24), 11020–11025. https://doi.org/10.1073/pnas.1000446107

17. Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. Biological Psychology, 71(1), 1–13. https://doi.org/10.1016/j.biopsycho.2005.01.005

18. World Health Organization. (2023). Depression and other common mental disorders: Global health estimates. World Health Organization.