Pakistan Journal of Advances in Medicine and Medical Research (PJAMMR)
ISSN: (Online) 2957-5893
Website: https://www.pjammr.com/index.php/pjammr/homepage

Open Access
Volume 4, Issue 2, 2026

Narrative review

Neuroimaging Findings in Major Depressive Disorder: An Integrative Narrative Review

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Keywords: Major depressive disorder, neuroimaging, resting-state functional MRI, functional connectivity, network neuroscience

Article History

Received: April 01, 2026
Revision: May 24, 2026
Accepted: June 05, 2026
Published: July 10, 2026

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DOI:10.69837/pjammr.v4i2.152

How to Cite

Akram A. Neuroimaging Findings in Major Depressive Disorder: An Integrative Narrative Review. Original Article. Pak J Adv Med Med Res. 2026;4(2):4–11.

ABSTRACT

Objective: To critically evaluate the alterations in large-scale brain networks and corticolimbic connectivity associated with major depressive disorder (MDD), focusing on their consistency, clinical significance, and reproducibility across neuroimaging studies.

Methodology: I categorized the 35 author-supplied publications by evidence type, anatomical scale, clinical context, and neuroimaging approach. I distinguished empirical studies from reviews, methodological reports, and studies of related disorders.

Results: Default mode, control, salience-related, amygdala–hippocampal, and anterior cingulate circuits have repeatedly been implicated in MDD. However, no single abnormal connection or uniform direction of connectivity change has emerged. Nuclei-specific and dynamic techniques provide complementary information, although they introduce trade-offs involving measurement reliability and multiple testing. Large-scale datasets strengthen confidence in some small population-level associations while also revealing the limits of case–control findings and imaging-only prediction.

Conclusions: A distributed, clinically heterogeneous model that accounts for measurement scale is the most defensible account of MDD. Translating imaging findings into clinically useful biomarkers will require specific hypotheses, careful image-quality control, transparent statistical correction, longitudinal assessment, and genuinely independent cross-site replication.

1. Introduction

Depression is not just one neurobiological state. Psychomotor changes, anxiety, loss of interest in activities (anhedonia), sleep, cognition, illness duration, and previous antidepressant treatment may differ between two individuals who meet the diagnostic criteria for major depression. These differences may be reflected in both the mean and the variation of functional connectivity estimates. Therefore, a repeated group difference may not appear in every clinical subgroup. This is important when interpreting the network-level syntheses of Kaiser et al. (2015) and Mulders et al. (2015), as well as more recent population-level evidence (Hamilton et al., 2026). Resting-state fMRI measures fluctuations in the blood-oxygen-level-dependent signal and estimates statistical dependence among brain regions or networks. A functional correlation is not an axonal tract, and a stronger correlation does not necessarily indicate more effective neural communication. Hemodynamic variability, physiological noise, head motion, preprocessing, and the definition of region boundaries further complicate interpretation. For this reason, the term “altered connectivity” is used rather than assuming that every difference represents a deficit or dysfunction.The literature examines a wide range of spatial scales, from intrinsic connectivity networks to specific amygdala nuclei and anterior cingulate subdivisions. A whole-network finding and a subregional finding may both be credible even if they point in different directions, because they describe different spatial units. Careful consideration of scale, clinical samples, analysis methods, and the intended inference is therefore essential (Tang et al., 2018; Jacob et al., 2022; Klein-Flügge et al., 2022). One of the main challenges in this field is that “abnormal connectivity” is often used to describe different phenomena: an average correlation between two regions, the functional integration of a network, changes in temporal configurations, or a predicted clinical outcome. These measures differ in their biological meaning and statistical requirements. A sound synthesis must therefore consider not only what was measured but also which inferences a study can support. Comparisons across studies also require caution when different atlases are used or patients are examined at different stages of illness (Kaiser et al., 2015; Mulders et al., 2015; Tang et al., 2018).

Newer population studies bring effect-size stability and generalizability into the discussion, moving beyond isolated, statistically significant connections. For instance, Hamilton et al. (2026) examined structural and functional correlates of depression in 23,417 participants from six population datasets. However, the cohorts differed in age and in their assessment of depressive symptoms. Such evidence helps explain population-level associations, but it does not, by itself, validate a diagnostic test for an individual patient. A balanced assessment should connect these larger studies with anatomically focused, mechanistic work.

2. Review question and objectives

Research question: Which alterations in large-scale brain networks and corticolimbic connectivity are most consistently seen in MDD, and how do anatomical specificity, clinical heterogeneity, and methodological differences influence their interpretation and reproducibility?

This review aims to synthesize recurring neural systems; consider whether anatomically refined limbic and anterior cingulate analyses provide information beyond whole-region findings; distinguish diagnostic associations from symptom severity and treatment-response prediction; and assess the methodological conditions under which findings may replicate. These aims address one overarching research question rather than separate hypotheses.

3. Review design and evidence synthesis

3.1 Review type and source identification

This paper is an integrative narrative review of 35 selected publications supplied by the author. The sources include original investigations, systematic and narrative reviews, methodological contributions, and studies of other psychiatric disorders included only for comparison and to provide context for the diagnostic specificity of MDD findings.

3.2 Organization and evaluation of the evidence

Publications were classified qualitatively along 5 dimensions: (1) anatomical scale (whole brain, intrinsic networks, circuits, nuclei, or subfields); (2) neuroimaging modality (resting-state fMRI, task fMRI, structural or diffusion MRI, or electrophysiology); (3) population (adults, adolescents, first-episode or recurrent illness, treated or drug-naïve participants); (4) inferential purpose (case–control comparison, symptom association, treatment-related change, or prediction); and (5) methodological features (sample size, site diversity, region definition, and validation strategy). Interpretations were assessed for relevance, directness, and reproducibility, rather than statistical significance alone.

3.3 Boundaries of interpretation

Studies of ADHD, bipolar disorder, and social anxiety were used only to raise cross-diagnostic issues (Gao et al., 2019; Syan et al., 2018; Young et al., 2017). Importantly, Jacob et al. (2022) used high-resolution structural and diffusion MRI rather than resting-state fMRI. Its contribution here concerns the anatomical and connectomic specificity of limbic subregions, not resting-state functional coupling.

3.4 Strengths and limits of integrative synthesis

Selection and interpretation bias are central limitations, particularly when the body of literature is determined beforehand. To minimize overinterpretation, this review distinguishes repeated investigation of a neural system from replication of a specific, directionally consistent effect. It also acknowledges null findings, alternative explanations, and the lack of clinically validated imaging tests.

4. Evidence synthesis

4.1 Large-scale brain networks

Commonly examined resting-state networks include the default mode network (DMN), frontoparietal control circuitry, salience-related networks, and interactions among them. Kaiser et al. (2015) reviewed changes in coupling across these systems, while Mulders et al. (2015) discussed differences in acquisition, sample composition, and analysis. The DMN includes regions associated with internally oriented processes, such as medial prefrontal and posterior midline areas. Its relevance to rumination in depression is clear, but not every DMN change should be treated as a marker of rumination. As Kaiser et al. (2015), Yan et al. (2019), and Zhang et al. (2024) emphasize, within-network integration, between-network coupling, and global connectivity summaries need to be distinguished. Frontoparietal control regions are associated with the flexible allocation of cognitive resources, while salience-related regions help detect and respond to relevant stimuli. Network coupling may vary with vigilance, cognitive state, and symptom dimensions. A finding limited to one connection between control and salience systems should not be extended to entire networks without convergent evidence (Kaiser et al., 2015; Luo et al., 2021). Models focused only on emotion circuitry may overlook sensorimotor and cerebellar findings. Global connectivity research has highlighted sensorimotor and default mode systems (Zhang et al., 2024), while Zhu et al. (2020) examined cerebellar–cerebral dynamic coupling. A useful comparison concerns the DMN. Yan et al. (2019) reported reduced DMN connectivity in recurrent MDD, while studies based on global connectivity or between-network metrics may emphasize other patterns (Zhang et al., 2024). Reduced integration within a network, increased coupling with an external circuit, and reduced global connectivity are three different measures. Even when results appear to contradict one another, they may not be directly comparable unless they examine the same anatomical relationship and statistical contrast. Age, cognitive state, nuisance regression, and global signal treatment further complicate comparison. Developmental and illness-history subgroups offer another reason to avoid universal descriptions. Research on adolescent depression (Ho et al., 2015; Lee et al., 2019) or drug-free adults (Luo et al., 2021) may not capture the same patterns as research on recurrent or treated adult MDD. The broader conclusion is the repeated involvement of large-scale networks, while more specific claims about the direction and location of effects remain dependent on the population and analysis.

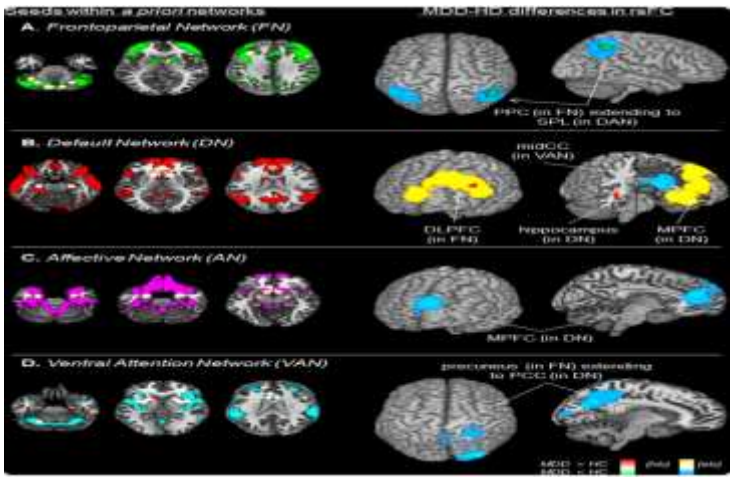


Figure 1. Resting-state functional connectivity differences between MDD and healthy controls across frontoparietal, default, affective, and ventral attention networks. Reproduced from the supplied published figure; original article attribution and reuse permission require verification.

4.2 Amygdala and hippocampal subregional organization

The amygdala is not a homogeneous region. Signals from nuclei with different structures and connections may cancel one another or reduce sensitivity to local changes when combined into broad seeds. Tang et al. (2018) examined amygdala resting-state connectivity in adults and adolescents with depression, while Guan et al. (2024) examined amygdala subregions. Together, these investigations make anatomical scale an important issue, but they do not establish that every subregional effect will appear consistently under different parcellations. The hippocampus supports memory, contextual representation, and affective processing. Differences between anterior and posterior regions, as well as among subfields, may help explain why whole-hippocampus connectivity findings vary across studies. Jacob et al. (2022) discuss the hierarchy of hippocampal and amygdala subregions, showing why spatial resolution is part of the biological question and not merely a technical detail. Nuclei-specific seeds may be small relative to the resolution and smoothing kernel of functional images. Estimated correlations can be sensitive to atlas boundaries, partial-volume effects, registration accuracy, and signal-to-noise ratio. Theoretical specificity must therefore be balanced against measurement reliability. Sensitivity analyses using alternative atlas definitions, bilateral homologs, and minimum ROI coverage thresholds can help determine whether an apparent effect is robust. A useful multimodal example comes from Jacob et al. (2022), who investigated amygdala and hippocampal subregion connectivity in patients with MDD (n = 38) and healthy controls (n = 40) using 7-Tesla anatomical and diffusion MRI. This structural connectomic approach complements BOLD correlation analyses, but graph-theoretic measures from diffusion imaging should not be presented as evidence of altered resting-state functional connectivity. Klein-Flügge et al. (2022) also examined nuclei-specific amygdala connectivity in relation to mental-health dimensions, illustrating the potential value of finer-grained circuits. Small seeds introduce a difficult trade-off: parcellation may improve biological specificity while reducing reliable voxel coverage and increasing the number of tests. Smoothing, head motion, and registration can disproportionately influence signal estimates. Biologically meaningful subregional findings should therefore be supported by coverage checks, clearly defined test families, multiple-testing corrections, and sensitivity analyses using alternative atlas boundaries. Beyond typical frontal targets, Wong et al. (2022) found an association between amygdala–pons coupling and depressive symptom severity, while Liu et al. (2024) explored transcriptional correlates of amygdala functional connectivity in first-episode, drug-naïve MDD. Yang et al. (2017) also examined amygdala atrophy and its functional disconnection from a cortico-striatal-pallidal-thalamic circuit in women with MDD. These findings broaden the scope for generating hypotheses, but molecular annotation of an imaging pattern does not establish that particular genes cause an individual's connection pattern or symptoms.

4.3 Prefrontal–limbic and anterior cingulate systems

Prefrontal–amygdala connectivity is often discussed in relation to emotion regulation, but resting-state correlations alone cannot demonstrate top-down causal control by the prefrontal cortex. Zhang et al. (2020) examined its relationship with depressive status, while Wu et al. (2020) included both functional and structural measures in first-episode adolescent depression. Differences in population and imaging approach limit direct pooling, although both studies highlight the relevance of prefrontal–limbic circuits. Areas within the anterior cingulate cortex (ACC) show different connectivity patterns and proposed roles in affective and cognitive processes. Zhang et al. (2022) and Zhou et al. (2024) support subdivision-aware analyses. The term ACC alone does not specify whether a finding concerns subgenual, pregenual, dorsal, or another area; the nomenclature and atlas definition should be stated. An electrophysiological study also examined prefrontal and posterior cingulate activity in MDD (Lin et al., 2021), although those measurements are not equivalent to resting-state fMRI connectivity. Acute stress may alter prefrontal–amygdala coupling, making current state an important consideration (Hossein et al., 2023). Adolescents may differ from adults in development and clinical presentation (Lee et al., 2019; Wu et al., 2020). Medication exposure, illness recurrence, and scan context further limit simple comparisons of the direction of reported effects. The ACC includes ventral, subgenual, pregenual, and dorsal divisions with different connectivity profiles. Zhang et al. (2022) examined functional connectivity in emotional ACC subregions, and Zhou et al. (2024) systematically reviewed intrinsic connectivity findings by subdivision. Averaging across the whole ACC may hide these differences, although small regions remain vulnerable to registration and parcellation issues. Findings should identify the anatomical definition of the subdivision and avoid using ACC and subgenual ACC interchangeably. The clinical relevance of the subgenual cingulate and nuclei-specific fronto-amygdala pathways is considerable, particularly in treatment research. Xue et al. (2026) examined a receptor-related fronto-amygdala pathway in relation to antidepressant selection, while Tura and Goya-Maldonado (2023) reviewed connectivity in the context of precision treatment. These studies offer hypotheses for further testing rather than proof that an MRI scan can already reliably select medication for an individual patient. Other mechanisms may connect hippocampal, cognitive, and limbic circuits. Ekpo et al. (2025) compared medial prefrontal–hippocampal connectivity during rest and task conditions, helping to clarify how a connection observed during an unconstrained resting scan may differ from one observed during active cognitive or emotional engagement. Interpretation is stronger when a study specifies whether it investigates a stable intrinsic association or a context-dependent response.

4.4 Dynamic functional connectivity and temporal states

Static functional connectivity measures average dependence between signals over a recording. Dynamic approaches, such as sliding windows and co-activation patterns, instead aim to describe changing configurations that averaging may obscure. An et al. (2024) examined resting-state co-activation dynamics, Javaheripour et al. (2023) investigated state- and trait-related features in MDD, and Zhu et al. (2020) focused on cerebellar–cerebral dynamic connectivity. These approaches address related, but not identical, temporal processes. Short recordings may produce unreliable temporal estimates, particularly when models have many parameters. Motion bursts, respiratory variation, drowsiness, and scanner drift can be mistaken for connectivity changes. Researchers should report framewise displacement, temporal filtering, scan duration, state-definition choices, and sensitivity to alternative parameter settings. Test–retest reliability is needed before claiming that a dynamic measure is clinically useful for an individual.

A state-sensitive marker changes with current symptoms or context, while a trait-related marker remains relatively stable over time and across clinical phases. This distinction requires empirical testing through repeated scans and symptom measurements. Javaheripour et al. (2023) highlight the issue, but cross-sectional differences alone cannot establish whether a measure is state-like or trait-like. The main methodological question is how to distinguish meaningful neural reconfiguration from noisy estimation. Apparent states may change with scan length, arousal, window selection, respiration, head motion, or model complexity. Even when a study finds large differences in state occupancy, those states may not remain stable across sessions or predict symptoms better than a static correlation. Prospective comparisons with matched preprocessing and independent testing are therefore important.

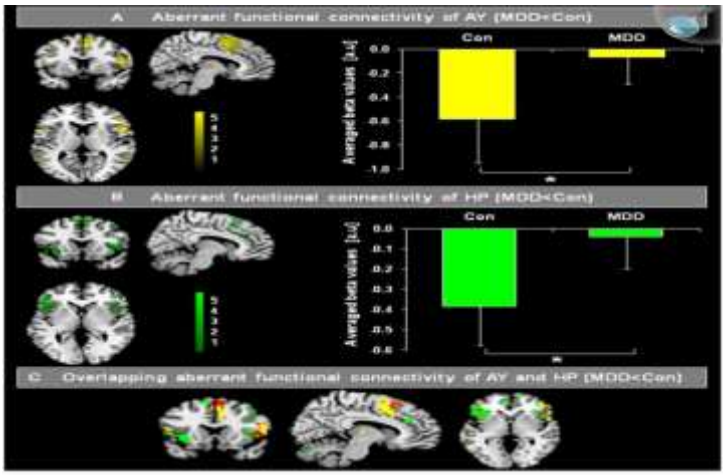


Figure 2. Aberrant functional connectivity of AY and HP in MDD relative to controls, including overlapping alterations (panels A–C). Anatomical abbreviations, source, and reuse permission require verification.

4.5 Clinical symptoms, heterogeneity, and treatment outcomes

Average differences between diagnostic groups and associations with symptom severity do not necessarily answer the same question. A connection between two regions may differ between cases and controls without varying directly with the Beck Depression Inventory or another symptom scale. Conversely, a symptom association may appear across psychiatric diagnoses without being specific to MDD. Restricted symptom distributions, differing scales, and adjustment for different covariates can create apparent contradictions between studies. Estimated associations may depend on age, sex, episode status, medication exposure, comorbidity, symptom subtype, and treatment history. Covariate adjustment can reduce confounding, but it can also change the analysis target. Studies should justify covariates in advance and distinguish possible confounders from variables that may mediate the relationship between illness and connectivity. Drysdale et al. (2017) proposed resting-state connectivity-based depression subtypes, bringing attention to treatment-oriented biological stratification. Later methodological work questioned the reliability of these clusters under different samples and statistical assessments. Biotypes should therefore be evaluated for stability across resampling, replication in independent clinical cohorts, and prospective prediction of treatment response, rather than only by how well clusters separate within the dataset in which they were identified. In a multisite study, Lan et al. (2025) examined emotion-regulation network connectivity and its relationship with symptom improvement. Wong et al. (2022) investigated an anatomically targeted connection in relation to symptom severity. These studies provide promising clinical hypotheses, but they do not establish that changing a connectivity measure would alter symptoms or that a fitted model would work well in an independent clinic. A neuroimaging model should be compared with simpler baseline measures, such as demographic characteristics, symptom severity, and previous treatment response. Statistical significance alone does not show that imaging will improve clinical decisions. A useful biomarker should also be feasible, reliable, affordable, interpretable, and robust to differences in scanning site and patient population (Tura & Goya-Maldonado, 2023). A translational imaging study should define the clinical decision it aims to support before fitting a model: identifying risk, distinguishing diagnoses, predicting severity or treatment response, or monitoring recovery. Test performance in genuinely held-out participants, and ensure the imaging model adds value over simpler, less expensive clinical measures. Even a reproducible group effect may be too small or variable to help at the individual-patient level.

4.6 Multisite replication and population-scale evidence

Multisite samples offer opportunities for broader generalization, but they also create challenges for measurement comparability. Scanners, acquisition protocols, demographics, clinical interviews, recruitment practices, medication exposure, and head-motion profiles may vary between sites. If patient proportions differ by site, technical differences may be mistaken for disease effects. Pooling can increase statistical power, but a pooled effect alone does not show that the association is the same at every center. Marek et al. (2022) showed why reproducible brain-wide associations often require much larger samples than traditional single-site studies. Hamilton et al. (2026) assessed brain correlates of depression in six population samples comprising 23,417 participants. Their findings strengthen the study of depression-related brain anatomy and functional organization at the population level, while showing that individual associations are often modest and that clinical phenotypes differ across datasets. Targeted multisite studies provide complementary evidence. Zhang et al. (2023) examined raphe nuclei functional connectivity, and Lan et al. (2025) investigated emotion-regulation networks. Ding et al. (2026) examined distance-related connectivity patterns in first-episode, drug-naïve MDD. Because these studies address different anatomical and clinical questions, it is important to distinguish evidence of some cross-site signal from replication of the same preselected connection. Statistical harmonization methods, including ComBat-type adjustments, may reduce scanner-related distributional differences, but they cannot replace representative data or careful assessment of confounding. If a harmonization model is fitted using information from the held-out test set, prediction performance may be biased. Fitting preprocessing on training data only and evaluating the model at a fully independent site provides a stronger estimate of generalizability. Authors should report site-specific effects and heterogeneity alongside pooled estimates, including null and inconsistent results.

5. Critical synthesis and comparison

5.1 Where does the evidence converge?

One of the clearest points of agreement across these studies is the involvement of distributed networks and limbic–cingulate systems in depression-related processes. This is an anatomical and conceptual convergence: the repeatedly examined regions support emotion, cognition, salience, and autobiographical processing. It is not the same as consistent evidence for one direction of connectivity change or one abnormal

connection specific to MDD. Keeping this distinction in mind helps prevent exaggerated agreement between network meta-analyses, subregional studies, structural connectomics, and population data. The most credible findings come from studies with well-defined clinical phenotypes, adequate sample sizes, prespecified anatomical targets, appropriate multiple-comparison correction, and independent replication. Large population datasets improve precision but may have limited clinical specificity. Reviews and meta-analyses can identify recurring patterns, but they remain vulnerable to heterogeneity and publication bias in the underlying literature. Smaller, carefully phenotyped cohorts may offer mechanistic insight but remain limited for biomarker development.

5.2 What explains apparently conflicting results?

Several factors may explain inconsistent findings. First, adolescent and adult patients differ developmentally, while treatment and recurrence of illness may also change observed associations. Second, studies examine different statistical measures, such as a connection between two regions, a network-wide summary, a dynamic occupancy measure, or a structural centrality index. Third, atlases, preprocessing pipelines, and correction methods can alter estimates. Fourth, small exploratory analyses are more vulnerable to unstable effect sizes and false discoveries. These differences should be examined before opposing findings are called direct replication failures. Comparison should begin with the closest possible match in population, ROI definition, direction of contrast, imaging modality, and treatment of covariates and noise. A significant effect at one site and a nonsignificant effect at another do not establish a site interaction unless the between-site difference is itself tested and its uncertainty reported. Likewise, a nonsignificant corrected result should not be described as proof that a circuit is unaffected.

5.3 Anatomical resolution versus measurement stability

Whole-network summaries may combine biologically distinct components, yet they can be relatively resistant to small registration differences. Nuclei-specific seeds may be more suitable for testing precise circuitry, but they often contain few usable voxels and can amplify noise. A hierarchical strategy is useful: define the main circuit-level question in advance, then examine narrower anatomical hypotheses with transparent family-wise error or false discovery rate correction and sensitivity checks. If a subregional finding disappears after small boundary changes, it may reflect uncertainty rather than clear biological localization.

5.4 Diagnostic association versus clinical usefulness

A consistent group difference, a symptom correlation, a mechanistic hypothesis, and an accurate prediction model are four separate achievements that should be assessed on their own merits. A clinical-use claim needs prospective, externally validated prediction with meaningful added benefit. Functional connectivity may eventually supplement clinical information for stratification or treatment monitoring, but current evidence does not establish it as a universal stand-alone biomarker for MDD.

Table 1. Evidence domains and interpretation of the representative literature.

Evidence domain	Representative publications	Critical appraisal
Distributed resting networks	Kaiser et al., 2015; Mulders et al., 2015; Yan et al., 2019; Zhang et al., 2024	Repeated network involvement; precise direction depends on connectivity metric and sample.
Amygdala and hippocampal anatomy	Tang et al., 2018; Jacob et al., 2022; Guan et al., 2024; Liu et al., 2024	Subregion specificity is informative but not functional, and diffusion-based results are not equivalent.
Prefrontal and ACC circuits	Wu et al., 2020; Zhang et al., 2022; Zhou et al., 2024	Subdivision, developmental stage, and modality affect interpretation.
Temporal connectivity	Zhu et al., 2020; Javaheripour et al., 2023; An et al., 2024	Potential state sensitivity requires motion control, stability, and incremental validity.
Population and external replication	Marek et al., 2022; Lan et al., 2025; Hamilton et al., 2026	Independent samples are necessary; site and phenotype differences remain important.

6. Methodological recommendations for future research

A staged evaluation is a practical way to align statistical claims with the evidence required to support them. First, determine whether a prespecified case–control effect remains after adjustment and appropriate multiple-testing correction. Second, assess whether the same association relates to symptom severity, reporting effect estimates and uncertainty; third, test sensitivity to atlas definitions, nuisance models, quality-control thresholds, and clinically relevant subgroups. Fourth, examine replication and transfer to independent sites or cohorts. These are connected questions, but they are not interchangeable.

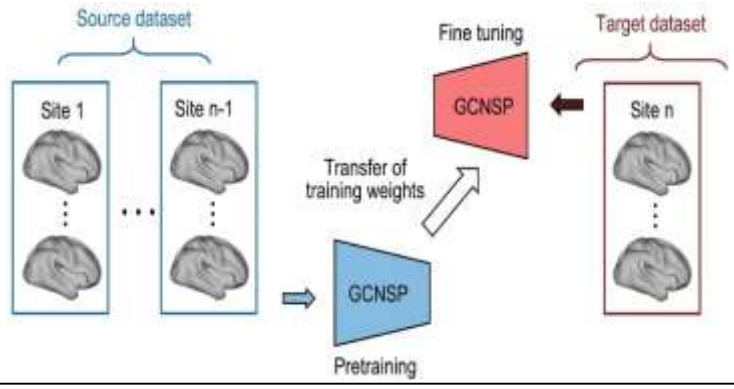


Figure 3. MDD classIdentification probability distributions. Contributions in discovery and independent validation datasets, shown overall and by imaging site. From the user-supplied published figure; confirm exact source and reuse license before submission.

Longitudinal phenotyping is particularly valuable. Repeated symptom ratings and MRI scans can help distinguish within-person variation from between-person variation and determine whether a proposed marker is sufficiently stable to be useful. Where possible, studies should measure anhedonia, cognitive symptoms, anxiety, medication exposure, and episode history rather than treating MDD as a single, uniform state. Primary analyses should be planned before the data are examined in multi-resolution designs. Broad network analyses can be combined with targeted ROIs in limbic and anterior cingulate regions, but recognize that additional seeds and models increase opportunities for false discovery. Convergence across structural MRI, diffusion MRI, electrophysiology, and task fMRI can provide multimodal support, but causal language should be reserved for study designs and analyses that justify it. Finally, reporting should clearly describe reproducibility. Studies should describe acquisition parameters, atlas versions, nuisance regressors, motion metrics, denoising methods, statistical thresholds, test families, sensitivity analyses, and model-development steps. Where ethically and legally appropriate, sharing de-identified derived data and executable code may help other researchers assess and build on the findings.

8. Conclusion

The neuroimaging literature supports a network-based understanding of MDD, with recurring involvement of amygdala and hippocampal circuits, anterior cingulate subdivisions, prefrontal networks, and other large-scale intrinsic networks. However, no single connectivity change has been consistently directional and specific across all patients with MDD. Apparent disagreements often reflect differences in clinical phenotype, anatomical resolution, imaging modality, statistical contrast, or preprocessing rather than opposing results for the same biological effect. Future progress requires linking anatomically specified models with reliable measurements, clear inferential logic, and independent replication. The path toward clinical translation begins with reproducible group and symptom-severity associations, confirmation that results remain robust under alternative analytical choices, and tests of whether a marker provides useful prospective information in new populations. Until those conditions are met, MDD connectivity findings should be presented as informative research observations rather than routine clinical diagnostic tools

Ethics Approval and Consent to Participate

Ethical approval was not required for this study because it is an integrative narrative review based exclusively on previously published literature. No human participants were recruited, no animal experiments were conducted, and no identifiable patient data were collected or analyzed. All sources have been appropriately acknowledged and cited in accordance with accepted academic standards.

Authors' Contributions

Arfa Akram conceptualized the review, conducted the literature review, collected and analyzed the relevant published evidence, interpreted the findings, and drafted the manuscript. The author critically revised the manuscript, approved the final version, and takes responsibility for the integrity and accuracy of the work.

Conflict of Interest

The author declares that there are no conflicts of interest related to the publication of this article.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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