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Review Article

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Bridging Neuroscience and Psychiatry through Brain Complexity and Neural Circuit Dysfunctions in Anxiety, Depression, and Schizophrenia

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Abstract

Anxiety, depression, and schizophrenia are complex psychiatric disorders characterized by disruptions in neural circuits, neurotransmitter systems, and brain connectivity, resulting in impairments in emotional regulation and cognitive functioning. This review examines the genetic, environmental, and neurobiological factors influencing these conditions, emphasizing the critical roles of neurotransmitters such as serotonin, dopamine, and norepinephrine in mood regulation, stress response, and neuroplasticity. These findings underscore the need for personalized treatment approaches. The review also explores integrative strategies that combine pharmacological interventions with non-pharmacological modalities, including acupuncture, herbal medicine, and mindfulness, which show promise for individualized care. Advances in neuroimaging and neurostimulation techniques, such as eigenvector centrality mapping and machine learning-driven analyses, provide deeper insights into brain connectivity and enable more targeted interventions. This is particularly significant for schizophrenia, where dopamine-mediated disruptions in striatal-prefrontal connectivity contribute to cognitive deficits and clinical symptoms. However, current limitations, such as an inadequate understanding of the neural circuits underlying these disorders and the limited effectiveness of conventional treatments for certain subgroups, highlight critical gaps in existing research and therapeutic approaches. Furthermore, the article discusses how integrating computational models with traditional medicine enhances our understanding of neurotransmitter interactions and neural pathways. This integration fosters innovative therapies that address both immediate symptoms and long-term resilience. This interdisciplinary approach bridges basic neuroscience and clinical practice, paving the way for effective, personalized treatments and offering renewed hope for individuals with psychiatric disorders.

Keywords: Anxiety; Depression; Schizophrenia; Neuroimaging and neurostimulation; Neural circuits; Neurotransmitter; Machine learning translational medicine; Integrative neuroscience.

Introduction

The brain's neural circuits play a pivotal role in regulating emotional states, particularly in the context of anxiety and depression.[1] These circuits, consisting of interconnected brain regions and complex networks, underlie the processing and regulation of emotional stimuli.[2,3] Disruptions within these neural circuits contribute to the maladaptive behaviors and emotional dysregulation observed in these disorders.[4–6] Recent advancements in neuroscience have shed light on specific pathways involved, including the prefrontal cortex (PFC), amygdala, and hippocampus. These regions are central to both emotional regulation and memory.[7,8] The interactions between these structures, modulated by neurotransmitters such as serotonin, dopamine, and norepinephrine, are critical to understanding how the brain transitions from a healthy emotional state to one marked by anxiety or depression.[9–11] Investigating these neural circuits provides insight into how these brain regions communicate and how their dysfunction leads to the persistence of negative emotional biases. This understanding forms the foundation for developing targeted therapeutic strategies aimed at restoring balance within these neural systems and improving outcomes for individuals with these mental health conditions.

Schizophrenia (SZ) is a complex neuropsychiatric disorder characterized by disruptions in cognitive, emotional,

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and perceptual functioning, with its pathophysiology closely linked to altered neural circuits.[12–14] These dysfunctions often involve abnormal connectivity patterns across brain regions, impairing the dynamic integration of information essential for adaptive behavior. The neural circuits implicated in SZ, particularly those within the PFC, striatum, and hippocampus, are critical for executive functions, memory, and sensory processing.[15–17] Disruptions in the connectivity and complexity of these circuits may underlie the cognitive impairments and clinical symptoms observed in SZ patients, including deficits in working memory, attention, and executive control.[18–20] Recent research has increasingly focused on brain complexity as a critical measure of network dynamics, highlighting the brain's capacity to integrate information and adapt to environmental demands.[6,21] Studies in this domain examine neural circuit dysfunctions in SZ using advanced brain imaging techniques to gain insights into the disorder's underlying mechanisms and its response to treatment.[22] By integrating complexity theory with neurobiological perspectives, this research aimed to deepen our understanding of SZ's neurophysiological foundations.

The intricate neural circuits involved in psychiatric disorders such as anxiety, depression, and SZ are central to the pathophysiology of these conditions, influencing emotional regulation, cognitive function, and behavior.[23–25] These circuits are often dysregulated, making treatment challenging, particularly with conventional pharmacological approaches. Neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and electroencephalography (EEG), have enhanced our understanding of these neural networks, revealing both structural and functional abnormalities underlying these disorders.[26–28] Concurrently, neurostimulation technologies, including transcranial electrical stimulation (TES), deep brain stimulation (DBS), and ultrasound neurostimulation, offer non-invasive alternatives to modulate these disrupted circuits and promote therapeutic effects.[29–31]

This review explores the complex interplay between neural circuits, neurobiology, and the treatment of psychiatric disorders, specifically anxiety, depression, and SZ. It examines the neurobiological mechanisms underlying these conditions, focusing on how disruptions in neural circuits contribute to the pathophysiology of mental illness. The limitations of traditional pharmacological approaches are discussed, alongside the genetic and connectivity alterations observed in SZ, which provide insights into how these changes affect brain function and treatment response. The latter part of the article highlights promising advancements in neuroimaging and neurostimulation techniques, emphasizing their translational potential in improving the diagnosis and treatment of anxiety, depression, and SZ. Through this analysis, the review aimed to bridge the gap between basic neuroscience and clinical practice, offering new avenues for more personalized and effective interventions.

This study reviews the literature integrating neuroscience and translational medicine to explore neural circuitry mechanisms in mood disorders (anxiety, depression), SZ, and treatment responses. The review examines brain complexity, cognitive function, neuroimaging, and neurostimulation, aiming to identify therapeutic targets for translational applications. Relevant databases such as PubMed, Scopus, Web of Science, and PsycINFO were searched using terms related to mood disorders, neural mechanisms, neurotransmitters, and therapeutic interventions. Studies published between 2014 and 2024, along with key earlier works, were included, focusing on peer-reviewed articles, systematic reviews, and clinical trials. Non-peer-reviewed studies, opinion pieces, and those not relevant to the disorders were excluded. References were managed using Zotero or EndNote, with Rayyan aiding in the screening process.

Integrating neuroscience and translational medicine in the neural circuitry mechanisms of anxiety and depression

Anxiety and depression are among the most prevalent mental health conditions worldwide, affecting millions of individuals. Despite their global significance, current treatments often fall short, providing limited relief for many.[32,33] This shortfall largely arises from an incomplete understanding of the neural mechanisms underlying these disorders. However, recent advances in neuroscience are beginning to unravel the neural circuits and neurochemical interactions involved in the shift from healthy to pathological brain states.[34,35]

Examining the neural circuitry underlying anxiety and depression raises several critical questions: What drives the transition from a healthy to a diseased brain state? How does this shift alter the brain's perception and processing of stimuli? Which neural pathways and neurotransmitters play a central role in this transformation? Addressing these questions requires a framework that integrates computational, algorithmic, and implementation perspectives on brain function and dysfunction.[36–38] Drawing inspiration from David Marr's three-level model, this analysis explores each level to investigate how disruptions in top-down and bottom-up brain processes contribute to the distinct neural states characteristic of anxiety and depression.[39,40]

In essence, the brain can be viewed as shifting between various "attractor states"—stable configurations representing specific patterns of neural activity.[41,42] Under normal conditions, these states enable the brain to process external stimuli and return to a baseline, healthy condition.[43,44] In anxiety and depression, however, the brain may settle into maladaptive attractor states that prevent a return to baseline, perpetuating negative processing biases and maintaining pathological states. By targeting these shifts, therapeutic interventions might restore the brain's resilience and adaptability.

Algorithmic perspectives on emotional processing

From an algorithmic perspective, altered emotional processing mechanisms are key to understanding and addressing the persistence of anxiety and depression.[45,46] In these mental health conditions, emotional valence processing—essentially the brain's interpretation of stimuli as positive, neutral, or negative—becomes skewed.[47,48] This skewed interpretation causes individuals with anxiety or depression to perceive stimuli differently from those without these conditions. For instance, a facial expression or tone of voice that might be perceived as neutral by a healthy individual could be interpreted as hostile, threatening, or distressing in someone experiencing anxiety or depression.[49,50] This altered processing can reinforce a cycle of negative emotions, where benign stimuli are regularly perceived as adverse or potentially harmful. As

a result, these biases become ingrained in daily interactions, contributing to a self-sustaining cycle of negative emotions and cognitive distortions.

To address these persistent distortions, researchers employ computational models that simulate how cognitive biases contribute to the maintenance of anxiety and depression.[51–54] By analyzing the algorithms governing emotional responses, researchers can better understand the mechanisms that reinforce maladaptive processing. These models often reveal that emotional processing is guided by the brain's tendency to prioritize certain types of information, a process influenced by previous experiences, genetic factors, and neural structure. In anxiety, for instance, there is often a bias toward threat-related information,[55,56] while in depression, there is a tendency to focus on negative self-referential thoughts.[51,57,58] These patterns not only shape how individuals experience their surroundings but also limit their ability to interpret stimuli as positive or neutral, reinforcing cycles of anxiety or depressive episodes.

Key brain structures, particularly the PFC and amygdala, are central to this biased processing in both top-down and bottom-up emotional regulation.[59–61] The PFC, responsible for executive functions like decision-making and emotional regulation, plays a top-down role by modulating emotional responses and suppressing excessive fear reactions.[62,63] In healthy individuals, the PFC regulates the intensity and appropriateness of responses generated by the amygdala, a structure responsible for detecting threats and initiating responses to perceived danger.[64–66] However, in individuals with anxiety or depression, disruptions in the interaction between the PFC and amygdala lead to patterns of hypervigilance or hypersensitivity to negative stimuli.[38,64,67] These maladaptive processing patterns prevent individuals from appropriately regulating emotional responses, sustaining symptoms of these disorders and interfering with healthy emotional functioning.

Therapeutic approaches targeting PFC-amygdala interactions offer promising avenues for restoring adaptive emotional regulation. For example, cognitive-behavioral therapy employs strategies to recalibrate biased cognitive processes, aiming to reduce maladaptive responses by encouraging the PFC to override automatic, distressing reactions.[68–71] Emerging neuromodulatory therapies, such as transcranial magnetic stimulation (TMS), also show potential in altering dysfunctional PFC-amygdala connectivity.[72–74] By targeting the neural circuits involved in emotional valence processing, these approaches aim to realign the brain's interpretation of stimuli, promoting a more balanced and accurate assessment of the surrounding environment.

Algorithmic analyses of emotional processing in anxiety and depression offer insights into how cognitive biases are sustained at the neural level. By focusing on the disrupted connectivity between the PFC and amygdala, researchers and clinicians can develop interventions that re-establish adaptive processing patterns, providing individuals with more effective tools for managing and ultimately overcoming these mental health challenges.

Neuromodulatory systems in emotional regulation and synaptic plasticity: Serotonin, dopamine, and norepinephrine

Neuromodulatory systems, particularly those involving serotonin (5-HT), dopamine, and norepinephrine, are fundamental to emotional processing, synaptic plasticity, and cognitive functioning.[75–79] These neurotransmitters each play a unique role in emotional regulation, and their dysfunction is closely linked to anxiety and depression.[80–82]

Serotonin (5-HT) plays a pivotal role in emotional regulation by influencing the processing of both threatening and benign stimuli.[83,84] The amygdala, a central structure in emotional processing, is particularly affected by serotonin, which modulates fear memory formation. For instance, increased serotonin levels in the basolateral amygdala during fear-inducing situations promote the consolidation of fear responses.[85,86] Studies using genetically modified animal models have demonstrated that serotonin influences both fear learning and extinction by modulating synaptic plasticity in the amygdala, which is crucial for emotional memory formation.[87] Beyond its role in fear, serotonin also enhances positive emotional processing.[88] Treatments such as selective serotonin reuptake inhibitors (SSRIs), which boost serotonin activity, have been shown to shift emotional biases towards positive stimuli, underscoring serotonin's involvement in mood regulation and synaptic plasticity, particularly in regions like the medial PFC (mPFC).[86,89–92]

Dopamine is essential for reward processing, motivation, and reinforcement learning, particularly within the mPFC and its associated circuits.[93,94] It regulates emotional responses by signaling reward prediction errors, which are crucial for adapting to both positive and negative experiences.[95] Dysregulated dopamine signaling, often seen in conditions such as anhedonia and major depression, impairs this adaptive function.[11,80] Abnormal dopamine release in the mPFC and ventral tegmental area circuits can contribute to core depressive symptoms, such as reduced motivation and pleasure.[80,96,97] Additionally, dopamine's interaction with N-methyl-D-aspartate receptors is vital for maintaining the balance between excitatory and inhibitory signaling, a key mechanism for mood regulation.[98,99]

Norepinephrine is critical for arousal and alertness and is released in response to stress, significantly influencing emotional responses.[100,101] It interacts with both gamma-aminobutyric acid and glutamate systems in the PFC and amygdala, helping to balance excitatory and inhibitory inputs.[102–104] In the amygdala, norepinephrine promotes the expression of brain-derived neurotrophic factor, which is vital for synaptic plasticity.[105,106] However, chronic stress can lead to excessive norepinephrine release, disrupting GABAergic control and impairing fear extinction, potentially contributing to persistent anxiety symptoms.[107] Elevated norepinephrine levels can reinforce maladaptive emotional responses, making recovery from stress-related disorders more challenging.[82,108]

Integrating traditional medicine with neuroscience: A translational approach

The integration of traditional medicine with modern neuroscience offers a promising translational framework for addressing anxiety and depression. Practices such as herbal medicine, acupuncture, and mind-body therapies (e.g., meditation and yoga) have long focused on restoring balance and well-being.[109,110] These concepts align closely with contemporary neuroscience, which emphasizes neurobiological resilience and homeostasis—key components in maintain-

ing mental health.

Recent research suggests that traditional interventions may influence neuromodulatory systems crucial for mood regulation.[5] For example, herbs like *St. John's Wort*, commonly used for mild depression, have been found to increase serotonin availability, mimicking the effects of SSRIs.[111,112] Additionally, compounds in *St. John's Wort* may interact with dopamine and norepinephrine pathways,[113] providing a multitarget approach to mood regulation that reflects the complexity of neurochemical imbalances seen in mood disorders.

Acupuncture, a cornerstone of traditional Chinese medicine, has shown translational potential in neuropsychiatry.[114,115] Neuroimaging studies indicate that acupuncture can modulate activity in brain regions critical for emotional regulation, particularly the PFC and amygdala.[116,117] This modulation may help restore emotional balance by influencing neurotransmitter release, including endorphins and serotonin, potentially alleviating symptoms of anxiety and depression.

Mind-body therapies, such as yoga and meditation, are increasingly recognized for their positive effects on brain structure and function. Regular practice has been associated with increased gray matter in the PFC and hippocampus—regions essential for emotional control and stress response.[118,119] These therapies enhance neuroplasticity, cognitive flexibility, and stress adaptation, while promoting parasympathetic nervous system activity, which fosters relaxation and overall physiological balance.

From a translational perspective, combining traditional medicine with neuroscience offers both immediate symptom relief and long-term emotional resilience. This approach aligns with the goals of personalized mental health care, where treatments are tailored to individual neurobiological and psychological profiles. For example, some individuals may benefit from SSRIs, while others might respond better to a combination of *St. John's Wort* and mindfulness practices, depending on their unique neurochemical and emotional needs.

The integration of traditional medicine with modern neuroscience provides a comprehensive and individualized approach to treating anxiety and depression. By leveraging the neuromodulatory and neuroplasticity-enhancing properties of these therapies, clinicians can develop personalized treatment strategies that address both immediate symptoms and the underlying neurobiology of mood disorders. This integrative model has the potential to reshape mental health care, combining the wisdom of traditional practices with the precision of scientific research for optimal patient outcomes.

SZ and brain complexity: Neurobiological mechanisms and treatment response

SZ is a complex and debilitating mental disorder characterized by positive symptoms (e.g., delusions, hallucinations), negative symptoms (e.g., emotional blunting, social withdrawal), and cognitive impairments.[120] While antipsychotic medications are commonly used to alleviate SZ symptoms, the neurobiological mechanisms underlying their efficacy remain poorly understood.[121,122] The application of complexity theory offers a novel framework for investigating the neurobiological basis of SZ and its response to treatment. Brain complexity metrics, as proposed by this approach, could provide valuable insights into both the disorder's symptomatology and the mechanisms driving therapeutic outcomes.

Complexity theory and brain dynamics

Complexity theory conceptualizes the brain as a self-organizing, nonlinear system with chaotic dynamics, reflecting the unpredictable thought and behavior patterns often seen in SZ.[123–126] This model suggests that the brain's ability to dynamically reorganize between stable and flexible states enables adaptive responses to environmental demands.[127–128] Resting-state models propose that flexibility in brain dynamics helps maintain optimal brain function with minimal energy expenditure. Additionally, stochastic resonance theory posits that dynamic variability allows subthreshold neurons to reach firing thresholds, enhancing information processing.[129–131]

In SZ and related psychiatric disorders, reduced neural complexity is thought to impair adaptability, contributing to cognitive and social deficits.[14,132] For example, SZ patients often exhibit repetitive behaviors during random task performance and generate more structured rhythmic sequences than healthy individuals, indicating reduced flexibility in information processing and difficulty adapting to unpredictable environments.[133–135]

Brain complexity, cognitive function, and striatal connectivity in SZ

Understanding brain complexity provides a framework for investigating the neural mechanisms underlying cognitive deficits in SZ. Information theory, particularly through entropy analysis, offers a quantitative approach to assessing brain complexity.[136,137] Techniques such as EEG, magnetoencephalography, and fMRI consistently demonstrate reduced brain complexity in individuals with SZ. However, the precise regional variations and temporal stability of these findings remain areas of ongoing investigation.[138–142] Given the inherently dynamic nature of brain activity, complexity measures hold significant promise for elucidating the cognitive impairments associated with SZ. This suggests that SZ patients exhibit diminished dynamic complexity in resting-state brain activity, which correlates with cognitive deficits and may be modifiable through targeted interventions.[19,143–146]

The caudate and putamen, key input nuclei of the basal ganglia, are densely interconnected with extensive cortical projections. In SZ, these regions exhibit reduced complexity, suggesting significant functional disruptions in striatal information processing.[147–150] Studies have reported decreased temporal and sample entropy in the caudate and putamen of SZ patients, indicating potential impacts on cognitive domains such as attention and motor control.[14,151–153] These findings implicate reduced striatal complexity as a contributor to the broader cognitive and motor deficits characteristic of SZ.

Regions with greater neural complexity are often associated with enhanced inter-regional connectivity, which supports efficient communication across the brain. Consistent with this principle, studies have observed that regions with reduced complexity also exhibit weakened connectivity. Specifically, striatal complexity showed a positive correlation with connection strength, corroborating fMRI findings that highlight disrupted connectivity between the striatum and the cortex in SZ.[144,154–157] This impaired connectivity likely contributes to diminished global brain integration, exacerbating cognitive and psychiatric symptoms.

Reduced brain and striatal complexity in SZ reflect a breakdown in both regional processing and network-wide integration, with significant implications for cognition and behavior.[143,158–160] Investigating these disruptions through advanced neuroimaging and complexity metrics offers critical insights into the pathophysiology of SZ and presents opportunities for developing targeted therapeutic strategies.

Cognitive impairments and treatment response in SZ

Cognitive deficits and clinical symptoms in SZ have been closely associated with reduced brain complexity.[14,132,161] Lower left caudate complexity has been linked to poorer performance on cognitive assessments such as the Continuous Performance Test and the Category Fluency Test, which evaluate attention, response control, and processing speed.[162–164] Additionally, structural imaging studies have demonstrated that reduced striatal volume correlates with impairments in attention, problem-solving, and working memory.[157,158,165,166] These findings support the hypothesis that diminished striatal complexity and connectivity significantly contribute to cognitive dysfunction in SZ.

Post-treatment analyses have shown an increase in left caudate complexity, suggesting that antipsychotic medications may enhance spontaneous neural activity in this region, facilitating functional recovery.[163,167,168] Furthermore, higher pre-treatment right caudate complexity has been associated with improved treatment outcomes, indicating that baseline complexity may serve as a predictive biomarker for therapeutic response.[169–171] Longitudinal studies have also reported increased low-frequency blood-oxygen-level-dependent fluctuations in the caudate after treatment, coinciding with symptom improvement.[172–174] These findings emphasize the pivotal role of caudate complexity in the manifestation of cognitive impairments and the effectiveness of treatment in SZ. By demonstrating a relationship between brain complexity and treatment response, this research highlights the potential of neuroimaging-based biomarkers to guide personalized interventions, ultimately improving therapeutic outcomes for individuals with SZ.

Resting-state connectivity and genetic insights in SZ through eigenvector centrality mapping and network dysconnectivity

SZ is a highly heritable psychiatric disorder, with genetic factors accounting for up to 80% of its variance.[175–178] A key neurobiological hallmark of SZ is disrupted brain connectivity, particularly within resting-state functional connectivity networks. These abnormalities, often referred to as dysconnectivity, are central to the disorder's pathology.[179–181] Investigating the application of eigenvector centrality mapping (ECM) to identify connectivity changes specific to SZ, alongside exploring the genetic mechanisms underlying these disruptions, is crucial for understanding the disorder.

Resting-state functional connectivity and dysconnectivity in SZ

Resting-state functional connectivity measures the synchronization of neural activity between different brain regions during periods of rest.[182,183] In SZ, disruptions in these connectivity patterns support the dysconnectivity hypothesis, which posits that abnormal interactions between brain networks contribute to the disorder's pathology.[181,184–186] Understanding these alterations is essential for elucidating SZ's complex neurobiological mechanisms and may provide valuable insights into potential therapeutic targets.

Two primary methodologies are commonly used to analyze resting-state connectivity in SZ: seed-based analysis and independent component analysis (ICA). Seed-based analysis involves selecting a predefined "seed" region and measuring its temporal correlation with other brain regions.[144,179,187,188] While this method offers focused insights into specific network interactions, variability in seed selection across studies complicates comparisons and reduces reproducibility. In contrast, ICA is a data-driven approach that decomposes resting-state fMRI data into independent spatial and temporal components, enabling the identification of intrinsic connectivity networks.[179,189,190] While ICA is valuable for examining within-network connectivity, its emphasis on internal dynamics limits its ability to assess cross-network interactions. This highlights the need for more integrative methods to capture the full spectrum of SZ-related dysconnectivity.

ECM: A comprehensive connectivity measure

ECM is a graph theory-based method that assesses the influence of each brain region within the overall brain network by measuring its connections to other highly connected nodes.[191,192] Unlike seed-based or ICA methods, ECM provides a more integrated perspective on brain connectivity, capturing the complex interrelationships within the network. By evaluating connectivity across the entire brain network, ECM offers a comprehensive understanding of functional dynamics in both typical and atypical brain functions.

Studies using ECM have identified distinctive connectivity alterations in SZ. Regions such as the inferior frontal gyrus and superior temporal gyrus—areas associated with language processing, auditory perception, and executive control—show increased eigenvector centrality, indicating hyperconnectivity. This hyperconnectivity may underlie symptoms such as auditory hallucinations and cognitive rigidity.[188,192] In contrast, reduced eigenvector centrality has been observed in the hippocampus and occipital cortex, areas critical for memory, spatial navigation, and visual processing. This reduced connectivity in SZ may be linked to cognitive and sensory integration deficits, which are commonly observed in patients. These findings underscore ECM's utility in mapping the connectivity disruptions characteristic of SZ.

However, variability in network definitions, sample sizes, and clinical diversity within SZ populations contributes to inconsistencies in ECM findings across studies.[188] These discrepancies highlight the need for replication studies using independent datasets to solidify our understanding of SZ-related connectivity alterations.

Genetic underpinnings of connectivity alterations in SZ

SZ is a polygenic disorder, with numerous genetic variants implicated in its development. Genome-wide association studies have identified several variants associated with SZ, many of which influence neurodevelopment, synaptic transmission, and immune function.[193–195] Given the heritability of resting-state connectivity, it is plausible that these genetic factors contribute to the connectivity alterations observed in

SZ.

Neurodevelopmental genes play a foundational role in shaping neural growth and differentiation, directly influencing the formation and stability of brain networks. Variants in these genes can disrupt the architecture of neural circuits, potentially leading to the dysconnectivity that characterizes SZ.[196,197] Similarly, genetic variants affecting neurotransmitter systems, particularly dopamine and glutamate, are critical for synaptic plasticity and connectivity.[198,199] Disruptions in these systems can impair neuronal communication, contributing to the functional and structural connectivity disturbances observed in SZ.

In addition to neurodevelopmental and neurotransmitter-related genetic factors, immune-related genetic variants also appear to play a significant role. These variants are linked to neuroinflammatory processes, which may further exacerbate connectivity deficits in SZ.[200–202] Together, these genetic influences converge on multiple pathways, disrupting brain network function and contributing to the pathophysiology of SZ.

Integrating extracellular matrix data with genetic and transcriptomic insights in SZ

Integrating extracellular matrix data with genetic and transcriptomic information provides a robust framework for exploring the underlying mechanisms of SZ.[188,203–206] This approach enables researchers to investigate how genetic variations influence brain connectivity and to identify pathways through which genetic risk factors contribute to network dysfunction. Such integrative analyses hold significant potential for uncovering novel biomarkers and therapeutic targets, ultimately enhancing the precision of SZ diagnosis and treatment strategies.

Despite its promise, studying brain connectivity in SZ presents substantial challenges. Variability in sample populations, imaging protocols, and data preprocessing methods often introduces inconsistencies, limiting the reproducibility and generalizability of findings.[139,185,207,208] Clinical heterogeneity further complicates efforts to identify consistent connectivity patterns, as symptom profiles and severities vary widely among patients.[14,194,209] Moreover, the predominance of cross-sectional study designs hampers the ability to track longitudinal changes in brain connectivity and evaluate dynamic alterations over time.[139,210]

To address these challenges, future research must prioritize the standardization of analytical methodologies, including those applied to integrating extracellular matrix data, to enhance comparability and replicability across studies. Large-scale collaborations and consortia should be encouraged to consolidate resources and data, fostering more comprehensive investigations. By advancing methodological rigor and promoting collaborative efforts, the field can progress toward a deeper understanding of SZ pathophysiology and the development of more effective interventions.

Neuroimaging and neurostimulation techniques: Translational medicine approaches for treating anxiety, depression, and SZ

Neuroimaging and neurostimulation technologies are revolutionizing neuroscience by providing unprecedented insights into brain function and enabling innovative therapeutic strategies for psychiatric disorders.[34,211] These advancements hold particular promise for conditions such as anxiety, depression, and SZ, which are characterized by dysfunctions in neural circuits. Despite significant progress in understanding these disorders, traditional pharmacological treatments often fall short,[212] leaving many patients with unmet clinical needs. The development of neuroimaging and neurostimulation techniques offers new avenues for more personalized and effective interventions (Table 1).[213–265] Therefore, exploring the therapeutic potential of these technologies is urgently needed. This exploration underscores the evolving role of translational medicine in bridging basic scientific discoveries with clinical practice.

Neuroimaging techniques in anxiety, depression, and SZ

Neuroimaging is an essential tool for investigating structural and functional brain abnormalities associated with psychiatric disorders. Techniques such as magnetic resonance imaging (MRI), EEG, and PET enable detailed mapping of brain anatomy and function, providing critical insights into the pathophysiology of conditions like anxiety, depression, and SZ (Table 1).[34,266] By identifying disruptions in neural circuits, these methods are vital for advancing the development of effective therapeutic strategies.

In SZ, neuroimaging studies, particularly those employing MRI, consistently highlight structural anomalies in regions such as the PFC, hippocampus, and temporal lobes—areas integral to cognitive and emotional regulation.[267–269] Functional PET imaging has been pivotal in examining dopamine receptor dysfunctions, a core aspect of SZ's pathophysiology.[148,270] Combined PET and MRI studies not only aid in diagnosing the disorder but also help identify biomarkers that guide personalized treatment approaches, particularly for patients unresponsive to standard medications.[157,271–273]

For anxiety and depression, fMRI has revealed altered connectivity between the PFC and amygdala—two regions critical for emotional regulation and fear processing.[274–277] This disrupted connectivity underscores the role of neural circuit dysregulation in these conditions.[278,279] Resting-state fMRI has further revealed how these abnormalities contribute to the chronic and recurrent nature of anxiety and depression.[280,281] Moreover, neuroimaging holds promise for identifying early biomarkers of these disorders, enabling timely intervention and the development of targeted therapies.[282–284]

The integration of machine learning with neuroimaging has significantly enhanced its diagnostic and prognostic capabilities. For example, machine learning algorithms have been applied to EEG data to predict symptom severity and classify subtypes of psychiatric disorders, including anxiety, depression, and SZ. These algorithms can identify subtle patterns in brain activity that might evade traditional analysis, paving the way for earlier and more accurate diagnoses.[285–287] Furthermore, machine learning can facilitate the identification of individualized neural biomarkers, a critical step in tailoring treatment strategies to meet the unique needs of each patient.[28,283,288]

Neurostimulation techniques: Emerging non-pharmacological interventions for psychiatric disorders

Neurostimulation techniques have emerged as promising

Table 1. Summary of state-of-the-art neuroimaging and neurostimulation methods used in the investigation and treatment of anxiety, depression, and schizophrenia

Category	Technique	Application	Key findings/insights	Translational potential	References
Neuroimaging	Magnetic Resonance Imaging (MRI)	Mapping structural abnormalities in psychiatric disorders.	Schizophrenia: Prefrontal cortex, hippocampus, and temporal lobe anomalies.	Biomarker identification for personalized treatment.	213–217
	Functional MRI (fMRI)	Investigating altered connectivity in emotional regulation circuits.	Anxiety/Depression: Disrupted connectivity between the prefrontal cortex and amygdala.	Early biomarker identification for timely interventions.	218–222
	Resting-State fMRI (rsfMRI)	Studying intrinsic brain network activity in psychiatric disorders.	Anxiety/Depression: Circuit abnormalities linked to chronic, recurrent symptoms.	Development of targeted therapies.	223–228
	Positron Emission Tomography (PET)	Examining neurotransmitter receptor dysfunctions.	Schizophrenia: Dopamine receptor abnormalities contributing to pathophysiology.	Diagnosis and treatment personalization based on receptor-specific activity.	229–232
	Electroencephalography (EEG)	Analyzing brain activity patterns in psychiatric disorders.	Machine learning studies reveal subtle patterns in EEG data predicting symptom severity.	Predictive diagnostics and personalized treatment guidance.	233–237
Neurostimulation	Low-Intensity Ultrasound	Non-invasive modulation of cortical excitability.	Depression/Anxiety: Enhances prefrontal cortex and amygdala activity without tissue damage.	Potential non-invasive alternative for pharmacological therapies.	238–244
	Transcranial Electrical Stimulation (TES)	Enhancing neuronal activity through techniques like tDCS and tACS.	Depression: Increased prefrontal cortex activity. Anxiety: Modulation of amygdala dysfunction.	Personalized TES protocols for diverse psychiatric conditions.	245–249
	Deep Brain Stimulation (DBS)	Continuous electrical impulses to regulate dysfunctional circuits.	Depression: Targets nucleus accumbens for treatment-resistant symptoms. Schizophrenia: Modulates dopamine dysregulation in psychotic symptoms.	Integration with neuroimaging for precise targeting and enhanced outcomes.	250–254
Machine Learning Integration	EEG, MRI, PET, fMRI Analysis	Advanced analysis of neuroimaging data to identify subtle activity patterns.	Enables classification of subtypes of psychiatric disorders and prediction of treatment outcomes.	Improved diagnostic accuracy and personalized therapeutic strategies.	255–261
Translational Applications	Neuroimaging-guided Neurostimulation	Combining imaging data with neurostimulation protocols for precision targeting.	Real-time mapping of brain activity improves stimulation accuracy, minimizing side effects.	Facilitates effective, personalized care, especially for treatment-resistant cases.	246,262–265

non-pharmacological interventions for psychiatric disorders, offering innovative ways to modulate brain activity in regions critical for mood regulation, cognitive function, and emotional processing.[289–293] These techniques—including low-intensity ultrasound, TES, and DBS—provide viable alternatives for patients who do not respond to conventional drug therapies.[29,30,294]

Low-intensity ultrasound is a non-invasive neurostimulation technique that modulates brain activity without causing tissue damage.[295–297] Research in animal models demonstrates its potential to enhance cortical excitability, positioning it as a possible treatment for conditions like depression and anxiety.[298–300] The non-invasive nature of focused ultrasound makes it an attractive alternative to more invasive

interventions, such as DBS, especially for patients seeking less intrusive treatments.[301,302]

TES encompasses techniques such as transcranial direct current stimulation and transcranial alternating current stimulation, both gaining recognition for their ability to influence cortical excitability.[303,304] In depression, TES has been shown to enhance neuronal activity in the PFC, which typically exhibits reduced activity.[305–307] In anxiety, TES targets the amygdala to address dysfunctions in fear processing.[308,309] Innovations such as alternating current square wave stimulation allow for precise cortical modulation, enabling highly personalized treatments.[211,310] The adaptability of TES to individual neural circuits underscores its transformative potential for managing a range of psychiatric conditions.

Originally developed for movement disorders like Parkinson's disease, DBS is now being explored for psychiatric conditions, including depression and SZ.[311,312] DBS delivers continuous electrical impulses to specific brain regions, such as the subthalamic nucleus, ventral capsule, or nucleus accumbens, to regulate neural circuits involved in mood regulation and psychotic symptoms.[313–315] In treatment-resistant depression, DBS has shown promising results, while in SZ, it targets areas involved in dopamine dysregulation to alleviate psychotic symptoms.[312,316–318] The integration of neuroimaging data into DBS protocols allows for the customization of stimulation parameters based on a patient's unique brain activity, enhancing therapeutic precision and outcomes.

Neurostimulation techniques are advancing rapidly as effective non-pharmacological treatments for psychiatric disorders. Innovations such as low-intensity ultrasound, TES, and DBS hold substantial potential for delivering personalized and targeted care, particularly for patients with complex, treatment-resistant conditions. By leveraging the synergy of neuroimaging and cutting-edge technologies, these techniques are poised to transform psychiatric care, offering hope for improved outcomes and a better quality of life for patients.

Translational medicine in psychiatry: Integrating neuroimaging, neurostimulation, and machine learning for precision treatment

Translational medicine aims to bridge the gap between basic scientific research and its clinical applications, with the ultimate goal of developing more effective, personalized treatments for a range of disorders. In the context of psychiatric conditions such as anxiety, depression, and SZ, the integration of advanced neuroimaging and neurostimulation techniques into clinical practice represents a significant leap forward in precision medicine.[284,319,320] By applying insights from basic neuroscience, translational approaches seek to create treatments tailored to individual patients, improving outcomes and reducing the trial-and-error process that often characterizes psychiatric care.

A key component of this translational process is the use of machine learning to enhance both diagnosis and treatment personalization. Machine learning algorithms are capable of analyzing large datasets derived from neuroimaging and neurostimulation, identifying distinct patterns of brain activity that correlate with specific subtypes of psychiatric disorders.[28,321] This data-driven approach enables the identification of biomarkers that predict how a patient will respond to particular treatments. For example, EEG biomarkers associated with specific subtypes of depression can help clinicians determine whether a patient is more likely to benefit from TES or DBS, streamlining treatment decisions and reducing the need for prolonged trial and error.[322–325]

One of the most promising examples of translational medicine in psychiatry is the use of neuroimaging to guide neurostimulation protocols. fMRI and other imaging modalities provide real-time insights into brain activity, allowing clinicians to personalize neurostimulation treatments with greater accuracy.[34,326–328] By mapping the brain regions involved in conditions like anxiety, depression, and SZ, neuroimaging enables precise targeting of neurostimulation therapies to the neural circuits implicated in these disorders.[34,329,330] This approach not only improves the likelihood of therapeutic success but also minimizes the risk of side effects, offering a more efficient and tailored treatment strategy.

fMRI has become a critical tool in the study of task-related brain activity, offering valuable insights into how psychiatric conditions affect the processing of specific stimuli.[331] fMRI allows for the identification of distinct brain activation patterns in regions important for cognitive and emotional processing, such as the precuneus, posterior cingulate gyrus, superior parietal lobule, and angular gyrus—all of which are associated with the default mode network (DMN), autobiographical memory, and self-referential thinking.[332] Disruptions in DMN functioning are frequently implicated in psychiatric disorders, contributing to cognitive and emotional dysregulation. To complement fMRI findings, dynamic causal modeling helps assess effective connectivity, revealing how brain regions interact. For example, altered connectivity, such as inhibitory connections between the dorsolateral PFC and the anterior insula, suggests disruptions in the interaction between the salience network and DMN, which may underlie cognitive and behavioral abnormalities. High-resolution resting-state fMRI further expands this analysis by examining functional connectivity between brain networks, providing evidence of heightened activity in areas like the precuneus and posterior cingulate cortex, suggesting DMN disruption.[332] Changes in regions such as the angular gyrus, involved in attention and cognitive processing, emphasize the importance of cognitive network disturbances in psychiatric conditions. These findings highlight the need to focus on dysfunctional brain network interactions rather than isolated abnormalities in specific regions. Neurostimulation techniques, such as repetitive TMS and transcranial direct current stimulation, which modulate brain network plasticity, show promise for restoring connectivity within these networks, offering potential clinical interventions for precision treatment in psychiatry.

The integration of neuroimaging, neurostimulation, and machine learning techniques is essential for advancing precision medicine in psychiatry. ICA is one such method used in this context. ICA identifies distinct brain activation components related to specific tasks and psychiatric conditions. By decomposing complex neuroimaging data into independent components, ICA enables the detection of subtle differences in brain activity patterns across psychiatric populations.[333] This approach facilitates the examination of large-scale brain networks, such as the salience network and DMN, and their alterations in conditions like SZ and depression. Additionally, machine learning techniques can be applied to further analyze these components, providing a more comprehensive understanding of individual variability and network-level

disruptions in psychiatric disorders. Combining ICA with machine learning enhances the sensitivity and precision of neuroimaging analyses, allowing for more accurate identification of biomarkers and guiding the development of personalized therapeutic interventions, including targeted neurostimulation strategies aimed at modulating specific brain circuits involved in cognitive, emotional, and motor processing.

The integration of neuroimaging, neurostimulation, and machine learning in psychiatry highlights the transformative potential of translational medicine in clinical practice. These advancements not only enhance our understanding of the brain but also facilitate the development of personalized treatments, offering hope for patients with complex, treatment-resistant conditions. By bridging basic neuroscience discoveries with clinical applications, translational medicine plays a critical role in improving psychiatric care. Combining neuroimaging data with neurostimulation protocols enables the precise targeting of dysfunctional brain regions, minimizing side effects and optimizing therapeutic outcomes. This personalized approach addresses the unique neural circuit disruptions present in each patient's condition. Together, neuroimaging and neurostimulation represent a paradigm shift in psychiatric treatment, providing novel, individualized therapeutic options. As the field progresses, the synergy between these technologies and translational medicine will be essential in meeting the unmet needs of patients with anxiety, depression, and SZ.

Limitations

While this article provides valuable insights into the neurobiological mechanisms underlying anxiety, depression, and SZ, several limitations must be considered. Most of the research in this field relies on cross-sectional data, which restricts our understanding of the long-term progression of these disorders and the sustained effects of treatments. Longitudinal studies are needed to examine how psychiatric conditions evolve over time and how interventions impact brain function in the long term. Additionally, variability in study protocols, clinical heterogeneity, and differences in patient populations complicate the generalization of findings and the development of standardized treatment protocols. Although neuroimaging and computational models show promise, their clinical application is still in its early stages and requires further validation before they can be integrated into therapeutic interventions. While precision psychiatry holds great potential, progress is impeded by the lack of reliable biomarkers to predict treatment responses and disease progression. Moreover, the use of traditional healing practices, such as acupuncture and herbal medicine, although increasingly supported by evidence, faces challenges in standardization and broader acceptance within mainstream medical practices. Personalized neurostimulation techniques, such as TMS and DBS, also require further optimization to accurately target specific brain regions and neural circuits. Rigorous clinical trials are necessary to ensure their safety and efficacy. Furthermore, current research often overlooks the potential protective effects of psychotherapy and counseling interventions, which are frequently used without prescriptions to prevent or alleviate symptoms at the onset of psychiatric conditions. The efficacy and mechanisms of these non-prescription, accessible therapies remain underexplored. Their role in early intervention and prevention strategies deserves further investigation to better understand their potential in reducing the severity or delaying the onset of mental health symptoms.

Discussion

Anxiety and depression are complex disorders characterized by disruptions in neural circuits and neurotransmitter systems, leading to impaired emotional regulation and cognitive functioning. These conditions are influenced by a combination of genetic, environmental, and neurobiological factors, with serotonin, dopamine, and norepinephrine playing central roles in regulating mood, stress responses, and cognitive flexibility.[10,334,335] The involvement of these neurotransmitters underscores the importance of personalized, targeted treatment strategies.

To address these challenges, researchers are increasingly adopting an integrative approach that combines computational models with insights from traditional medicine. This interdisciplinary framework enhances our understanding of anxiety and depression by simulating neural pathways and neurotransmitter interactions, while also exploring the therapeutic potential of traditional practices such as herbal medicine, acupuncture, and mind-body therapies.[37,336–338] These practices provide valuable insights into modulating neurotransmitter systems and promoting neurobiological resilience.

The roles of serotonin, dopamine, and norepinephrine in emotional regulation and neuroplasticity emphasize the need for personalized treatments. Variations in these systems contribute to the onset and progression of anxiety and depression, making tailored interventions essential.[11,339] For example, while SSRIs may be effective for some individuals, others may benefit from a combination of pharmacological and non-pharmacological therapies, such as acupuncture or mindfulness, that target multiple neurochemical systems.[89,340,341]

This integrative approach holds great promise for developing future therapies that move beyond the "one-size-fits-all" model. By combining cutting-edge neuroscience with the wisdom of traditional medicine, researchers are opening new pathways for treatment. This strategy not only aims to provide immediate symptom relief but also promotes long-term resilience, offering hope for individuals affected by anxiety and depression.[342,343]

Research has also highlighted the importance of reduced brain complexity in SZ and its relationship to cognitive deficits and clinical symptoms.[344,345] The striatum, particularly the caudate, plays a critical role in the complexity disruptions observed in SZ, likely influenced by dopamine dysregulation. This dysregulation may impair connectivity between the striatum and PFC, contributing to deficits in cognitive integration.[147,148,158,346,347] Antipsychotic treatments targeting D2 receptors may restore caudate functionality, potentially improving both cognitive and clinical outcomes. These findings suggest that dynamic brain complexity could serve as a promising biomarker for understanding SZ pathophysiology and assessing treatment efficacy.[33,348]

SZ is a multifaceted psychiatric disorder, marked by pronounced disruptions in brain connectivity, particularly within resting-state functional networks.[181,349,350] This dysconnec-

tivity is a central contributor to the neurobiological dysfunction associated with the disorder.[139] Notably, hyperconnectivity is observed in regions such as the inferior frontal gyrus and superior temporal gyrus, while reduced connectivity is found in areas such as the hippocampus and occipital cortex.[351–353] ECM offers a robust method for analyzing these connectivity alterations, providing deeper insights into network dynamics compared to traditional methods. Furthermore, genetic factors, including polygenic contributions to neurodevelopment, neurotransmitter systems, and immune function, significantly influence these disruptions. However, challenges such as clinical heterogeneity, variability in study protocols, and the lack of longitudinal data continue to hinder progress in this field.

Neuroimaging and neurostimulation techniques are driving a paradigm shift in our understanding of psychiatric disorders, including anxiety, depression, and SZ.[34,354,355] These advanced tools provide critical insights into the brain's intricate neural circuits and offer innovative therapeutic solutions for patients who do not respond to traditional pharmacological treatments. The integration of machine learning with personalized neurostimulation, guided by neuroimaging data, is revolutionizing psychiatric care by enabling more precise and targeted interventions. As these technologies continue to advance, their application in translational medicine holds the potential to reshape the therapeutic landscape, offering renewed hope for patients and clinicians alike.

Conclusions

The future of psychiatric care is on the verge of a significant transformation, driven by advancements in neuroimaging, neurostimulation, and computational modeling. As research uncovers more about the complexities of the brain, the potential for clinical applications that offer more precise and individualized treatment strategies continues to grow. Several key areas hold great promises for both clinical practice and future research directions.

One of the most exciting developments in psychiatric treatment is the emergence of precision psychiatry. This approach marks a shift from the traditional "one-size-fits-all" model to a more personalized form of care, based on individual neurobiological profiles. By using neuroimaging techniques to assess brain connectivity and computational models to predict treatment responses, clinicians can tailor interventions more effectively. This precision approach extends beyond pharmacological treatments, incorporating non-pharmacological therapies such as neurostimulation, mindfulness, and cognitive therapies. By targeting specific neurobiological dysfunctions contributing to a patient's condition, precision psychiatry aims to optimize treatment efficacy and improve long-term outcomes, providing a more comprehensive and individualized approach to care.

Another critical focus for future research is the identification of biomarkers that can predict both treatment response and disease progression. Biomarkers serve as key indicators of disease state, improving diagnostic accuracy and helping clinicians make more informed decisions about treatment strategies. Advances in neuroimaging technologies, coupled with machine learning algorithms capable of analyzing large datasets, are paving the way for the discovery of biomarkers associated with psychiatric disorders such as anxiety, depression, and SZ. Identifying these biomarkers will not only refine diagnostic processes but also facilitate the development of targeted treatments that are more effective and personalized. The ability to predict how a patient will respond to a particular intervention will reduce reliance on trial-and-error treatment approaches, significantly improving overall clinical outcomes.

Longitudinal studies are crucial for advancing our understanding of psychiatric disorders and their long-term progression. By tracking changes in brain function and connectivity over time, these studies provide valuable insights into how psychiatric conditions evolve and how treatments affect the brain. When combined with data from neuroimaging, neurostimulation, and computational models, longitudinal research will offer a more comprehensive understanding of the chronic nature of these disorders and their responses to various interventions. This data will be key in developing sustainable treatment strategies that not only alleviate symptoms but also promote long-term neurobiological health and resilience.

Personalized neurostimulation techniques, such as TMS and DBS, show significant promise in treating refractory psychiatric disorders. These methods, which stimulate specific brain regions to modulate neural activity, are increasingly explored as adjuncts to traditional pharmacotherapy. Future research should focus on optimizing these neurostimulation techniques by precisely targeting disrupted brain regions and neural circuits involved in conditions like depression and SZ. By fine-tuning stimulation parameters and combining neurostimulation with pharmacological treatments and other therapeutic interventions, researchers could achieve synergistic effects, improving both symptom management and cognitive function. This approach holds potential for providing more effective treatments, especially for patients who have not responded well to conventional therapies.

An exciting area of future research involves integrating traditional healing practices with modern psychiatric treatments. Therapies such as acupuncture, herbal medicine, and other mind-body interventions have been used for centuries to enhance mental well-being and modulate neurotransmitter systems. Growing evidence suggests that these traditional approaches may boost neuroplasticity, complementing the effects of pharmacological treatments. By combining these time-tested therapies with advanced neuroimaging techniques and computational models, researchers could create a more holistic approach to psychiatric care. This integrative model would offer patients a broader range of therapeutic options and could lead to more effective, individualized treatments that address the underlying causes of psychiatric disorders from multiple perspectives.

The transformative potential of personalized medicine and neurotechnological advancements in psychiatric treatment is immense. Integrating neuroimaging and computational models into clinical practice is particularly promising, as it enables clinicians to identify biomarkers and mechanisms unique to each patient's condition. This precision approach, when combined with traditional healing practices, provides a deeper understanding of psychiatric disorders and facilitates more effective and tailored treatments. As research continues to explore the roles of brain circuits, neurotransmitter systems, and neuroplasticity, we can expect significant improvements in treatment outcomes, ultimately leading to a

better quality of life for patients. The future of psychiatry lies in a holistic, personalized approach that integrates cutting-edge technologies with a comprehensive understanding of both neurobiological and environmental factors influencing mental health. This paradigm shift has the potential to revolutionize psychiatric care and provide more sustainable solutions for patients suffering from complex disorders such as anxiety, depression, and SZ.

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Conflict of interest

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Author contributions

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