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Neuromodulation and neural networks in psychiatric disorders: current status and emerging prospects

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Review Article

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Abstract

Psychiatric disorders lead to disability, premature mortality and economic burden, highlighting the urgent need for more effective treatments. The understanding of psychiatric disorders as conditions of large-scale brain networks has created new opportunities for developing targeted, personalised, and mechanism-based therapeutic interventions. Non-invasive brain stimulation (NIBS) techniques, such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS), can directly modulate dysfunctional neural networks, enabling treatments tailored to the individual's unique functional network patterns.

As NIBS techniques depend on our understanding of the neural networks involved in psychiatric disorders, this review offers a neural network-informed perspective on their applications. We focus on key disorders, including depression, schizophrenia, and obsessive-compulsive disorder, and examine the role of NIBS on cognitive impairment, a transdiagnostic feature that does not respond to conventional treatments. We discuss the advancements in identifying NIBS response biomarkers with the use of electrophysiology and neuroimaging, which can inform the development of optimised, mechanism-based, personalised NIBS treatment protocols.

We address key challenges, including the need for more precise, individualised targeting of dysfunctional networks through integration of neurophysiological, neuroimaging and genetic data and the use of emerging techniques, such as low- intensity focused ultrasound, which has the potential to improve spatial precision and target access. We finally explore future directions to improve treatment protocols and promote widespread clinical use of NIBS as a safe, effective and patient-centred treatment for psychiatric disorders.

Introduction

Psychiatric disorders are a leading cause of global disability, accounting for 32.4% of years lived with disability and 13% of disability-adjusted life years, comparable to cardiovascular and circulatory diseases (Vigo, Thornicroft, & Atun, 2016). Medications and psychotherapies offer modest symptom improvements but do not significantly alleviate disability or improve functional outcomes (Leichsenring, Steinert, Rabung, & Ioannidis, 2022). Additionally, 20%–60% of individuals fail to respond adequately to optimal treatments (Howes, Thase, & Pillinger, 2022), and medications often have side effects that limit adherence and acceptability. The economic burden is substantial, with mental disorders costing £300 billion in England in 2022 through premature mortality, direct losses to the economy through unemployment, and indirect losses related to health and care costs (Cardoso & McHayle, 2024). These highlight the urgent need for novel, mechanism-based, safe, effective, and acceptable treatments as alternatives or additions to existing treatments to enhance functional outcomes and quality of life of people with mental disorders.

Psychiatric disorders are increasingly understood as conditions of large-scale brain networks rather than abnormalities within isolated brain regions. These large-scale brain networks are neural systems distributed across most of the brain, anatomically interconnected and functionally synchronized, and support the necessary cognitive, emotional, and sensorimotor processes underpinning complex human behaviors. Dysfunctional information processing within and between these networks is thought to contribute to the pathophysiology of psychiatric disorders and the manifestation of their symptoms (Menon, 2011; Sporns, 2014).

In this context, modulation of specific brain networks through externally applied electromagnetic stimulation (collectively known as 'neuromodulation' or 'neurostimulation') is used

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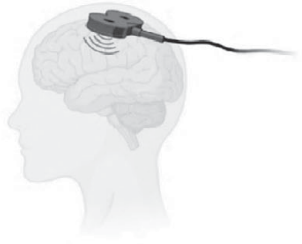
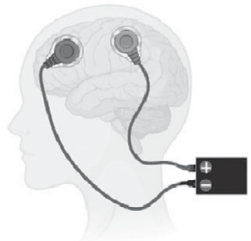
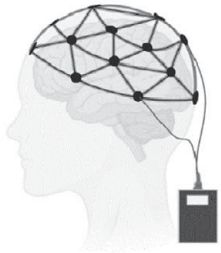
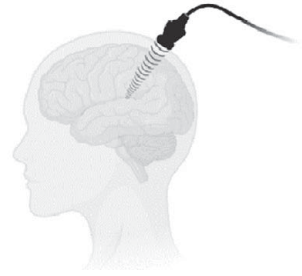
Non-Invasive Brain StimulationTechniques	Application/Action
Transcranial Magnetic Stimulation (TMS) 	• Generates brief magnetic pulses (up to 3T) using a figure-8 coil, inducing electric currents that cause de/hyperpolarisation and modulation of cortical excitability (Chail et al., 2018). • Stimulates at 2–4 cm below the skull surface, while deep TMS reaches up to 6 cm (Chail et al., 2018). • Low-frequency (<1 Hz) reduces cortical excitability, while high-frequency (>5 Hz) enhances it (Cheng et al., 2023). • Variants include repetitive TMS (rTMS), deep TMS, intermittent Theta-Burst Stimulation (iTBS) and continuous Theta-Burst Stimulation (cTBS).
Transcranial Direct Current Stimulation (tDCS) 	• Applies a weak, constant current (1-2 mA) between two electrodes (positive 'anode' and negative 'cathode') with current flowing from anode to cathode (Kesikburun, 2022). • Anodal stimulation increases cortical excitability by depolarising neuronal membranes, while cathodal stimulation decreases it through hyperpolarisation (Kesikburun, 2022).
Transcranial Alternating Current Stimulation (tACS) 	• Applies sinusoidal or rectangular alternating electric current to entrain/synchronise intrinsic brain oscillations to the frequency of the applied current (Herrmann et al., 2013). • Selectively modulates brain functions by targeting frequency-specific neural activity (Herrmann et al., 2013).
Low-Intensity Focused Ultrasound (FUS) 	• Applies low intensity sound waves (≤ diagnostic ultrasound). • Modulates membrane potential by altering ion channel permeability (Cox et al., 2025). • Enables deep and more focal brain stimulation with high spatial accuracy. • Can transiently open the blood-brain barrier for targeted drug delivery when used with specific protocols (Cox et al., 2025). • High-intensity FUS, an invasive method, may ablate tissues by creating thermal lesions.

Figure 1. Non-invasive brain stimulation techniques and their mode of application and action.

to directly modify 'abnormal' neural network activity in psychiatric disorders. While medications and psychotherapies indirectly modulate neural activity (Celada, Puig, & Artigas, 2013; Schrammen et al., 2022), 'neuromodulation' offers the opportunity for a more selective, targeted network-based approach, which has not been feasible so far with medications and psychotherapies. Furthermore, unlike traditional treatments, 'neuromodulation' enables personalized interventions based on an individual's unique brain network dysfunction. 'Neuromodulation' includes invasive and non-invasive brain stimulation (NIBS) techniques, with the latter extensively used for research and treatment in psychiatric disorders, particularly in treatment-resistant disorders (e.g. depression) and difficult-to-treat specific symptoms (e.g. negative symptoms and cognitive impairment) (Figure 1).

Both invasive and NIBS techniques depend on our understanding of brain networks involved in psychiatric disorders. Functional neuroimaging techniques, particularly resting state functional MRI (rsfMRI) functional connectivity (FC) analyses, are the most widely used techniques for the identification of large-scale network abnormalities in psychiatric disorders. FC measures temporal correlations in activity between spatially distant brain regions, revealing spontaneous activity patterns without task-related interference, and has led to the identification of core brain networks, which are hypothesized to play key roles in psychiatric disorders (Yeo et al., 2011) (Table 1).

Table 1. Major brain networks involved in psychiatric disorders: Core nodes and key functions

Major brain networks	Major nodes	Key functions
Default Mode Network (DMN) (Menon, 2023; Smith et al., 2009 ; Yeo et al., 2011)	• PCC • mPFC • Precuneus • Angular Gyrus	• More active at rest than active task engagement • Introspection, internal narrative, self-reflection • Mind wandering and spontaneous cognition • Autobiographical memory retrieval and prospective memory • Theory of mind and mentalizing
Central Executive Network (CEN) / Frontoparietal Network (FPN) (Smith et al., 2009; Yeo et al., 2011)	• Bilateral DLPFC • Posterior Parietal Cortex • Intraparietal Sulcus	• Executive functions, including working memory, cognitive control, decision-making, problem-solving, and cognitive resource allocation to goal-directed behaviors • 'Anticorrelated' with the DMN and works in concert with the SN
Salience Network (SN) (Schimmelpfennig, Topczewski, Zajkowski, & Jankowiak-Siuda, 2023; Uddin, 2015)	• ACC • Anterior Insula • vmPFC • Amygdala • Substantia Nigra	• Salient stimuli identification (bottom-up) and arousal (top-down) allocation • Switching between DMN & CEN • Emotional processing • Regulation of autonomic responses
Affective Network (AN) (Zeng et al., 2012)	• OFC • sgACC • Limbic structures (amygdala, hippocampus, insula)	• Emotion generation and assigning emotional valence to stimuli • Emotional processing and regulation
Reward Network (RN) (Haber, 2009)	• Frontal cortex • ACC • Dorsal striatum (caudate and putamen) • Ventral striatum (NAc) • VTA • Limbic structures (amygdala, hippocampus)	• Ventral striatum • Reward-related pleasure generation • Motivation and incentive salience • Reinforcement learning • Fronto-basal RN • Reward value and reward-related risks evaluation • Reward prediction and anticipation • Decision-making on reward/risk evaluation, action planning
Dorsal Attention Network (DAN) (Corbetta & Shulman, 2002)	• FEF • Intraparietal Sulcus • Superior Parietal Lobule • MT+	• Voluntary, top-down attention • Goal-directed voluntary control of visuospatial attention
Ventral Attention Network (VAN) (Corbetta & Shulman, 2002)	• TPJ • Inferior Frontal Gyrus • Ventral Frontal Cortex • Middle Frontal Gyrus	• Involuntary, bottom-up attention • Unattended/unexpected stimuli detection and attention shifting • VAN and DAN interaction switches attention between top-down goals and bottom-up stimuli
Limbic Network (Yeo et al., 2011)	• Amygdala • Hippocampus • OFC • Cingulate Cortex • Hypothalamus • NAc	• Emotional processing • Motivation • Memory encoding and retrieval • Visceromotor regulation
Sensorimotor Network (SMN) (Uddin, Yeo, & Spreng, 2019; Xiong, Tian, Zeng, Huang, & Wang, 2021)	• M1 • S1 • Premotor Cortex • SMA • Middle cingulate cortex • Basal Ganglia – Thalamus • Cerebellum	• Processing sensory inputs and motor outputs • Motor coordination • Proprioception • Sensory Integration
Visual Network (VN) (Xiong et al., 2021)	• V1 • LGN • Visual Association Areas (V2, V3, V4, V5/MT) • Occipital Lobe	• Visual perception, object recognition, spatial awareness
Auditory network (Kuiper et al., 2020)	• A1 in Heschl's Gyrus • A2 • STG • MGN • Insula • IFG	• Sound localization and discrimination, language processing

Abbreviations: PCC: Posterior Cingulate Cortex; mPFC: medial Prefrontal Cortex;; DLPFC: Dorsolateral Prefrontal Cortex; dAIC: dorsal Anterior Insular Cortex; ACC: Anterior Cingulate Cortex; vmPFC: Ventromedial Prefrontal Cortex; OFC: Orbitofrontal Cortex; sgACC: subgenual Anterior Cingulate Cortex; VTA: Ventral Tegmental Area; NAc: Nucleus accumbens; FEF: Frontal Eye Fields; MT+: Middle Temporal Motion Complex; TPJ: Temporoparietal Junction; M1: Primary Motor Cortex; S1: Primary Somatosensory Cortex; SMA: Supplementary Motor Area; V1: Primary Visual Cortex; LGN: Lateral Geniculate Nucleus (thalamus); A1: Primary Auditory Cortex; A2: Auditory Association Cortex; STG: Superior Temporal Gyrus; MGN: Medial Geniculate Body (thalamus); IFG: Inferior Frontal Gyrus.

Findings from large-scale neuroimaging databases have challenged the classical views that specific disorders map onto distinct brain regions or even networks and suggest overlap of neural networks in psychiatric disorders, along with more disorder-specific effects. For example, the 'triple network model for psychopathology' proposes that DMN, Frontoparietal Network (FPN)/Central Executive Network (CEN), and Salience Network (SN) are implicated in multiple psychiatric disorders (Menon, 2011). SN (Downar, Blumberger, & Daskalakis, 2016; Segal et al., 2023) and the Limbic Network (LIN) (Ishida et al., 2023) have also been proposed as 'common core' neural networks for psychiatric disorders. The involvement of common neural networks across psychiatric disorders has been suggested as 'transdiagnostic' biomarkers, while the involvement of additional neural networks in each disorder may contribute to the phenotypic differences among psychiatric disorders (Chavez-Baldini et al., 2023; Segal et al., 2023). This is particularly significant for NIBS, as it implies that single brain targets for each disorder may be insufficient and points to the integration of psychiatric diagnosis, individual symptom profiles, and brain networks to improve our understanding of the pathophysiology of disorders and develop tailored NIBS interventions.

In this review, we offer a neural network-informed perspective on NIBS applications. We focus on key disorders, including depression, schizophrenia, and obsessive-compulsive disorder, and examine the impact of NIBS on cognitive impairment, a transdiagnostic feature that does not respond to conventional treatments. We discuss the advancements in identifying NIBS response biomarkers with the use of electrophysiology and neuroimaging, and conclude with key challenges and future directions to improve treatment protocols and promote widespread clinical use of NIBS as a safe, effective, and patient-centred treatment for psychiatric disorders.

Major depressive disorder

Major depressive disorder (MDD) is a highly heterogeneous syndrome affecting 320 million people globally and is a leading cause of disability (World Health Organization, 2017). Around 30% of patients develop treatment-resistant depression (TRD), defined as non-response to two adequate antidepressant trials (McIntyre et al., 2023). TRD was the first psychiatric disorder for which a NIBS treatment was approved, with rTMS over the left dorsolateral prefrontal cortex (lDLPFC) receiving FDA clearance in 2008. The UK's NICE guidelines also recommend rTMS for TRD (National Institute for Health and Care Excellence, 2015).

Depression is currently conceptualized as a systems-level disorder caused by disrupted network regulation under cognitive, emotional, or physical stress (Mayberg, 2003). The key brain networks associated with MDD include:

- Affective Network (AN) with increased FC between AN regions, which is associated with excessive negative feelings (Li et al., 2018), while the onset of depression is associated with increased FC between amygdala and subgenual ACC (sgACC) (Davey et al., 2015) and the strength of connectivity between sgACC and dorsomedial frontal cortex correlates with the severity of depression (Davey, Harrison, Yücel, & Allen, 2012). Hypoconnectivity between AN and the prefrontal cortex (PFC) may underlie top-down emotion regulation deficits in MDD (Kaiser, Andrews-Hanna, Wager, & Pizzagalli, 2015).
- Salience Network (SN) with recent neuroimaging evidence showing almost twice as large SN in MDD compared to controls, including the regions of anterior insula, ACC, and lateral PFC. This enlargement precedes clinical depression in children who later develop depression, suggesting a potential risk biomarker (Lynch et al., 2024). During a depressive episode, changes in FC between SN nodes, particularly nucleus accumbens (NAc) with ACC and anterior insula, predict the emergence and remission of anhedonia and anxiety, respectively (Lynch et al., 2024).
- Reward Network (RN) with reduced connectivity between the regions of RN, which is associated with diminished motivation, interest, and anhedonia (Li et al., 2018). MDD is also associated with activation imbalances between RN (ventral striatum – reduced activation) and AN (Orbitofrontal Cortex – OFC – increased activation) during reward processing tasks (Ng, Alloy, & Smith, 2019).
- Default Mode Network (DMN) with hyperconnectivity within DMN nodes, which has been suggested to underlie excessive rumination in MDD, as an impairment of DMN-related self-referential mental activity (Kaiser et al., 2015).
- Central Executive Network (CEN) with increased DMN-CEN connectivity, which is viewed as DMN suppressing CEN and leading to a bias towards internal self-referential/ruminative thoughts (Young et al., 2023). Additionally, increased connectivity between CEN and the dorsal attention network has been found and is thought to be associated with diminished attention towards the external environment in MDD (Kaiser et al., 2015). CEN also shows increased connectivity with subcortical structures, such as the hippocampus, which may be related to the biased focus on unpleasant memories in MDD (Young et al., 2023). fMRI studies have shown DLPFC underactivation and VMPFC overactivation in depression (Koenigs & Grafman, 2009), while recovery is linked with 'normalization' of this pattern (Brody et al., 2001). The 'prefrontal asymmetry' theory is one of the classic theories in MDD, based on fMRI evidence of hypoactivity of the left and hyperactivity of the right DLPFC (rDLPFC) (Bruder, Stewart, & McGrath, 2017).

Brain lesion, TMS, and DBS studies identified a shared neural network for depression, including DLPFC, sgACC, and ventromedial prefrontal cortices (vmPFC). These regions align with the CEN and dorsal attention network (DAN) and correlate negatively with DMN and limbic networks (Siddiqi et al., 2021). These findings suggest that depression symptoms, whether caused by a primary psychiatric disorder (e.g. MDD) or structural brain lesions, may share common brain networks and highlight the potential of NIBS in studying and treating transdiagnostic psychiatric symptoms.

NIBS for MDD treatment

rTMS is the NIBS technique with the most robust evidence for clinical efficacy and treatment effect estimates for MDD, while evidence for the use of tDCS is evolving.

lDLPFC is the primary target for rTMS and tDCS using 'excitation' protocols to target 'prefrontal asymmetry'. It is assumed that high-frequency (HF >5 Hz) rTMS (HF-rTMS) induces cortical excitation, while low-frequency (LF 1 Hz) rTMS (LF-rTMS) induces inhibition (Pascual-Leone, Valls-Solé, Wassermann, & Hallett, 1994). Standard rTMS uses 10 Hz on lDLPFC, while tDCS applies 1–2 mA currents, both of which increase local cortical excitability (Brunoni et al., 2016).

Meta-analyses confirm the efficacy of rTMS as monotherapy and adjunctive treatment for depression (Brunoni et al., 2017; Vida

et al., 2023). Around 40% of TRD patients respond to rTMS versus 10% to sham, with remission rates of 36% versus 8% (Vida et al., 2023). rTMS is well-tolerated, acceptable, and cost-effective compared to multiple medication trials (Nguyen & Gordon, 2015; Voigt, Carpenter, & Leuchter, 2017). However, rTMS response rates vary widely, prompting protocol modifications such as bilateral DLPFC stimulation (HF on lDLPFC, and LF on rDLPFC) and priming (HF-rTMS before LF-rTMS) to optimize effects (Fitzgerald et al., 2008). Network analyses favor priming, bilateral rTMS, and bilateral theta burst stimulation, while accelerated, synchronized, and deep rTMS show no advantage over sham (Brunoni et al., 2017; Mutz et al., 2019; Shi et al., 2024).

tDCS in depression shows response and remission rates of 34% and 23%, respectively (Brunoni et al., 2016). Efficacy declines with treatment resistance (Brunoni et al., 2016; Mutz et al., 2019) and improves with longer sessions (Brunoni et al., 2016). A recent multisite home-based RCT found 2–3 times higher response and remission rates versus sham (Woodham et al., 2025). High acceptability, safety, portability, and cost-effectiveness position tDCS as a potential first-line depression treatment (Woodham et al., 2025).

Since depression symptoms have been shown to share neural networks regardless of their cause (Siddiqi et al., 2021), this suggests that both unipolar and bipolar depression should respond to similar NIBS protocols. Meta-analyses show that this may indeed be the case, with rTMS showing small but significant improvements in bipolar depression (Tee & Au, 2020). However, polarity-specific analyses found rTMS effective for unipolar but not bipolar depression (Hyde et al., 2022). Additionally, an iTBS trial targeting the left DLPFC in bipolar depression showed no efficacy (McGirr et al., 2021). In contrast, a more recent iTBS trial in bipolar depression using personalized lDLPFC targeting based on the FC between the sgACC and lDLPFC reported significant clinical improvements (Appelbaum et al., 2025). These conflicting findings emphasize the importance of personalized targeting to optimize the clinical efficacy of rTMS and to clarify whether rTMS can effectively treat bipolar depression or whether distinct pathophysiological patterns differentiate two similar phenotypes (unipolar and bipolar depression), requiring disorder-specific NIBS treatment protocols.

NIBS biomarkers

Combining TMS/tDCS with EEG and neuroimaging has shown potential for the identification of biomarkers of treatment response, enabling patient stratification and individualized treatment protocols to optimize treatment response.

EEG biomarkers

EEG predicts rTMS response more accurately than antidepressant response, as it better captures neural activity in targeted cortical networks (Watts et al., 2022), while being accessible, tolerable, and cost-effective. Individual Alpha Peak Frequency (IAPF) is the frequency of the strongest alpha oscillation (7–13 Hz). Patients with IAPF near 10 Hz show higher remission rates with 10 Hz lDLPFC rTMS, while those with higher IAPF respond better to 1 Hz right DLPFC rTMS (Voetterl et al., 2023), highlighting the potential of IAPF to stratify patients to more effective rTMS protocols based on individual pre-treatment oscillatory activity. Task-Induced Frontal-Midline Theta Power reflects task-induced rostral ACC (rACC) activity, a key hub of DMN, which plays an important role in depression pathophysiology. Changes in frontal-midline theta power following rTMS may differentiate responders from non-responders (Bailey et al., 2018; Li, et al., 2016).

Neuroimaging and TMS-EEG biomarkers

Dysfunctional sgACC is central to the pathophysiology of depression. It shows increased activity with reciprocal decreased rDLPFC activity during depressive episodes, with reversal of this pattern during depression recovery (Mayberg et al., 1999). Evidence suggests that DLPFC-sgACC connectivity may be a marker of rTMS in depression. TMS stimulation of the lDLPFC regions, which were more negatively correlated ('anti-correlated') with sgACC showed better clinical efficacy in MDD (Fox, Buckner, White, Greicius, & Pascual-Leone, 2012), highlighting the potential for the development of FC-based biomarkers to optimize clinical outcomes. More recently, computational models have been developed in large-scale FC datasets to enable FC-guided (sgACC-lDLPFC) personalization of rTMS in depression (Cash et al., 2021) and have recently been used in an iTBS trial in bipolar depression with positive results, as discussed above (Appelbaum et al., 2025). Combining TMS with electroencephalography (TMS-EEG) showed increased sgACC excitability and stronger effective connectivity between the sgACC and lDLPFC in depression, both of which decreased after rTMS treatment over the lDLPFC, and the reduction in connectivity correlated with symptom improvement (Hadas et al., 2019). Lower baseline glutamate in ACC is associated with better rTMS response (Gonsalves et al., 2024). tDCS was more effective in MDD patients with higher pre-treatment activation levels of the left PFC (Nord et al., 2019) and larger left PFC volumes (Bulubas et al., 2019).

Schizophrenia

Schizophrenia (SCZ) is a severe mental disorder affecting 1% of the population and characterized by significant heterogeneity in symptom presentation, treatment response, and prognosis. Current evidence suggests a multifactorial etiology involving neurodevelopmental, genetic, and environmental factors (Murray, Bhavsar, Tripoli, & Howes, 2017).

SCZ symptoms are grouped into positive, negative, and cognitive clusters, and empirical evidence from rsfMRI studies supports the '**disconnection hypothesis**,' which links symptoms to altered FC between PFC, subcortical (e.g. thalamic), and associative cortical (e.g. temporal) regions (Friston, Brown, Siemerkus, & Stephan, 2016; Friston & Frith, 1995). Hypoconnectivity is particularly evident in the frontal brain (Pettersson-Yeo, Allen, Benetti, McGuire, & Mechelli, 2011). Concurrent hypo- and hyperconnectivity patterns have been shown with reduced connectivity between DLPFC-limbic cortices and the mediodorsal thalamus and increased connectivity between primary-sensorimotor cortices and ventral thalamic nuclei. These FC alterations have been associated with SCZ symptoms (e.g. Anticevic et al., 2015).

Positive symptoms in SCZ correlate with hyperconnectivity of the primary-sensorimotor cortices to thalamic and striatal nuclei (Avram, Brandl, Bäuml, & Sorg, 2018). AVHs correlate with hyperconnectivity in the left auditory cortex and increased activity within the left temporoparietal cortex alongside reduced prefrontal top-down control (Shao, Liao, Gu, Chen, & Tang, 2021; Shergill, Brammer, Williams, Murray, & McGuire, 2000).

Negative symptoms have long been linked to dysfunctional PFC (Liddle, 1987), with functional neuroimaging studies showing associations with DLPFC and ventrolateral prefrontal cortex (VLPFC) activity (Goghari, Sponheim, & MacDonald, 2010). Negative symptoms have also been associated with altered FC between DLPFC and DMN-cerebellar circuits (Brady et al., 2019). Patients with SCZ and prominent avolition show disrupted FC between the ventral tegmental area (VTA) (a key source of mesocorticolimbic dopamine involved in reward and motivation) and cortical regions related to value processing and action selection, such as the bilateral VLPFC, insular cortex, lateral occipital cortex, and DLPFC (Giordano et al., 2018).

While the underlying causes of brain functional dysconnectivity in SCZ remain unclear, an optimal balance between excitatory (glutamate-mediated) and inhibitory (GABA-mediated) systems is critical for regulating information processing within and between neural networks (Turrigiano & Nelson, 2004). Disruption of the Excitation/Inhibition (E/I) balance is linked to SCZ pathophysiology, the lack of response of negative and cognitive symptoms to antipsychotics, as well as treatment resistance, which is observed in approximately 30% of patients (Howes & Shatalina, 2022).

TMS is uniquely placed for studying E/I balance and connectivity. Combined with electromyography (EMG), it enables non-invasive assessment of E/I indices via standardized primary motor cortex (M1) protocols, serving as a proxy for cortical dysfunction. A meta-analysis of TMS-EMG studies in SCZ found significant inhibition deficits, as measured by Short Interval Cortical Inhibition (SICI) (d = 0.62), supporting the E/I imbalance hypothesis and showing potential as a diagnostic and treatment biomarker (Lányi et al., 2024). TMS-EEG, which extends the methodology beyond M1, shows potential as a treatment response biomarker. This has been demonstrated in epilepsy (Gefferie et al., 2023) and is currently being investigated in SCZ (Di Hou, Santoro, Biondi, Shergill, & Premoli, 2021; Santoro et al., 2024).

NIBS treatments for Schizophrenia

NIBS has been used to treat treatment-resistant symptoms, including persistent positive (primarily AVHs) but also negative and cognitive symptoms, which do not respond to current treatments (Fusar-Poli et al., 2015).

AVHs

Most NIBS trials for AVHs apply left temporoparietal area (TPA) inhibition and frontal activation protocols based on evidence that therapeutic effects may result from the normalization of hyperconnectivity and increased activity in the left auditory cortex/TPA, as well as the restoration of the diminished top-down control from the PFC (Gromann et al., 2012). Typically, rTMS studies apply low-frequency rTMS (inhibition) (1 Hz) (LF-rTMS) to lTPA, while tDCS studies apply concurrent cathodal stimulation (inhibition) to lTPA and anodal stimulation (activation) to lDLPFC.

The treatment effects of both techniques are significant but small, ranging between 0.19 and 0.49 for rTMS (He et al., 2017; Hyde et al., 2022; Li, Cao, Liu, Li, & Xu, 2020; Otani, Shiozawa, Cordeiro, & Uchida, 2015; Slotema, Blom, Van Lutterveld, Hoek, & Sommer, 2014), with some trials reporting negative results (Li et al., 2020), and 0.42 for tDCS (Hyde et al., 2022). Both techniques have good tolerability with no significant differences in attrition rates between active and sham treatments (Slotema, Aleman, Daskalakis, & Sommer, 2012; Valiengo et al., 2020). The efficacy of rTMS on other positive symptoms, particularly delusions, is less robust and more variable across studies (Kennedy, Lee, & Frangou, 2018).

Combining neuroimaging with NIBS has highlighted the role of the left temporoparietal network in treatment response. Higher blood flow in the left superior temporal gyrus (STG) predicts rTMS response for AVHs (Homan, Kindler, Hauf, Hubl, & Dierks, 2012), while left STG FC predicts tDCS response for AVHs (Paul et al., 2022). Pre-treatment FC alterations in STG and decreased Degree Centrality (DC), which quantifies the magnitude of neural activity in a specific brain region relative to overall brain activity (Tomasi, Shokri-Kojori, & Volkow, 2016), in prefrontal and occipital cortices reverse post-treatment and correlate with symptom improvement (Xie et al., 2023).

Negative symptoms

Overall, NIBS has shown promising effects on negative symptoms, which are typically resistant to standard treatments and have a substantial impact on the functional outcomes and prognosis of SCZ (Rabinowitz et al., 2012).

Meta-analyses of rTMS RCTs showed significant small (0.41) to medium (0.64) effect sizes (Aleman, Enriquez-Geppert, Knegtering, & Dlabac-de Lange, 2018; Lorentzen, Nguyen, McGirr, Hieronymus, & Østergaard, 2022) compared to sham and significant small effects for tDCS (0.50) (Aleman et al., 2018). The most common targeted area across studies is the lDLPFC, with HF being the most efficacious for both rTMS (Lorentzen et al., 2022) and tDCS (Yu et al., 2020). A recent meta-analysis found that iTBS on the left dorsal PFC was effective for negative symptoms (Tan et al., 2023).

FC patterns in early SCZ have shown that greater negative symptom severity correlates with reduced rDLPFC connectivity to a network spanning cerebral and cerebellar DMN nodes, with the midline cerebellar node being the strongest predictor of symptom severity. rTMS targeting this region led to both symptomatic improvement and enhanced DLPFC-cerebellar FC, indicating a mechanism of clinical benefits (Brady et al., 2019). FC between VTA and DLPFC could be explored for personalized DLPFC targeting and prediction of treatment response in patients with prominent avolition (Giordano et al., 2018). Beyond FC patterns, structural markers such as pre-treatment grey matter density reductions in the prefrontal, insular, medial temporal, and cerebellar cortices, alongside increases in parietal and thalamic structures, have also been linked to rTMS response in predominantly negative SCZ (Koutsouleris et al., 2018).

Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD) is a chronic, heterogeneous disorder affecting 1%–4% of the population. Standard treatments include SSRIs and psychotherapies, but 30% of cases are treatment-resistant, affecting functional outcomes and quality of life (National Institute for Health and Care Excellence, 2024) and emphasizing the need for more effective treatments.

Traditionally, OCD has been associated with dysfunctional cortico-striato-thalamo-cortical (CSTC) networks (Alexander & Crutcher, 1990), resulting in hyperactive OFC-ventromedial caudate networks and hypoactive executive networks, including DLPFC and dorsolateral caudate.

FC studies showed altered connectivity within CSTC, including (a) dysconnectivity between striatal and cortical networks

(i.e. caudate hyperconnectivity with the fronto-limbic network and hypoconnectivity with frontoparietal network regions, along with NAc hypoconnectivity with fronto-limbic network regions); (b) hyperconnectivity between thalamus and striatum (putamen and caudate); and (c) dysconnectivity between ACC and fronto-limbic networks (Liu et al., 2022). The dorsal ACC, which is considered a 'hub' of OCD with dense connections to ventral affective and dorsal cognitive networks, is involved in cognitive control (CC) impairments in OCD and shows hyperactivity in rsfMRI studies (McGovern & Sheth, 2017).

Current OCD models have proposed the following networks in OCD (Shephard et al., 2021; van den Heuvel et al., 2016):

- The fronto-limbic network, which includes the amygdala and vmPFC, is involved in emotional responses, such as fear and anxiety, and shows aberrant activation in OCD during emotional processing tasks (Thorsen et al., 2018).
- The Sensorimotor Network (SMN). In the largest FC whole-brain analysis in OCD to date (Bruin et al., 2023), SMN showed the most significant FC hypoconnections among OCD brain networks. SMN has been linked with ordering, arranging, counting, and repeating compulsions and with sensory perceptions of 'feeling dirty' and associated washing and cleaning compulsions (Shephard et al., 2021). Among SMN nodes, fMRI studies have shown middle cingulate cortex hypoactivation and bilateral ACC hyperactivation in OCD with prominent washing (Yu et al., 2022) and supplementary motor area (SMA) hyperactivity, possibly reflecting response inhibition impairment in OCD (de Wit et al., 2012).
- The ventral cognitive network, which involves the inferior frontal gyrus (IFG), VLPFC, ventral caudate, and thalamus, and mediates response inhibition. IFG and caudate activity are reduced during inhibition tasks in OCD (Abramovitch, Abramowitz, & Mittelman, 2013).
- The ventral affective network, involving OFC, NAc, and thalamus, regulates reward processing. fMRI studies show hypoactivation of the NAc and OFC during reward anticipation and decision-making in OCD (Figee et al., 2011; Norman et al., 2018), along with hypoconnectivity between these regions, which correlates with more severe symptoms (Liu et al., 2022), suggesting reward-related impairments in OCD.
- The dorsal cognitive network, which is connected to CEN and is involved in top-down control of emotional, motor, and cognitive processes, such as response inhibition and cognitive flexibility, and working memory. It includes DLPFC, dorsomedial prefrontal cortex (dmPFC), dorsal caudate, thalamus, and pre-SMA and is suggested to contribute to OCD through executive dysfunction (Shephard et al., 2021).
- The triple model networks (DMN, FPN, SN) exhibit hypoconnectivity in OCD, potentially related to difficulties switching between repetitive thoughts and goal-directed actions (Bruin et al., 2023). Alteration in error-processing is a robust finding in OCD and is associated with increased activity in SN, DMN, SMN, and fronto-limbic networks and has been proposed as an endophenotype for OCD (Riesel et al., 2019).

NIBS treatments for OCD

Deep rTMS, which penetrates deeper brain structures compared to traditional TMS, received FDA approval for OCD in 2018 with HF (20 Hz) bilateral medial PFC/ACC stimulation, showing a 38% response rate versus 11% for sham. This was followed by approval of HF bilateral deep rTMS over dmPFC in 2020. NICE considers the evidence insufficient to recommend rTMS for OCD (National Institute for Health and Care Excellence, 2020).

rTMS is effective for OCD, with effect sizes ranging from small (0.43) to large (0.79) with high heterogeneity in most studies (Kar, Agrawal, Silva-dos-Santos, Gupta, & Deng, 2024), with deep TMS being superior to traditional (Suhas et al., 2023). DLPFC, pre-SMA, and OFC have also been targeted in OCD studies, but evidence remains inconclusive due to small samples and protocol variabilities (Grassi, Moradei, & Cecchelli, 2023).

FDA-approved OCD protocols involved HF ('excitatory') rTMS, despite targeting hyperactive CSTC networks. This may seem paradoxical, as LF ('inhibitory') protocols would be expected to induce therapeutic effects in this case. Accumulating evidence suggests that the distinction between HF-excitatory/LF-inhibitory stimulation may be oversimplified. For example, in smokers, HF and not LF rTMS to the hyperactive insula reduced cigarette consumption (Dinur-Klein et al., 2014). HF rTMS acts as a neuromodulator and not just as an excitatory tool, potentially 'resetting' dysregulated networks through synaptic plasticity changes, altered inhibitory interneuron activity, modified oscillatory patterns, and restored FC (Fitzsimmons, Oostra, Postma, Van Der Werf, & Van Den Heuvel, 2024). However, both HF and LF are effective in OCD, though iTBS, an excitatory protocol, has not shown efficacy (Kar et al., 2024). Research is needed to understand this lack of clinical benefit and further explore excitatory/inhibitory rTMS protocols. On the other hand, tDCS results are inconsistent, with some studies showing improvement (Xie et al., 2024) and others showing no effects (Pinto et al., 2022).

Treatment biomarkers for rTMS in OCD are under investigation. SMN and SN may have potential as treatment biomarkers. SMN shows the most significant hypoconnections in OCD and its error-related activity has been associated with treatment response in CBT with higher levels of pre-treatment activity predicting better response (Grützmann et al., 2022). Increases in FC between the SMN and DMN correlated with symptomatic improvement in a small tDCS clinical trial (Echevarria et al., 2024). Hypoconnectivity between SN and frontoparietal networks and increased SN activity in activation studies have also been consistently shown in OCD (Perera, Gotsis, Bailey, Fitzgibbon, & Fitzgerald, 2024).

Cognitive impairment

Cognitive impairment (CI) is a common feature across multiple psychiatric disorders. A recent systematic review of meta-analyses of neurocognitive studies showed impaired cognition across all psychiatric disorders, indicating CI as a transdiagnostic feature. Most disorders show small to medium effect sizes of impairment across cognitive domains, while SCZ and bipolar disorder typically exhibit larger effect sizes (Abramovitch, Short, & Schweiger, 2021). CI significantly impairs functional outcomes, particularly in psychotic disorders, and conventional treatments offer little benefit, highlighting the need for more effective treatments (Sheffield, Karcher, & Barch, 2018).

In line with neurocognitive evidence, current neuroimaging evidence suggests a unifying network model for CI across psychiatric disorders. rsfMRI meta-analysis showed common FC alterations in the 'triple network model' associated with CI across eight psychiatric disorders (including SCZ, Bipolar Disorder,

Depression, and OCD), with hypoconnectivity between DMN and ventral SN and between SN and FPN, and hyperconnectivity between DMN and FPN and between DMN and dorsal SN (Sha, Wager, Mechelli, & He, 2019). In a meta-analysis of fMRI studies in SCZ, unipolar and bipolar depression, anxiety disorders, and substance use, transdiagnostic abnormal activation was found in SN areas, including left PFC, anterior insula, right VLPFC, right intraparietal sulcus, mid-cingulate/pre-SMA, and dorsal ACC (McTeague et al., 2017). The triple model networks are involved in cognitive control (CC), the ability to regulate goal-directed behavior flexibly and adaptively in response to changing environmental demands, and CC has been suggested to underlie CI across psychiatric disorders (McTeague, Goodkind, & Etkin, 2016; Menon, 2020).

So far, the effects of NIBS on cognitive symptoms are rather inconsistent and appear to be domain-specific. For example, improvements in working memory and executive functions have been shown with corresponding changes in frontal cortical activity in a combined tDCS-fMRI study (Orlov et al., 2017). A recent meta-analysis found small but significant transdiagnostic effects of TMS and tDCS on working memory, with tDCS also improving attention/vigilance across brain disorders (including SCZ depression, dementia, Parkinson's disease, stroke, traumatic brain injury, and multiple sclerosis), with no significant differences among disorders (Begemann, Brand, Ćurčić-Blake, Aleman, & Sommer, 2020). Combining tDCS with cognitive training showed significant longer-term improvements on working memory (Orlov et al., 2017) and stochastic learning in SCZ (Orlov et al., 2022). A recent systematic review and meta-analysis showed small yet significant improvements in attention and working memory in neurological and psychiatric disorders, including SCZ (Burton et al., 2023). lDLPFC is the most common target in rTMS (Jiang et al., 2019), while tDCS studies commonly apply anodal stimulation of lPFC/lDLPFC with various cathodal placements (Stuchlíková & Klírová, 2022).

The unifying model of CI across psychiatric disorders highlights CC as a key target for NIBS treatments and cognitive training. CC impairments have also been linked to persistent psychotic symptoms (Horne et al., 2022) and treatment-resistant SCZ (Horne et al., 2021), and combining NIBS with cognitive training targeting CC may also offer a promising approach for difficult-to-treat SCZ.

Concluding remarks and emerging prospects

Understanding psychiatric disorders as brain network-based conditions has created new opportunities for targeted, personalized, and mechanism-based therapeutic interventions. The growing body of NIBS research in psychiatric disorders has recognized the variability in its response and is evolving to develop novel treatment protocols and identify biomarkers of response. However, there is a corpus of challenges to widespread therapeutic use.

A major challenge is precision in brain targeting, which depends on our understanding of the neural networks implicated in psychiatric disorders and on patient-specific patterns of network dysfunction. At the disorder level, as discussed above, neuroimaging studies have shown both common and distinct neural networks involved in psychiatric disorders, highlighting the importance of exploring the effects of targeting both for effective treatments. At the patient level, adapting NIBS protocols based on individual network dysfunction patterns has improved outcomes. For example, as discussed above, stimulation of lDLPFC regions, which were negatively correlated with sgACC showed better clinical efficacy in unipolar and bipolar depression (Appelbaum et al., 2025; Fox et al., 2012; Hadas et al., 2019). This suggests that a 'one brain site fits all' approach, using a single brain target for all patients with a specific disorder, is unlikely to further improve treatment effectiveness. A more comprehensive understanding of patient-specific brain changes, and their network context, will be necessary to develop more effective, and personally tailored, interventions. To this end, the combination of NIBS with neuroimaging/TMS methods is essential for meaningful research in the network abnormalities at the patient level and the mechanisms of treatment response. Furthermore, the inclusion of mechanistic studies in the treatment trials (e.g. EEG, MRI) is essential to explore the mechanisms of action of NIBS and their association with symptomatic improvements.

Precision in brain targeting can be improved with emerging NIBS techniques, such as low-intensity focused ultrasound (FUS) (Figure 1), which allows for deeper and more targeted stimulation of both cortical and subcortical brain regions with millimetre precision relative to TMS and tDCS. Though still in early research stages, FUS has shown promising results in preliminary trials for depression, SCZ, and anxiety (Shi & Wu, 2025).

While neuroimaging, especially rsFC, has revealed network abnormalities in psychiatric disorders and informed personalized NIBS protocols, it remains unclear whether these abnormalities are causes or consequences of the disorders since rsFC is inherently correlational. Integrating genetic and rsFC data can clarify causal links and inform treatment targets. In this context, a recent study integrating genetic and rsfMRI found that schizophrenia risk was linked to increased DMN and CEN connectivity and reduced attention network connectivity (Mu, Dang, & Luo, 2024). These findings are significant for NIBS, not only for treatment but also for preventative targets for at-risk individuals and may enable earlier interventions to modify the course of psychiatric disorders.

Inclusion of pre-treatment network physiological properties in NIBS studies is an important factor for treatment response. For example, pre-TMS neural activity predicts post-TMS responses (Pasley, Allen, & Freeman, 2009), supporting its use for patient stratification and treatment optimisation. Pre-NIBS treatment measures include EEG, fMRI for activity levels, rsfMRI for connectivity strength, and TMS measures of cortical E/I. One such example is SICI, a marker of cortical GABA-A inhibition, which is reliably reduced in SCZ and may enable disorder-specific and treatment biomarkers (Lányi et al., 2024).

Variations in the stimulation parameters, including intensity, number of pulses, sham procedures for rTMS (Li et al., 2020), and number of sessions and frequency of stimulation for tDCS (Yang et al., 2019) may affect their therapeutic efficacy and highlight the need for refinement and standardization of treatment protocols.

NIBS offers a promising therapeutic strategy, either alone or in combination with existing therapeutic approaches for psychiatric disorders and symptoms that fail to respond to conventional treatments. Large-scale, RCTs with long-term follow-up are essential to establish optimal protocols and evaluate safety comprehensively. Efforts should also be directed towards the development of more practical and accessible treatment systems and training programs to facilitate more widespread clinical use. While challenges remain, ongoing research is bringing NIBS closer to becoming a mainstream, patient-centered, mechanism-based treatment for psychiatric disorders and potentially offering earlier interventions that could modify the course of psychiatric disorders.

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