

Modulation of cortical excitability through non-invasive brain stimulation: Evidence from fMRI and EEG studies.

Hiroshi Tanaka*

Department of Neurophysiology, Kyoto University, Japan.

*Correspondence to: Hiroshi Tanaka, Department of Neurophysiology, Kyoto University, Japan, E-mail: h.tanaka@kyoto.edu

Received: 03-Jan-2025, Manuscript No. AANR-25-169335; **Editor assigned:** 04-Jan-2025, PreQC No. AANR-25-169335(PQ); **Reviewed:** 18-Jan-2025, QC No. AANR-25-169335; **Revised:** 21-Jan-2025, Manuscript No. AANR-25-169335(R); **Published:** 28-Jan-2025, DOI:10.35841/aanr-7.1.178

Introduction

Non-invasive brain stimulation (NIBS) techniques have emerged as valuable tools for modulating cortical excitability and probing the causal relationships between brain activity and behavior. Techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) offer the ability to influence neural processing without the need for surgical intervention. These methods apply magnetic or electrical currents to the scalp to alter membrane potentials and synaptic plasticity in targeted cortical areas. As a result, NIBS has been widely adopted in both basic neuroscience research and clinical rehabilitation. The study of how these interventions modulate cortical excitability is central to understanding their mechanism of action, and neuroimaging tools such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) have provided rich evidence regarding their neurophysiological impact. Integrating these imaging techniques with NIBS protocols has helped delineate the spatial and temporal characteristics of induced cortical changes, leading to improved targeting, efficacy, and personalization of interventions [1].

TMS, in particular, has been used extensively to investigate the effects of stimulation on localized cortical regions and their connected networks. Single-pulse TMS allows researchers to assess motor evoked potentials and transient cortical inhibition, whereas repetitive TMS (rTMS) can induce lasting changes in excitability that resemble long-term potentiation (LTP) or long-term depression (LTD). Studies using concurrent TMS-fMRI setups have shown that high-frequency stimulation over the motor cortex increases BOLD signal not only at the site of stimulation but also in remote, functionally connected regions such as the premotor and supplementary motor areas. These findings support the hypothesis that TMS induces widespread network-level changes, possibly mediated by oscillatory entrainment or synaptic modulation. Similarly, paired-pulse TMS studies coupled with EEG have revealed that different interstimulus intervals can modulate intracortical inhibition or facilitation, offering insight into the balance between excitatory and inhibitory influences in cortical circuits. Such electrophysiological correlates are invaluable for tailoring stimulation protocols to maximize therapeutic benefits and minimize adverse effects [2].

tDCS, though less focal than TMS, has gained popularity due to its ease of use, safety, and low cost.

Citation: Tanaka H. Modulation of cortical excitability through non-invasive brain stimulation: Evidence from fMRI and EEG studies. *Neurophysiol Res.* 2025;7(1):178.

It applies weak electrical currents through scalp electrodes to induce polarity-specific shifts in neuronal excitability. Anodal stimulation is generally associated with depolarization and increased excitability, whereas cathodal stimulation tends to hyperpolarize neurons and reduce excitability. The modulatory effects of tDCS are often subtle and state-dependent, influenced by ongoing brain activity, individual differences, and task engagement. When combined with fMRI, tDCS has been shown to modulate task-related BOLD responses, particularly in prefrontal and parietal areas involved in working memory, attention, and motor planning. These changes may not always align with behavioral outcomes, underscoring the complex interplay between neural modulation and cognitive performance. EEG studies have further revealed that tDCS can alter resting-state oscillatory activity, particularly in theta and alpha bands, and these changes may correlate with improvements in learning or cognitive flexibility. The ability to monitor such dynamics in real time enhances our understanding of how tDCS affects network-level communication [3].

The integration of NIBS with imaging modalities has also been instrumental in uncovering individual variability in stimulation responses. Inter-individual differences in skull thickness, cortical folding, and baseline excitability can significantly alter the electric field distribution and its effects on brain function. Recent advances in computational modeling now allow researchers to simulate current flow and optimize electrode placement for maximal efficacy. fMRI studies using individualized stimulation maps have shown improved outcomes in both research and clinical settings. EEG-informed closed-loop NIBS protocols are also being explored, wherein stimulation is delivered in synchrony with specific oscillatory phases or patterns. This approach holds promise for enhancing plasticity in a targeted and

efficient manner. Moreover, studies using simultaneous TMS-EEG have identified evoked potentials and oscillatory phase resetting as biomarkers of effective stimulation. These metrics may serve as indicators for real-time adjustment of stimulation parameters, advancing the goal of precision neuromodulation. Such developments are particularly valuable in clinical populations where heterogeneity in response to treatment is a major challenge [4].

Applications of NIBS have extended across various domains including motor rehabilitation post-stroke, cognitive enhancement in healthy individuals, and symptom relief in psychiatric disorders such as depression, anxiety, and schizophrenia. In depression, for example, high-frequency rTMS targeting the left dorsolateral prefrontal cortex has shown consistent antidepressant effects, corroborated by increased connectivity within mood-regulating networks as observed in fMRI studies. In Parkinson's disease, NIBS has been used to improve motor function by modulating cortical excitability in the motor cortex and its downstream circuits. EEG studies in these contexts have provided markers of disease severity and treatment responsiveness, such as changes in beta band power or coherence. These findings not only validate the physiological impact of NIBS but also inform protocol design for maximizing therapeutic outcomes. Furthermore, combining NIBS with behavioral training or cognitive tasks may produce synergistic effects by engaging neural circuits during plasticity-inducing stimulation. Continued integration of multimodal neuroimaging with NIBS will refine our ability to target specific brain networks and dynamically monitor treatment effects [5].

Conclusion

The modulation of cortical excitability through non-invasive brain stimulation techniques like TMS and tDCS has opened new avenues for both neuroscience research and clinical intervention. Coupling these stimulation methods with neuroimaging tools such as fMRI and EEG has significantly advanced our

Citation: Tanaka H. Modulation of cortical excitability through non-invasive brain stimulation: Evidence from fMRI and EEG studies. *Neurophysiol Res.* 2025;7(1):178.

understanding of the mechanisms underlying their effects. These technologies allow researchers to observe changes in brain activity with high spatial and temporal resolution, capturing both local and network-level dynamics. The evidence from imaging studies supports the idea that NIBS can induce plastic changes in neural circuits, though the extent and direction of these changes depend on several factors including stimulation parameters, individual brain anatomy, and cognitive context. As personalized medicine continues to evolve, the ability to model, monitor, and adapt brain stimulation in real time will become increasingly central to therapeutic applications. Ultimately, the synergy between NIBS and neuroimaging offers a powerful framework for exploring and enhancing brain function, with promising implications for the treatment of neurological and psychiatric conditions.

References

1. Chaurasia BD. Calvarial defect in human anencephaly. *Teratol.* 1984;29:165-72.
2. Muller F, O'Rahilly R. Cerebral dysraphia (future anencephaly) in a human twin embryo at stage 13. *Teratol.* 1984;30:167-77.
3. Papp Z, Csecsei K, Toth Z, et al. Exencephaly in human fetuses. *Clin Genet.* 1986;30:440-4.
4. Sadovnick AD, Baird PA. Congenital malformations associated with anencephaly in liveborn and stillborn infants. *Teratol.* 1985;32:355-61.
5. Urich H, Herrick MK. The amniotic band syndrome as a cause of anencephaly: Report of a case. *Acta Neuropathol.* 1985;67:190-4.