Short Communication
Volume 11:01, 2025

Clinical Depression

ISSN: 2572-0791

Open Access

Neuroplasticity and Mood Regulation: Exploring the Mechanisms of tDCS in Major Depression

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Introduction

Major Depressive Disorder (MDD) is a complex and multifaceted psychiatric condition that continues to pose a significant challenge for clinicians and researchers alike. Characterized by persistent sadness, loss of interest or pleasure in daily activities, cognitive disturbances, and functional impairment, MDD affects over 264 million people globally and is a leading cause of disability worldwide. Despite decades of research and a range of available treatments, a substantial proportion of individuals with MDD either fail to respond to first-line interventions or experience only partial relief from symptoms. This has led to a growing interest in novel therapeutic strategies, especially those that target the brain's underlying functional and structural abnormalities. Among these, non-invasive brain stimulation techniques such as transcranial direct Current Stimulation (tDCS) have emerged as promising tools in the treatment of mood disorders. Central to the potential efficacy of tDCS is its impact on neuroplasticity-the brain's ability to adapt and reorganize itself functionally and structurally in response to internal and external stimuli [1].

Description

Transcranial direct current stimulation operates by delivering a low-intensity electrical current, typically 1 to 2 milliamperes, through electrodes placed on the scalp. This current modulates neuronal excitability in a polarity-dependent manner: anodal stimulation generally enhances cortical excitability, while cathodal stimulation reduces it. When applied repeatedly over time, these modulations can lead to enduring changes in synaptic efficacy and network dynamics-hallmarks of neuroplasticity. The dorsolateral prefrontal cortex (DLPFC), often targeted in tDCS protocols for depression, plays a key role in emotion regulation, executive functioning, and cognitive control [2]. In individuals with depression, neuroimaging studies frequently reveal hypoactivity in the left DLPFC and hyperactivity in limbic structures such as the amygdala, reflecting an imbalance in top-down regulatory mechanisms. tDCS, by modulating DLPFC excitability, may help restore this balance, improving mood and cognitive function [3].

The neuroplastic mechanisms underlying tDCS involve a complex interplay of cellular, molecular, and network-level changes. At the synaptic level, tDCS has been shown to facilitate long-term potentiation (LTP) and long-term depression (LTD)-like processes, akin to those observed in learning and memory. These changes are thought to be mediated by alterations in NMDA receptor activity, intracellular calcium signaling, and the expression of neurotrophic factors such as brain-derived neurotrophic factor (BDNF). BDNF, in particular, has been implicated in both the pathophysiology of depression and the therapeutic response to antidepressant treatments. Reduced levels of BDNF are commonly observed in individuals with MDD and are associated with impaired synaptic plasticity and neuronal atrophy, particularly in the hippocampus and prefrontal cortex. Studies have demonstrated that tDCS can upregulate BDNF expression, thereby supporting synaptic resilience and promoting the restoration of functional neural circuits [4].

Beyond the molecular level, tDCS exerts significant influence on large-scale brain networks involved in mood regulation. Functional connectivity studies using resting-state fMRI have shown that tDCS modulates activity in the default mode network (DMN), salience network, and cognitive control network-systems that are often dysregulated in depression. For instance, hyperconnectivity within the DMN is associated with maladaptive rumination, a core feature of depression, while impaired connectivity in the cognitive control network correlates with difficulties in executive function and emotional regulation. Anodal tDCS applied to the left DLPFC has been found to reduce DMN hyperconnectivity and enhance connectivity within the cognitive control network, thereby shifting the brain's functional architecture towards a more adaptive state. These changes are not merely correlational; emerging evidence suggests that the degree of network modulation following tDCS may predict clinical response, highlighting its potential as both a therapeutic and diagnostic tool [5].

Conclusion

In conclusion, transcranial direct current stimulation represents a compelling advancement in the treatment of major depression, particularly in its ability to directly modulate neuroplasticity and large-scale brain networks implicated in mood regulation. Unlike traditional pharmacological interventions that target neurotransmitter systems diffusely, tDCS offers a more targeted and customizable approach that aligns with the evolving neurocircuitry models of depression. Its capacity to induce LTP-like changes, modulate functional connectivity, and enhance neurotrophic support underscores its relevance as both a therapeutic tool and a window into the mechanisms of emotional regulation. As the field moves toward more personalized, circuit-based models of care, the role of tDCS in promoting adaptive plasticity and restoring affective balance is likely to grow. However, to fully realize its clinical potential, ongoing research must continue to refine its application, identify predictors of response, and integrate it thoughtfully into broader treatment ecosystems. Through such efforts, tDCS may help reshape not only the treatment of depression but also our broader understanding of how the brain heals and adapts in the face of emotional suffering.

Acknowledgment

None.

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Received: 28 January, 2025, Manuscript No. cdp-25-165826; **Editor assigned:** 30 January, 2025, Pre QC No. P-165826; **Reviewed:** 13 February, 2025, QC No. Q-165826; **Revised:** 20 February, 2025, Manuscript No. R-165826; **Published:** 27 February, 2025, DOI: 10.37421/2572-0791.2025.11.163

Conflict of Interest

None.

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How to cite this article: Hannon, Sheline. "Neuroplasticity and Mood Regulation: Exploring the Mechanisms of tDCS in Major Depression." *Clin Depress* 11 (2025): 163.