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Frontal Cortex Modulation *via* tDCS in Patients with Anhedonia: Targeting the Reward Circuitry in Depression

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Introduction

Major Depressive Disorder (MDD) is a heterogeneous psychiatric condition characterized by a wide range of affective, cognitive, and somatic symptoms. Among its core symptoms, anhedonia—defined as a marked reduction in the ability to experience pleasure or interest in previously enjoyable activities—stands out as a particularly debilitating feature. Unlike general low mood, anhedonia is more directly linked to disruptions in reward processing and motivation. It is associated with poor prognosis, lower treatment response, and higher rates of chronicity. Anhedonia has been increasingly conceptualized not only as a symptom but as a distinct dimension of depression tied to dysfunction within the brain's reward circuitry. Central to this system are frontostriatal pathways, particularly involving the prefrontal cortex, ventral striatum, and anterior cingulate cortex. Emerging research indicates that targeted modulation of this circuitry may alleviate anhedonia more effectively than conventional antidepressant treatments, which often fail to address motivational deficits. In this context, transcranial Direct Current Stimulation (tDCS), a non-invasive neuromodulation technique, is gaining traction as a potential tool for modulating frontal cortex activity and influencing downstream reward-related processes [1].

Description

tDCS involves the application of a low-intensity, continuous electrical current via electrodes placed on the scalp. Typically, anodal stimulation increases cortical excitability, while cathodal stimulation decreases it. This modulation of neural excitability is believed to influence synaptic plasticity and alter functional connectivity within and between key brain networks. In the treatment of depression, tDCS most often targets the Dorsolateral Prefrontal Cortex (DLPFC), especially the left hemisphere, which is implicated in executive control, emotion regulation, and goal-directed behavior. In individuals with MDD and pronounced anhedonia, the DLPFC is frequently hypoactive, while regions involved in negative affect and internal mentation, such as the subgenual anterior cingulate cortex and default mode network (DMN), show hyperconnectivity. These imbalances contribute to diminished top-down regulation of the reward system, impairing the motivational salience of rewarding stimuli [2].

By increasing activity in the left DLPFC, tDCS may help restore balance in prefrontal-limbic interactions and enhance reward responsiveness. Studies employing behavioral and neuroimaging paradigms suggest that tDCS can improve reward-related processing by influencing dopaminergic pathways and modulating striatal activity indirectly [3,4]. This is particularly relevant for anhedonia, which has been associated with blunted responses in the nucleus accumbens and ventral tegmental area—regions critical for coding reward prediction and hedonic impact. Although tDCS does not directly stimulate subcortical structures, its influence on cortical regions that project to the striatum may facilitate downstream effects on dopamine release and reward anticipation. Several trials have explored this hypothesis by examining the impact of repeated anodal tDCS over the left DLPFC in patients with MDD and elevated anhedonic symptoms. Findings suggest that this approach can lead to measurable improvements in reward sensitivity, motivation, and engagement in pleasurable activities, though variability in outcomes persists.

From a mechanistic standpoint, tDCS-induced changes in cortical excitability can lead to alterations in neuroplasticity, including enhanced long-term potentiation (LTP)-like effects, increased expression of brain-derived neurotrophic factor (BDNF), and improved functional connectivity within frontostriatal networks. These changes are not immediate but accumulate over time with repeated stimulation sessions. This time-dependent nature of tDCS is consistent with the slow development of therapeutic effects and highlights the importance of treatment adherence and protocol optimization. Moreover, the state-dependency of tDCS effects implies that concurrent cognitive or emotional engagement during stimulation may enhance its impact. For instance, coupling tDCS with reward-based tasks or behavioral activation strategies may facilitate targeted neuroplasticity within the desired neural circuits, increasing the salience and reinforcing value of positive experiences [5].

Conclusion

In conclusion, transcranial direct current stimulation targeting the frontal cortex represents a promising intervention for patients with depression characterized by anhedonia. By modulating activity in the DLPFC and its associated reward circuitry, tDCS can influence key neural processes underlying motivational and hedonic deficits. Through mechanisms involving neuroplasticity, dopaminergic modulation, and changes in network connectivity, tDCS offers a pathway to improve symptoms that are often refractory to conventional treatments. The emerging evidence supports the therapeutic potential of this technique, especially when integrated with behavioral strategies that promote reward engagement. However, to fully establish its role in clinical practice, further research is needed to refine treatment protocols, identify biomarkers of response, and determine long-term efficacy. As our understanding of the neural basis of anhedonia deepens, targeted neuromodulation strategies like tDCS may play an increasingly central role in the personalized treatment of depression, helping patients reconnect with experiences of pleasure and meaning that are so often lost in this condition.

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Conflict of Interest

None.

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