Induction of Neuroplasticity by Transcranial Direct Current Stimulation

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Brain stimulation through feeble direct current has recently been declared as a new technique to induction of neuroplasticity of the cerebral cortex for improvement of neurological disorders like Schizophrenia, Alzheimer’s and Parkinson’s diseases (PD). Although brain imaging and electroencephalography (EEG) are used in the early assessment of neurological disorders [1-3], it’s unfortunate that many symptoms of neurological disorders appear after the extensive loss of structure and function of neurons, consequently, these methods are not useful for early detection or prevention of these diseases. Despite the remarkable therapeutic effects of brain stimulation, the exact mechanisms underlying the therapeutic effects of transcranial direct current stimulation (tDCS) and other brain stimulation methods have not been wholly understood [3]. However, several recent studies have emphasized the potential of non-invasive brain stimulation (NIBS) to improve neuroplasticity in neurological disorders [4-8]. Neuroplasticity is defined as the brain’s capability to reorganize itself via creating new neural networks and allows the neurons to regulate their actions in response to new circumstances. It has been shown that tDCS as a NIBS is talented to enrich neuroplasticity processes at least in healthy elderly [9]. It has been demonstrated that anodal tDCS (Positive stimulation) increases excitability and causes a depolarization of the resting membrane potential, which increases neuronal excitability and allows for more spontaneous cell firing, while cathodal tDCS (negative stimulation) reduces it and causes a hyperpolarization of the resting membrane potential [5, 6]. In spite of the exact mechanisms underlying the therapeutic effects of tDCS has not been completely understood, however, it has been proposed that anodal tDCS induces long-term potentiation and long-term depression (LTP/LTD)-like plasticity [6, 10, 11]. For instance, the NMDA receptors (glutamatergic system) seem to be indispensable for induction and the preservation of neuroplastic after-effects excitability induced by tDCS [12]. Along these lines, Liebetanz and coworkers (2002) have demonstrated that the NMDA antagonist dextromethorphan prevent lasting effects of tDCS on motor evoked potentials [13]. In addition, the duration of tDCS-induced excitability enrichment selectively potentiates by d-cycloserine (a partial NMDA-agonist) [14]. Likewise, NIBS could result in alterations in inhibitory GABAergic (Gamma-Amino Butyric Acid) systems which play a critical role in improving neuroplasticity.
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Furthermore, magnetic resonance spectroscopy has shown that anodal tDCS have been reduced the concentration of GABA [16]. Fritsch and colleagues (2010) have displayed that anodal tDCS with repetitive low-frequency synaptic (LFS) increase Brain-Derived Neurotrophic Factor (BDNF) secretion and Tropomyosin receptor kinase B (TrkB) activation, suggesting that BDNF is a crucial mediator of the tDCS-induced after-effects [17]. Additionally, the expression of zif268 (Zinc Finger Protein 268) and c-fos (two intensely involved proteins in schizophrenia) were quickly increased by tDCS which probably mediated through LTP [18].

It has been reported that anodal tDCS on the left dorsolateral prefrontal cortex (current strength: 2 mA) has improved the performance of working memory as an important executive function [5, 19, 20]. It has been demonstrated that tDCS is well-stabilized new intervention to modify attention [21], learning and memory [22], episodic memory [23, 24] and the other cognitive and motor performance. Moreover, it has been found that tDCS adjusts functional connectivity of the thalamocortical and corticostriatal circuits [25] and it was proposed that tDCS could also have a neuroprotective role in PD via reducing the oxidative stress in the dopaminergic terminals [26].

Despite increasing use of tDCS in experimental and clinical situations, numerous open questions persist as to the detailed mechanisms and functions of tDCS; predominantly in terms of the neurochemical and genomic effects of tDCS in the brain. Nevertheless, several of the cellular and molecular studies are required to determine the exact mechanism of tDCS.

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Conflict of Interest
There is no actual or potential conflict of interest regarding this article.

References


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