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OPINION

Integrating EMDR with tDCS in Obsessive–Compulsive Disorder: An Opinion Paper

Niccolò Pozzato[1†], Daniele Saccenti[1,2†], Andrea Stefano Moro[1,2,3], Walter Sapuppo[1,4,5], Jacopo Lamanna[3,6], and Mattia Ferro[1,2,3*]

[1]Department of Psychology, Sigmund Freud PrivatUniversität Wien GmbH, Milan, Italy. [2]Brain and Behaviour SFU Lab, Sigmund Freud PrivatUniversität Wien GmbH, Milan, Italy. [3]Center for Behavioral Neuroscience and Communication (BNC), Vita-Salute San Raffaele University, Milan, Italy. [4]Studi Cognitivi, Cognitive Psychotherapy School and Research Center, Milan, Italy. [5]Inclusive Network for Sport, Personal growth, envIronmental health and REsearch (INSPIRE) Lab, Sigmund Freud PrivatUniversität Wien GmbH, Milan, Italy. [6]Faculty of Psychology, Vita-Salute San Raffaele University, Milan, Italy.

*Address correspondence to: m.ferro@milano-sfu.it
†These authors contributed equally to this work.

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Current evidence indicates that approximately 25% of patients with obsessive–compulsive disorder (OCD) fail to respond to conventional therapies. Although trauma exerts a substantial role in the etiology of OCD, eye movement desensitization and reprocessing (EMDR) has shown limited evidence in treating such condition, highlighting the need for novel interventions. As a result, recent clinical advances suggest that combined approaches integrating multiple psychological interventions could be effective in yielding positive therapeutic outcomes. Concurrently, neuroimaging studies indicate that EMDR modulates neuronal activity within the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC), both implicated in OCD pathophysiology. Building on this evidence, noninvasive brain stimulation techniques, including transcranial direct current stimulation (tDCS), have demonstrated promise for OCD treatment by modulating OFC neuroplasticity. Hence, this opinion paper proposes a therapeutic strategy combining EMDR with tDCS to concomitantly target trauma-related psychological processes and rehabilitate neural dysfunction in the OFC and ACC. Such a novel therapeutic approach may enhance treatment effectiveness and offer an option for treatment-resistant OCD.

Introduction to Obsessive–Compulsive Disorder: Etiology, Pathophysiology, and Clinical Challenges

Obsessive–compulsive disorder (OCD) is a psychopathological condition characterized by the presence of obsessions and/or compulsions, with a lifetime prevalence estimated at 2% to 3% [1]. While obsessions are defined as images, thoughts, or impulses that are intrusive, persistent, and associated with anxiety, compulsions are behaviors or mental acts performed repetitively in response to an obsession to alleviate anxiety [2]. OCD onset generally occurs between the ages of 18 and 29 [3]; however, 30% to 50% of cases are identified in childhood [4].

According to the latest National Institute for Health and Care Excellence guidelines, the first-line interventions for OCD are exposure and response prevention (ERP) and selective serotonin reuptake inhibitors (SSRIs). Nevertheless, pharmacological interventions are often accompanied by negative side effects that lead many individuals to discontinue treatment [5], with an estimated 25% to 30% of patients failing to respond to first-line therapies [6]. Thus, treatment-resistant OCD patients represent a critical challenge for clinicians.

Understanding the etiology of OCD may be pivotal in overcoming treatment resistance. However, the disorder's underlying mechanisms remain unclear: according to Pauls et al. [4], OCD's etiology is multifactorial, involving interactions between genetic and environmental risk factors. Among these, scholars have investigated the contribution of psychological trauma to OCD development: some patients attribute OCD onset to identifiable stressful life events (SLEs), with childhood emotional abuse and parental neglect being associated with greater symptom severity [7]. Indeed, evidence suggests that childhood trauma substantially increases the risk of OCD onset [8]. Moreover, according to Cromer et al. [9], 54% of OCD patients report experiencing at least one traumatic life event. Notably, Gershuny et al. [10] indicated that 82% of treatment-resistant OCD patients disclosed a history of trauma, with approximately 40% meeting the diagnostic criteria for posttraumatic stress disorder (PTSD). Interestingly, Ruscio et al. [3] observed that the lifetime comorbidity rate between PTSD and OCD is

around 19%. Importantly, OCD and PTSD exhibit comparable symptoms, including persistent intrusions, depressive symptoms, and abnormal arousal responses [11].

Researchers have also explored the pathophysiological substrates of OCD to gain a broader understanding of the disorder: neuroimaging studies identified the orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC), both components of the prefrontal cortex (PFC), as 2 regions whose hyperactivation is implicated in OCD [12]. Generally, precortical areas play a fundamental role in coordinating neural integration and enabling cognitive processes. In particular, the OFC is involved in several cognitive functions, including emotional processing and decision-making strategies that are shaped by emotions, since the OFC operates as a reward system involved in reward-and-punishment-related behaviors [13]. Similarly, the ACC has been implicated in affective processing [14].

While various portions of the PFC have been associated with the generation of obsessive thoughts [15], a positive correlation exists between OFC hyperactivation and anxiety levels [16]. Remarkably, prescribed therapies for OCD are associated with a reduction in OFC activity [17]. Neuroimaging studies of OCD also report hyperactivation of the ACC [18]: this abnormal activity has been attributed to error-related negativity, a phenomenon frequently observed in OCD patients [19].

From a neurobiological perspective, the prevailing theory of OCD posits dysfunction in the cortico-striatal-thalamo-cortical (CSTC) circuit, which is organized as follows: precortical neurons, mainly from the OFC and ACC, project to the striatum, where spiny projection neurons transmit information to the thalamus via the substantia nigra pars reticulata. The thalamus then relays the processed signal back to the frontal cortex [20]. The CSTC circuit includes a direct pathway, which promotes excitation, and an indirect pathway, which exerts inhibition [14]. A chemical imbalance between these pathways results in overactivity of the direct one and, consequently, of the entire CSTC circuit, contributing to obsessive–compulsive symptoms [4]. Among the various neurotransmitters associated with this imbalance, glutamate [21], dopamine [22], and serotonin [23] have been the most extensively investigated. Specifically, the serotonin hypothesis is supported by evidence indicating that the activity of the OFC is strongly modulated by the raphe nucleus, a structure densely populated with serotonergic neurons [24]. Additionally, some researchers proposed a neuroepigenetic model of OCD, suggesting that trauma directly influences the expression of genes encoding neurotransmitter systems. This effect appears to be mediated by epigenetic modifications, which may contribute to OCD onset or exacerbate symptoms. Consistent with this framework, Bey et al. [25] reported that the effects of childhood trauma on OCD patients appear to be mediated through DNA hypermethylation of genes implicated in its pathogenesis, including the oxytocin receptor gene. Recently, Seo et al. [26] provided evidence that epigenetic alterations induced by early-life trauma, specifically methylation of the hypothalamic–pituitary–adrenal (HPA) axis-related gene FKBP5, contribute to OCD susceptibility. Remarkably, Kuhlman et al. [27] suggested that exposure to SLEs during childhood can disrupt the HPA axis, the brain system responsible for regulating physiological and psychological stress responses via cortisol secretion. Several studies demonstrate that cortisol levels are higher in patients with OCD [28] and PTSD [29]. Such dysregulation in stress reactivity has been shown to increase vulnerability to the development of various psychopathologies, including OCD [30].

Considering the current limitations of first-line therapies in achieving optimal treatment outcomes, further investigation is warranted into how alternative interventions may modulate the activity of the OFC and ACC while minimizing the adverse effects associated with pharmacological treatments. Given the substantial role trauma plays in OCD, addressing such experiences could represent an effective therapeutic strategy. In this context, we propose an integrated approach combining eye movement desensitization and reprocessing (EMDR) with noninvasive brain stimulation (NIBS) for the treatment of OCD. EMDR is a psychological intervention developed for PTSD that facilitates the reprocessing of traumatic memories [31]. Neuroimaging studies show that EMDR targets the OFC and ACC, promoting subcortical regulation through cortical neuromodulation [32]. Moreover, NIBS are well-established techniques employed to stimulate the brain without penetrating the tissue in awake humans and animals, resulting in positive therapeutic outcomes [33]. In addition, NIBS offers the advantage of selectively modulating the activity of targeted cortical areas, enabling a level of precision unattainable with either pharmacological or psychotherapeutic interventions.

Recent research is examining the combination of NIBS with classical psychotherapies; however, to our best knowledge, this is the first proposal in the literature to integrate EMDR with NIBS for the treatment of OCD. Thus, the aim of this manuscript is to justify the implementation of an EMDR–NIBS approach by elucidating the neurophysiological mechanisms underlying EMDR's efficacy and outlining the potential benefits of combining EMDR with NIBS to achieve comprehensive symptom improvement.

EMDR: Neural Mechanisms and Evidence from Current Interventions in OCD

EMDR represents a promising approach for OCD, particularly when the disorder stems from a history of trauma. During therapy, patients receive alternating bilateral stimulation (ABS), producing right–left sensory stimulation [34]. In EMDR's theoretical framework, traumatic memories stem from dysfunction in the adaptive information processing system, which normally integrates negative and positive experiences. When this fails, traumatic memories are formed [35].

Several neuroscientific studies have approached EMDR to elucidate the neural mechanisms behind its effectiveness, as it has proven effective across various psychopathologies [36]. Grounded in studies investigating the pathophysiology of PTSD, the focus has been centered on cortical areas implicated in the dysregulation of the amygdala, a subcortical brain structure whose hyperactivation in response to fearful stimuli critically contributes to PTSD symptom severity [37]. Of note, neuroimaging studies have produced evidence implicating dysfunctions in the ventromedial PFC, OFC, and ACC in the etiology of PTSD [38]. In this regard, an early study by Levin et al. [39] demonstrated that EMDR induces activation of both the ACC and the left frontal lobe. Recently, Pierce and Black [32] reached similar conclusions, suggesting that the ABS modulates brain regions involved in emotional regulation, including the ACC, the medial PFC, and the OFC, which in turn regulate abnormal subcortical activity. Notably, Corrigan [40] proposed that EMDR facilitates the transfer of memories associated with negative emotions from paralimbic circuits to cortical memory networks, where their affective charge is reduced. According to this hypothesis, the

beginning of an EMDR session is marked by activation of the ventral–affective subdivision of the ACC, which subsequently shifts to increased activation of the dorsal–cognitive subdivision, thereby restoring reciprocal inhibition between these portions that had been disrupted by the traumatic experience.

The OFC has also been implicated in EMDR mechanisms: Pagani et al. [41] firstly showed a modulation of the OFC during EMDR. Similarly, Amano and Toichi [42] found that in PTSD patients, OFC exhibited strong activation at the beginning of an EMDR session, which progressively lessened throughout the session, culminating in decreased activation following treatment. Pagani et al. [43] also observed heightened activation of the OFC and ACC at the start of therapy, which was followed by increased activation in temporo-occipital regions posttreatment. The activation of temporo-occipital areas may reflect enhanced cognitive processing and integration abilities, potentially resulting from OFC-mediated modulation of traumatic images facilitated by EMDR.

Thus, EMDR appears to modulate the OFC and ACC, 2 regions critically implicated in OCD pathophysiology. The convergence of these neurocircuitries, along with evidence linking trauma to OCD development, supports considering EMDR as an intervention for OCD. Notably, some clinicians have already explored its application: Böhm and Voderholzer [44] reported an average 60% reduction in OCD symptoms across 3 patients following an EMDR protocol (the posttreatment Yale–Brown Obsessive Compulsive Scale [Y-BOCS] score was 10.67 ± 1.25). Similarly, Marr [45] observed a decrease in symptoms of 81% in 4 patients, maintained at 6-month follow-up. Marsden [46] also reported symptom improvement across 3 patients treated with EMDR. At posttreatment, both patients with available data scored below the Y-BOCS diagnostic cutoff of 16, indicating nonclinical symptom levels. At the 6-month follow-up, all 3 patients continued to exhibit subclinical obsessive–compulsive symptoms. Likewise, Keenan et al. [47] documented a clinical case series of 8 patients with refractory OCD, showing that Y-BOCS scores decreased markedly during EMDR treatment, and these improvements were maintained at 6-month follow-up.

Furthermore, EMDR may be effective for individuals who developed OCD in childhood following traumatic experiences: Cusimano [48] reported improvement in a 13-year-old patient, diagnosed at age 11 after an unspecified trauma. The Children's Y-BOCS score decreased from 17 before the treatment, indicating moderate symptoms, to 3 after 15 EMDR sessions. One potential explanation for EMDR's effectiveness in trauma-related OCD is that it may reduce arousal by regulating cortisol levels. In a study assessing salivary cortisol concentrations in PTSD patients, Gerardi et al. [49] reported that cortisol levels decreased in treatment responders following EMDR. Nonetheless, this hypothesis remains theoretical, as no study has ever measured cortisol trends in traumatized OCD patients undergoing EMDR.

Crucially, some studies have investigated the integration of EMDR with different approaches for OCD: Mazzoni et al. [50] combined EMDR with ERP in 3 treatment-resistant OCD patients, showing reductions in symptoms. Consistent with this perspective, Potik [51] coupled EMDR with psychodynamic psychotherapy and cognitive therapy in treating a 27-year-old man who developed OCD after exposure to traumatic images, resulting in a reduction in symptoms from a Y-BOCS score of 19 to 3, maintained at 1-year follow-up. The same research group also combined EMDR with SSRI medication (i.e., fluoxetine) for a client with comorbid OCD and schizoaffective disorder, leading to substantial obsessive–compulsive symptom reductions maintained at posttreatment and at 6-month and 1-year follow-up [52].

Moreover, a randomized controlled trial (RCT) comparing citalopram with EMDR is of particular interest, as it found that the latter treatment resulted in a greater reduction in obsessive–compulsive symptoms [53]. At present, this remains the only RCT in the literature to directly evaluate EMDR against SSRIs, and the initial results appear encouraging, as the average Y-BOCS score after 12 weeks of EMDR treatment was 13.6 ± 5.48, falling within the subclinical range.

Furthermore, Marsden et al. [54] conducted an RCT comparing EMDR with ERP. Among the 29 recruited participants, 17 completed all the 16 EMDR sessions. In the EMDR group, the pretreatment Y-BOCS score was 25.07 ± 6.23; posttreatment scores decreased to the moderate range (18.72 ± 8.01), maintained at the 6-month follow-up (18.24 ± 8.59). Interestingly, the primary outcome indicated no significant difference in efficacy between EMDR and ERP at posttreatment ($d = -0.24$, $P = 0.38$) or at 6 months ($d = -0.03$, $P = 0.90$). These results suggest that EMDR may be as effective as ERP. Nonetheless, the Y-BOCS scores after treatment did not reach the subclinical range. Furthermore, only 6 patients in the EMDR group fell within the nonclinically relevant Y-BOCS range posttreatment. Conversely, Sarichloo et al. [55] reported in medication-resistant OCD patients with trauma histories that although both ERP alone and EMDR + ERP reduced obsessive–compulsive symptoms, the combined treatment yielded greater symptom reduction and a higher rate of treatment completion, providing evidence that integrated interventions may offer superior therapeutic benefit for severely impaired patients. Overall, these results suggest that the high success rates reported in case studies are not consistently replicated when EMDR alone is applied to larger samples. Table 1 summarizes the demographic characteristics and Y-BOCS scores for each study included in this review.

The uncertainty surrounding these findings, stemming from methodological biases, small sample sizes, and the limited number of studies, warrants consideration of how EMDR's effectiveness might be supported. Although a substantial overlap between the neural targets of EMDR and the regions implicated in OCD exists, current evidence suggests that its therapeutic impact remains unclear [56]. However, given the encouraging outcomes of EMDR combined with other therapies, future research should focus on designing multimodal approaches to enhance its efficacy. In this context, leveraging recent advances in NIBS alongside established psychotherapies may help overcome treatment resistance in OCD.

NIBS: Principles and Current Evidence in the Treatment of OCD

In light of the discussion presented in this manuscript, we propose the use of NIBS to augment EMDR's efficacy. Among the available NIBS modalities, two have attracted particular attention: transcranial magnetic stimulation (TMS) [57] and transcranial electrical stimulation (tES), encompassing transcranial direct current stimulation (tDCS), transcranial alternating current stimulation, and transcranial random noise stimulation [58]. NIBS exert their effects by influencing neuroplasticity in the cortical regions where they are applied. Neuroplasticity refers to the capacity of the brain to modify its structural and

Table 1. Demographic characteristics and Y-BOCS scores of the studies employing EMDR to treat OCD

Reference	Sample size (*n*) (M; F)	Age, mean (SD)	Study design	Adjunctive therapy	Y-BOCS total score			
					Pre-treatment; mean (SD)	Post-treatment; mean (SD)	6 Months follow-up; mean (SD)	1 Year follow-up; mean (SD)
Böhm and Voderholzer (2010) [44]	3 (2; 1)	28.33 (5.13)	Case series	None	27.67 (10.40)	10.67 (1.53)	9.00 (1.00)	n/a
Marr (2012) [45]	4 (4; 0)	24.25 (3.34)	Case series	None	35.30	8.50	7.50	n/a
Marsden (2016) [46]	3 (0; 3)	36.00 (16.37)	Case series	None	17.00 (7.00)	n/a	4.67 (4.16)	n/a
Keenan et al. (2019) [47]	8 (4; 4)	40.00	Case series	None	23.50	12.50	n/a	n/a
Cusimano (2018) [48]	1 (1; 0)	13.00	Case report	None	17.00	3.00	n/a	n/a
Mazzoni et al. (2017) [50]	3 (1; 2)	38.67 (6.85)	Case series	ERP	29.33 (4.51)	9.67 (8.50)	n/a	n/a
Potik (2017) [51]	1 (1; 0)	27.00	Case report	PT; CT	19.00	3.00	3.00	3.00
Potik et al. (2020) [52]	1 (1; 0)	28.00	Case report	SSRIs	19.00	7.00	4.00	4.00
Nazari et al. (2011) [53]	30 (20; 10)	n/a	RCT	None	24.83 (5.35)	13.60 (5.48)	n/a	n/a
Marsden et al. (2018) [54]	29 (12; 17)	30.90 (9.79)	RCT	None	25.07 (6.23)	18.72 (8.01)	18.24 (8.59)	n/a
Sarichloo et al. (2020) [55]	26 (8; 18)	n/a	RCT	ERP; SSRIs	24.26 (5.00)	5.50 (1.96)	n/a	n/a

Y-BOCS, Yale–Brown Obsessive Compulsive Scale; n/a, not available; ERP, exposure and response prevention; RCT, randomized controlled trial; CT, cognitive therapy; PT, psychodynamic therapy; SSRIs, selective serotonin reuptake inhibitors

functional organization in response to learning, adaptation, and environmental factors [59]. While TMS induces electrical currents in the targeted brain regions through magnetic fields delivered by a coil, tES modulates neuroplasticity through an anodal–excitatory and cathodal–inhibitory low electrical current, with the former inducing long-term potentiation (LTP) and the latter inducing long-term depression (LTD) synaptic mechanisms. While LTP strengthens synaptic connections, LTD weakens them [60].

TMS has been extensively validated for the treatment of psychiatric disorders, including major depressive disorder (MDD) [61], substance use disorder (SUD) [62], and generalized anxiety disorder (GAD) [63], among others. tES is likewise regarded as a promising intervention for a range of psychopathologies, including GAD [64], SUD [65], and anorexia nervosa [66]. Recently, investigations into the neurobiological principles of NIBS have demonstrated their capacity to induce synaptic plasticity in patients undergoing such interventions [67]. Notably, some scholars have reported that psychotherapy can also promote neuroplasticity, contributing to clinical improvement [68]. Having analyzed OCD pathophysiology, it is evident that patients exhibit aberrant activity in the OFC. Remarkably, neuromodulation of the OFC could directly normalize its hyperactivity, thereby leading to the regulation of the CSTC circuit [69]. Given that EMDR has not been shown to modulate the entire CSTC circuit, and that NIBS can target CSTC oscillations to promote cognitive flexibility [70], we propose using tDCS to specifically neuromodulate the OFC. Compared to TMS, tDCS is more cost-effective, can be easily implemented in a clinical setting, and can efficiently target the OFC without causing excessive discomfort, a result that is often difficult to achieve with TMS [71]. Additionally, although TMS has been shown to alleviate OCD symptoms, evidence indicates that it does not improve cognitive functioning [72]. By contrast, tDCS has been shown to yield improvements in cognitive performance among OCD patients [73]. Furthermore, Liu et al. [74] reported that OCD patients exhibit dysconnectivity between the ACC and the fronto-limbic network. EMDR's limited effectiveness in large OCD cohorts may reflect inadequate neuromodulation of frontal cortical networks, including OFC and ACC, resulting in suboptimal connectivity changes. In this context, tDCS could prove crucial, potentially enhancing frontal network activity and strengthening therapeutic outcomes. Notably, Lu et al. [75] demonstrated that tDCS is able to increase functional connectivity between the ACC and the frontal areas, a phenomenon associated with enhancement of cognitive functions. Of note, a dual-cathode tDCS design, targeting the OFC and pre-supplementary motor area (pre-SMA), has been shown to enhance connectivity in frontal networks in treatment-resistant OCD patients, resulting in 20.75% ± 8.70% symptom reduction after 20 sessions [76]. Interestingly, a cathode-left OFC and anode-right occipital montage produced a significant reduction in symptoms at the 1-month follow-up among patients receiving 2 mA tDCS ($d = 1.01$, $P = 0.004$) [73]. In addition, cathodal stimulation of the supraorbital region yields promising therapeutic outcomes in patients with severe OCD: Najafi et al. [77] reported that in 42 treatment-resistant OCD patients with a baseline Y-BOCS score of 29 ± 2.77, 15 tDCS sessions reduced the mean score to a subclinical range (10.6 ± 5.78).

Positive effects have also been reported when tDCS targets the dorsolateral PFC, where average Y-BOCS scores decreased from the moderate range (22.62 ± 4.81) to subclinical levels (15.50 ± 6.76) in a sample of 10 patients [78], and the pre-SMA, whose stimulation is usually associated with symptom improvement [79].

Finally, a recent meta-analysis by Moshfeghinia et al. [80] provided evidence supporting tDCS for OCD, demonstrating reductions in Y-BOCS scores and minimal adverse events following stimulation. Nevertheless, the findings show substantial heterogeneity: Ibrahim et al. [81] highlighted that both the efficacy and standardized methodology of tDCS for OCD remain unestablished, although underscoring that tDCS holds particular promise as an adjunctive therapy. Similarly, Rapinesi et al. [82] concluded that, despite data heterogeneity, NIBS, especially cathodal-tDCS over the OFC, represents a valuable adjunctive intervention for treatment-refractory OCD. In this regard, some clinicians have begun exploring integrated approaches that combine NIBS with psychotherapy [83]. Psychotherapy-induced neuroplasticity may be further enhanced through NIBS, with such integrative interventions potentially improving long-term therapeutic outcomes [84]. Given EMDR's positive outcomes in OCD and tDCS's ability to induce neuroplasticity in OFC, we propose combining these interventions in an integrated approach.

Integrating EMDR with tDCS for the Treatment of OCD

Prior work has explored tDCS as an adjunctive intervention to enhance treatment outcomes. For instance, tDCS combined with CT yielded symptom improvement in a sample of treatment-resistant MDD patients [85]. Similarly, tDCS paired with CT led to symptom amelioration in the treatment of binge eating disorder [86] and PTSD [87]. In this regard, a systematic review examining the efficacy of tDCS for PTSD [88] included 2 studies that applied NIBS protocols to patients with PTSD and comorbid OCD, both of which reported reductions in overall symptom severity [89,90]. These results indicate that NIBS can neuromodulate cortical areas involved in PTSD pathophysiology, producing symptom reduction and better management of trauma-related distress. Notably, the greatest symptom improvements were observed in studies pairing NIBS with a standard therapeutic intervention.

Emerging data indicate that tDCS represents a promising augmentation strategy for OCD when coupled with SSRIs [91] and ERP [92]. Arguably, the same result could be achieved by employing tDCS to enhance EMDR's efficacy. In this regard, one study proposed the integration of EMDR with multifocal tDCS, specifically for the treatment of fibromyalgia [93]. The rationale for this intervention is that EMDR's effectiveness could be enhanced through multifocal tDCS-induced neuroplasticity. However, to date, no clinical data are available.

These findings support the hypothesis that tDCS may enhance EMDR's therapeutic benefits: integrating the 2 approaches could mutually strengthen their effects and help address current therapeutic challenges. To our knowledge, no published studies have yet examined the integration of tDCS and EMDR for the treatment of OCD. However, we speculate that EMDR-induced neuroplasticity, and in particular the cortical–subcortical molecular phenomena that allow for trauma processing [94], could be enhanced by synaptic mechanisms triggered by tDCS, in a neurophysiological phenomenon termed metaplasticity, i.e., the modification of plasticity by the prior activity of the same neural network [95]. Some scholars have hypothesized that NIBS may have the potential to promote metaplasticity when combined with psychotherapy

[96]. Specifically, given the evidence of OFC hyperactivation in OCD patients and the apparent insufficiency of EMDR to modulate this region, we propose applying LTD-inducing cathodal tDCS over the OFC during EMDR sessions to boost its effects. Indeed, it remains uncertain whether EMDR alone fully normalizes CSTC circuit dynamics across patients: the addition of cathodal tDCS may help reinforce or prolong EMDR's modulatory effects, potentially enhancing therapeutic outcomes.

EMDR is structured around 8 phases: client history, preparation, assessment, desensitization, installation, body scan, closure, and reassessment. We suggest that tDCS could enhance metaplastic mechanisms during the desensitization and installation phases. During desensitization, traumatic memories are processed through ABS and focused attention on distressing thoughts, with negative feelings emerging. In the installation phase, these are replaced by positive perceptions [97]. Therefore, the patient's ability to engage with distressing memories and consolidate positive cognitions could be enhanced by tDCS-induced therapeutic plasticity, which would not impede the proper conduct of the EMDR session and might improve its efficacy during a critical phase of the process. From a neuronal perspective, intercellular signaling molecules have been shown to induce long-lasting modifications in synaptic plasticity across multiple brain regions [98], supporting the rationale for concurrent combined approaches. In addition, metaplastic phenomena can persist for days or even weeks [96], emphasizing the potential of integrating psychotherapy with NIBS to achieve sustained therapeutic effects.

Notwithstanding the need to establish the feasibility of this approach in real-life clinical settings, including ensuring that one intervention does not counteract or interfere with the mechanisms of the other, and that OCD patients can tolerate brain stimulation during EMDR sessions, some trials have already tested tDCS administered alongside psychotherapy sessions. For instance, the combination of tDCS with virtual reality exposure in a sample of 12 PTSD patients resulted in a marked reduction in symptom severity [87]. Similarly, tDCS applied during metacognitive therapy decreases anxiety, depressive symptoms, and repetitive negative thinking among 30 MDD and 42 high-anxiety patients [99]. Building on these studies, we propose employing EMDR to modulate the OFC and ACC in trauma-related circuits, while leveraging tDCS-induced OFC neuroplasticity, thereby promoting enhanced therapeutic outcomes following metaplastic adaptations.

Conclusion

Integrating EMDR with tDCS could enhance treatment outcomes by modulating ACC and OFC activity while facilitating trauma processing. This proposal represents a potential alternative to conventional therapeutic approaches for OCD. Furthermore, by relying on well-established techniques such as NIBS, this approach could also advance our understanding of OCD pathophysiology. Specifically, tDCS allows for the modulation of specific cortical regions implicated in obsessive–compulsive symptoms and provides insights into their activity, a result not attainable through pharmacological or psychotherapeutic interventions. However, empirical investigation into the combined use of EMDR and tDCS for OCD is currently absent. Given the speculative nature of this proposal, systematic research is required to evaluate its feasibility, refine methodological parameters, and assess preliminary therapeutic effects through pilot studies before advancing to larger clinical samples, with careful monitoring for possible adverse events. Future studies should prioritize the recruitment of treatment-resistant OCD patients, as they may offer particularly valuable insights into the potential benefits of this integrated approach. Moreover, to test the hypothesis that EMDR may be especially effective in individuals whose OCD emerged following a traumatic experience, research should include this subgroup and compare them with patients whose OCD developed in the absence of trauma. Such a design would help determine whether trauma-related treatment-resistant individuals represent the population most likely to respond to the proposed intervention. In addition, rigorous clinical trials should incorporate real versus sham tDCS conditions to clarify the added value of combining NIBS with EMDR. Primary outcome measures should include reductions in obsessive–compulsive symptoms, assessed using the Y-BOCS, along with broader improvements in quality of life and decision-making. Collectively, such findings could provide the empirical foundation necessary to support the future implementation of an integrated EMDR-tDCS approach for OCD.

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Integrating EMDR with tDCS in Obsessive–Compulsive Disorder: An Opinion Paper

Niccolò Pozzato, Daniele Saccenti, Andrea Stefano Moro, Walter Sapuppo, Jacopo Lamanna, and Mattia Ferro

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Current evidence indicates that approximately 25% of patients with obsessive–compulsive disorder (OCD) fail to respond to conventional therapies. Although trauma exerts a substantial role in the etiology of OCD, eye movement desensitization and reprocessing (EMDR) has shown limited evidence in treating such condition, highlighting the need for novel interventions. As a result, recent clinical advances suggest that combined approaches integrating multiple psychological interventions could be effective in yielding positive therapeutic outcomes. Concurrently, neuroimaging studies indicate that EMDR modulates neuronal activity within the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC), both implicated in OCD pathophysiology. Building on this evidence, noninvasive brain stimulation techniques, including transcranial direct current stimulation (tDCS), have demonstrated promise for OCD treatment by modulating OFC neuroplasticity. Hence, this opinion paper proposes a therapeutic strategy combining EMDR with tDCS to concomitantly target trauma-related psychological processes and rehabilitate neural dysfunction in the OFC and ACC. Such a novel therapeutic approach may enhance treatment effectiveness and offer an option for treatment-resistant OCD.