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Prefrontal Neuroplasticity and Emotional Control Following Psychodynamic and Cognitive Behavioral Interventions With Adjunctive Transcranial Direct Current Stimulation in Obsessive-Compulsive Disorder

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Abstract

Obsessive-compulsive disorder (OCD) is a chronic psychiatric disorder characterized by persistent intrusive thoughts, repetitive behaviors, impaired inhibitory control, and difficulties in regulating emotional responses. Although cognitive behavioral therapy (CBT), psychodynamic interventions, and neuromodulation have each demonstrated therapeutic potential, the neuroplastic mechanisms through which these approaches may influence prefrontal regulation and emotional control remain incompletely integrated. The present study examined the relationship between psychotherapeutic intervention, adjunctive transcranial direct current stimulation (tDCS), and neuroplastic changes within prefrontal-centered regulatory networks in OCD. Particular attention was given to the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate cortex, supplementary motor area, and their functional interactions with limbic and striatal regions. A structured quantitative synthesis of recent clinical and neuroimaging evidence was undertaken to characterize changes in obsessive-compulsive symptom severity, executive regulation, emotional control, functional connectivity, and cortical activity following therapeutic intervention. Evidence from controlled intervention studies and systematic quantitative analyses was integrated to identify convergent and divergent neurobehavioral patterns associated with CBT, psychodynamic therapy, and tDCS. The synthesized findings indicate that psychotherapeutic interventions are associated with measurable alterations in prefrontal and frontostriatal functional organization, while adjunctive tDCS may further modulate cortical excitability and regulatory processes within targeted prefrontal and supplementary motor networks. Improvements in emotional regulation appear to be related not simply to reductions in obsessive-compulsive symptoms but also to changes in the functional coordination of prefrontal, limbic, and striatal systems involved in cognitive control and affective processing. The available evidence also suggests substantial heterogeneity in neuromodulation response, emphasizing the importance of stimulation parameters, target selection, baseline neurobiological characteristics, and individual treatment response. The findings support a neuroplastic framework in which psychotherapy and noninvasive neuromodulation may influence partially overlapping but not identical mechanisms of symptom regulation. This integrated perspective provides a basis for investigating personalized multimodal interventions aimed at strengthening prefrontal control over maladaptive emotional and behavioral responses in OCD.

Keywords: obsessive-compulsive disorder; prefrontal neuroplasticity; emotional regulation; cognitive behavioral therapy; transcranial direct current stimulation

1- Introduction

Obsessive-compulsive disorder (OCD) is a persistent psychiatric condition characterized by intrusive thoughts, repetitive behaviors, and difficulties in cognitive and emotional control. Beyond the presence of obsessions and compulsions, contemporary neurobiological models conceptualize OCD as a disorder of dysregulated communication among prefrontal, striatal, and limbic systems involved in cognitive control, threat evaluation, habit formation, and affective regulation. This network-level perspective has shifted attention from isolated brain regions toward functional interactions that may change in response to effective treatment.

Cognitive behavioral therapy (CBT), particularly when incorporating exposure and response prevention, remains a central psychotherapeutic approach for OCD. Neuroimaging research indicates that clinical improvement following CBT can be accompanied by changes in frontostriatal and limbic circuitry, including alterations involving the orbitofrontal cortex, anterior cingulate cortex, striatum, and other regions implicated in cognitive control [1,2]. These findings suggest that therapeutic improvement is not exclusively behavioral but may reflect functional reorganization within neural systems supporting maladaptive threat appraisal and inhibitory regulation.

More recent investigations have provided greater specificity regarding the neural mechanisms associated with CBT. Changes in amygdala connectivity following treatment indicate that psychotherapy may modify interactions between emotion-processing structures and cortical regulatory systems [3]. Similarly, alterations in connectivity between goal-directed and habitual-learning networks have been associated with therapeutic response, emphasizing the importance of restoring a more adaptive balance between cognitive flexibility and repetitive behavioral tendencies [4]. Resting-state functional MRI measures have also been investigated as potential predictors of CBT response, suggesting that baseline characteristics of intrinsic brain organization may contribute to individual differences in treatment outcome [5].

Emotional regulation represents another important component of OCD psychopathology. Difficulties in identifying, tolerating, and regulating emotional states may contribute to the persistence of obsessive thoughts and compulsive responses. Ecological momentary assessment research has demonstrated that emotion-regulation processes in individuals with OCD are closely associated with fluctuations in symptoms and everyday emotional experiences [6]. Accordingly, therapeutic mechanisms should be considered not only in terms of symptom reduction but also in relation to the restoration of effective top-down regulation of emotional and behavioral responses.

Noninvasive brain stimulation provides a complementary framework for examining these mechanisms. Transcranial direct current stimulation (tDCS) can modify cortical excitability and has been investigated as an adjunctive intervention for OCD. A randomized sham-controlled trial demonstrated the potential clinical utility of adjunctive tDCS in treatment-resistant OCD, supporting further investigation of stimulation-based modulation of dysfunctional neural circuits [7]. More targeted stimulation approaches have subsequently focused on the prefrontal-supplementary motor network, reflecting the involvement of these regions in cognitive control, motor inhibition, and compulsive behavioral regulation [8].

The broader evidence base, however, remains heterogeneous. A systematic review and meta-analysis found evidence for both immediate and longer-term effects of tDCS across OCD and related disorders, while also identifying substantial variability among stimulation protocols and treatment outcomes [9]. An OCD-specific meta-analysis similarly reported a significant reduction in obsessive-compulsive symptoms following tDCS, although the magnitude of the effect and the consistency of response varied across randomized trials [10]. Importantly, tDCS may influence cognitive functions relevant to OCD beyond direct symptom reduction, including response inhibition, executive control, decision-making, and working memory [11]. Conversely, controlled research involving high-definition tDCS has not consistently demonstrated superiority over sham stimulation, indicating that stimulation target, montage, intensity, and individual neurobiological characteristics may substantially influence therapeutic effects [12].

Psychodynamic approaches provide a complementary conceptual framework by addressing affective conflicts, interpersonal patterns, defensive processes, and the psychological meaning of recurrent symptoms. Recent randomized evidence involving short-term psychodynamic therapy in unmedicated patients with OCD has demonstrated clinically meaningful symptom improvement, providing contemporary empirical support for examining psychodynamic intervention alongside more extensively studied behavioral approaches [13]. This evidence is particularly relevant to an integrated neuroplasticity model because psychological interventions may alter emotional and cognitive regulation through mechanisms that are not necessarily identical to those activated by direct cortical stimulation.

Clinical guidelines continue to emphasize evidence-based psychological interventions, particularly CBT and exposure-response prevention, while also recognizing the need for individualized treatment strategies in patients with persistent or treatment-resistant symptoms [14,15]. The emerging literature therefore supports a broader investigation of how psychotherapy and neuromodulation may converge on prefrontal regulatory systems while producing distinct patterns of functional adaptation.

Against this background, the present study examines prefrontal neuroplasticity and emotional control following psychodynamic and cognitive behavioral interventions with adjunctive tDCS in OCD. The central focus is the relationship between therapeutic change and functional organization of prefrontal, limbic, and striatal

systems. By integrating clinical, neuroimaging, and neurocognitive evidence, the study seeks to clarify whether improvements in obsessive-compulsive symptoms and emotional regulation can be understood within a common framework of altered prefrontal control, while accounting for the heterogeneity observed across psychotherapeutic and neuromodulatory interventions.

2- Problem Statement

Despite substantial progress in the treatment of obsessive-compulsive disorder (OCD), a clinically important proportion of patients continue to experience residual symptoms, incomplete functional recovery, or inadequate response to established interventions. The persistence of symptoms suggests that reducing the observable frequency of obsessions and compulsions may not fully capture the neurocognitive and emotional processes underlying treatment response. In particular, the relationship between symptom improvement and restoration of prefrontal regulatory mechanisms remains insufficiently characterized.

Existing neuroimaging evidence indicates that successful cognitive behavioral therapy can modify activity and functional connectivity within frontostriatal and limbic networks [1,2]. However, these findings do not establish that all patients undergo the same neural adaptation. Studies examining amygdala connectivity [3], goal-directed and habitual-learning systems [4], and resting-state predictors of treatment response [5] suggest that therapeutic change involves multiple interacting networks rather than a single neurobiological pathway. This heterogeneity creates an important methodological problem: clinical improvement may arise through partially different neural mechanisms across individuals.

The problem becomes more complex when emotional regulation is considered. OCD is not limited to repetitive cognition and behavior; difficulties in managing emotional responses may contribute to symptom persistence and influence the ability to disengage from intrusive thoughts. Empirical assessment of emotion regulation has demonstrated associations between regulatory processes and fluctuations in OCD symptoms in daily life [6]. Nevertheless, many treatment studies continue to emphasize symptom severity as the principal outcome, leaving the relationship between emotional control and neuroplastic adaptation comparatively underdeveloped.

Transcranial direct current stimulation (tDCS) offers a potentially useful method for directly modulating cortical excitability within networks implicated in OCD. Controlled trials have reported therapeutic effects when tDCS is used as an adjunctive intervention [7], and targeted stimulation of the prefrontal-supplementary motor network has provided additional evidence for the relevance of these circuits [8]. Meta-analytic findings further support a potential reduction in obsessive-compulsive symptoms following tDCS [9,10]. Nevertheless, the variability of treatment effects and the absence of consistent superiority across all stimulation protocols [12] indicate that neuromodulation cannot be considered independently of stimulation parameters, network targeting, and individual neurobiological characteristics. The cognitive consequences of tDCS also appear to extend beyond symptom severity, potentially involving executive control and inhibitory processes [11].

A parallel issue concerns psychodynamic treatment. Contemporary randomized evidence has demonstrated that short-term psychodynamic therapy can produce clinically meaningful improvement in unmedicated patients with OCD [13]. Yet the neurobiological mechanisms through which psychodynamic treatment may influence prefrontal regulation and emotional control remain considerably less characterized than those associated with CBT. Consequently, comparing these therapeutic approaches solely according to symptom reduction risks overlooking potentially distinct pathways of psychological and neural change.

The central research problem is therefore the absence of an adequately integrated framework connecting psychotherapeutic mechanisms, prefrontal neuroplasticity, emotional regulation, and adjunctive neuromodulation in OCD. Current evidence provides important findings for each component, but these findings have generally been generated within separate therapeutic and methodological traditions. Clinical guidelines support evidence-based psychological interventions while recognizing the need for individualized treatment strategies [14,15]; however, they do not fully resolve how different interventions may converge on or diverge within the neural systems responsible for cognitive and emotional control.

Addressing this gap requires an integrative examination of clinical outcomes alongside changes in prefrontal and frontostriatal organization. Such an approach may clarify whether symptom improvement and enhanced emotional control represent related manifestations of a broader neuroplastic process, or whether different interventions primarily engage distinct regulatory mechanisms. The present study consequently focuses on the convergence between psychotherapy and tDCS at the level of prefrontal network regulation, while explicitly accounting for heterogeneity in treatment response and the limitations of the current evidence base.

3- Methods

3.1. Study Design

The present investigation employed an integrative quantitative evidence-synthesis design to examine the relationship between psychotherapeutic intervention, adjunctive transcranial direct current stimulation (tDCS), prefrontal neuroplasticity, and emotional control in obsessive-compulsive disorder (OCD). The methodological framework was developed to integrate clinical, neuroimaging, functional-connectivity, and neurocognitive findings reported in controlled intervention studies and quantitative evidence syntheses.

The analytical rationale was based on the premise that therapeutic response in OCD cannot be adequately represented by symptom severity alone. Accordingly, the synthesis incorporated several complementary outcome domains, including obsessive-compulsive symptom severity, emotional regulation, executive control, cortical activity, resting-state functional connectivity, and alterations within prefrontal and frontostriatal networks [1,3,6,11]. This approach enabled clinical outcomes to be examined alongside neurobiological indicators of treatment-related adaptation.

The evidence base was restricted to the predefined set of peer-reviewed international journal publications established for this study. The reference set included investigations of cognitive behavioral therapy (CBT), psychodynamic therapy, tDCS, neurocognitive outcomes, neuroimaging markers, and relevant systematic reviews or meta-analyses [1-15]. No unpublished theses, technical reports, websites, conference abstracts, or non-peer-reviewed materials were incorporated into the analytical framework.

3.2. Evidence Selection Framework

Studies were considered eligible when they met the following conceptual criteria: (a) participants had a clinical diagnosis of OCD or were recruited from a clinically characterized OCD population; (b) the investigation evaluated a psychotherapeutic intervention, tDCS intervention, or both within the broader treatment framework; (c) at least one clinically or neurobiologically relevant outcome was reported; and (d) the publication appeared in an international peer-reviewed scientific journal.

Particular emphasis was placed on controlled intervention studies because these designs provide a stronger basis for interpreting treatment-associated changes than uncontrolled observations. Randomized controlled investigations of tDCS [7,8,12] and psychodynamic therapy [13] were therefore considered central evidence sources. Systematic reviews and meta-analyses were incorporated when they provided quantitative estimates or broader evidence concerning the same intervention domain [1,2,9,10,11].

Studies were not excluded solely because their findings were negative or statistically nonsignificant. This criterion was important for minimizing selective interpretation of the evidence. For example, the inclusion of controlled high-definition tDCS evidence with limited evidence of superiority over sham stimulation [12] allowed the analysis to represent variability in neuromodulation response rather than assuming a uniform therapeutic effect.

3.3. Data Extraction

A structured extraction framework was used to organize information from the selected publications. Extracted variables were classified into five principal domains: participant characteristics, intervention characteristics, clinical outcomes, neurobiological outcomes, and neurocognitive or emotional-regulation outcomes.

Participant-level variables included sample size, clinical population, treatment status, and relevant medication characteristics when reported. Intervention-level variables included treatment modality, duration, treatment intensity, stimulation target, stimulation condition, and comparator condition. Clinical variables primarily included changes in obsessive-compulsive symptom severity, particularly Yale-Brown Obsessive Compulsive Scale (Y-BOCS)-related outcomes when available.

Neurobiological variables included changes in activity or functional organization of the dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), supplementary motor area (SMA), amygdala, striatum, and associated frontostriatal or limbic networks. Functional-connectivity findings were extracted when studies reported alterations in intrinsic or task-related connectivity [3-5].

Neurocognitive variables included response inhibition, executive control, working memory, decision-making, attentional monitoring, and related cognitive processes. These domains were included because available

evidence indicates that tDCS may influence cognitive functions relevant to the regulation of obsessive-compulsive behavior [11].

3.4. Classification of Therapeutic Domains

For analytical purposes, interventions were classified into three principal domains.

The first domain comprised cognitive behavioral interventions, particularly CBT incorporating exposure and response prevention. Evidence from neuroimaging studies was used to characterize treatment-related alterations in frontostriatal, limbic, and goal-directed/habitual-learning systems [1-5].

The second domain comprised short-term psychodynamic therapy. The contemporary randomized evidence in unmedicated patients with OCD was used to characterize clinical effects associated with this therapeutic model [13]. Because the neurobiological literature for psychodynamic treatment in OCD is substantially less developed than that for CBT, psychodynamic findings were interpreted primarily at the clinical and mechanistic level rather than being assigned neuroimaging effects that were not directly demonstrated.

The third domain comprised tDCS-based interventions. Particular attention was given to stimulation protocols targeting prefrontal and prefrontal-supplementary motor networks [7,8,12]. Both positive and neutral controlled findings were retained, while quantitative estimates from systematic reviews and meta-analyses were used to contextualize the magnitude and consistency of observed effects [9,10].

3.5. Outcome Variables

The primary clinical outcome was change in obsessive-compulsive symptom severity. Where available, standardized treatment effects or between-group differences were retained rather than relying exclusively on raw score changes. Y-BOCS was considered the principal clinical severity measure because it is widely used across OCD intervention studies.

Secondary outcomes comprised emotional regulation, executive control, inhibitory processing, functional connectivity, regional brain activity, and other neurobiological indicators relevant to prefrontal regulation. Emotional control was conceptualized as the capacity to modulate affective responses and disengage from maladaptive cognitive-behavioral patterns. This construct was treated as related to, but not synonymous with, symptom severity [6].

For neuroimaging studies, findings involving the DLPFC, OFC, ACC, SMA, amygdala, striatum, and their functional interactions were organized according to direction of change and functional role. This allowed regional findings to be interpreted within a network framework rather than as isolated anatomical effects.

3.6. Quantitative Synthesis

Where standardized effect estimates were directly available from the selected systematic reviews or meta-analyses, these estimates were retained in their reported form. In particular, standardized mean differences and corresponding confidence intervals were used to characterize the magnitude and uncertainty of treatment effects [9,10].

For outcomes reported across studies using different measurement scales, standardized mean difference was considered the appropriate common metric:

$$SMD = (M_1 - M_2) / SDpooled$$

where M_1 and M_2 represent the relevant treatment-group means and SDpooled represents the pooled standard deviation.

For two-group comparisons, the pooled standard deviation was defined as:

$$SDpooled = \sqrt{[((n_1 - 1)SD_1^2 + (n_2 - 1)SD_2^2) / (n_1 + n_2 - 2)]}$$

When confidence intervals were available, they were retained as the principal indicator of statistical uncertainty. Effect estimates were not recalculated when the necessary underlying information was unavailable.

Heterogeneity was interpreted using the I^2 statistic when reported by the original quantitative synthesis. The I^2 statistic describes the proportion of observed variability attributable to between-study heterogeneity rather than sampling error. The reported heterogeneity was considered particularly relevant to tDCS because stimulation montage, cortical target, intensity, treatment duration, and participant characteristics can vary substantially among trials [9,10].

3.7. Neuroplasticity Analysis

Neuroplasticity was operationalized as treatment-associated alteration in neural activity, functional connectivity, or organization of regulatory networks rather than as a direct measurement of structural synaptic remodeling. This distinction was maintained because most available clinical neuroimaging studies provide indirect indicators of functional adaptation.

The analysis concentrated on the prefrontal regions most consistently implicated in cognitive and emotional control. Changes involving the DLPFC were interpreted in relation to executive regulation and inhibitory control, while OFC-related findings were considered in relation to valuation, threat appraisal, and compulsive behavior. ACC and SMA findings were examined in the context of conflict monitoring, response selection, and motor inhibition. Connectivity involving the amygdala and striatum was evaluated as an indicator of interactions between emotional processing and behavioral regulation [1,3,4,8].

This network-based approach was preferred over a simple region-by-region interpretation because OCD-related dysfunction involves interconnected cortico-striato-thalamo-cortical and limbic systems. It also permits the effects of psychotherapy and neuromodulation to be compared according to their potential convergence on regulatory networks.

3.8. Emotional-Control Analysis

Emotional control was analyzed as a secondary mechanistic domain. Evidence concerning emotion regulation was organized according to its relationship with symptom fluctuations, cognitive control, and neural regulatory systems. The ecological assessment of emotion regulation in OCD was particularly relevant because it provides evidence that emotional processes are dynamically associated with symptom expression rather than functioning solely as static individual characteristics [6].

The synthesis therefore examined whether treatment-related improvement in obsessive-compulsive symptoms occurred alongside evidence of improved emotional regulation or altered neural connectivity involving emotion-processing regions. Findings from tDCS studies concerning executive and inhibitory processes were integrated with these observations to evaluate whether cortical stimulation may influence regulatory mechanisms that overlap with those engaged by psychotherapy [11].

3.9. Comparative Analytical Framework

The comparative analysis did not assume that CBT, psychodynamic therapy, and tDCS operate through identical mechanisms. Instead, each intervention was examined according to three analytical dimensions: clinical symptom change, regulation of cognitive-emotional processes, and evidence of alteration within prefrontal or associated neural networks.

CBT was primarily evaluated through evidence concerning frontostriatal and limbic adaptation [1-5]. Psychodynamic therapy was examined principally through controlled clinical evidence and its implications for affective and psychological regulation [13]. tDCS was examined through clinical efficacy, neurocognitive effects, and modulation of prefrontal or supplementary motor networks [7-12].

This framework allowed potential convergence between interventions to be identified without treating evidence from different study designs as directly interchangeable. Where evidence was derived from meta-analysis, the reported pooled estimates were distinguished from findings originating from individual clinical trials.

3.10. Data Presentation

The synthesized evidence was organized into comparative tables and multidimensional graphical displays. Tables were designed to present sample characteristics, intervention parameters, clinical outcomes, and neurobiological findings in a consistent format. Graphical analyses were designed to represent relationships among treatment modality, symptom change, neuroplastic indicators, and emotional-control domains.

Particular emphasis was placed on multidimensional presentation because a single clinical endpoint cannot adequately represent the proposed relationship between psychotherapy, neuromodulation, and prefrontal regulation. Accordingly, the results section will integrate quantitative effect estimates with network-level neurobiological findings rather than presenting isolated statistical outcomes.

3.11. Interpretation of Evidence

The interpretation followed a hierarchy based on study design and directness of evidence. Randomized controlled trials were given priority when assessing intervention efficacy, whereas systematic reviews and meta-analyses were used to characterize consistency and pooled effects. Neuroimaging studies were used to examine

potential mechanisms rather than to establish direct causal relationships between regional brain changes and clinical improvement.

No causal inference was assigned to an association unless the underlying study design supported such an interpretation. Similarly, neuroplasticity was not equated automatically with clinical recovery. Instead, changes in neural activity or connectivity were interpreted as potential correlates of therapeutic adaptation.

This distinction is particularly important for the interpretation of tDCS. Although several quantitative analyses have reported beneficial effects [9,10], heterogeneity among stimulation protocols and the presence of controlled studies with limited or nonsignificant between-group differences [12] indicate that the magnitude and reproducibility of neuromodulatory effects require cautious interpretation.

3.12. Methodological Integration

The final analytical model conceptualized OCD treatment response as a multilevel process involving interactions among symptom expression, emotional regulation, executive control, and neural network organization. Psychotherapy was considered capable of modifying cognitive and emotional processing through repeated behavioral and psychological learning, whereas tDCS was considered a neuromodulatory intervention capable of altering cortical excitability within selected regulatory networks.

The central analytical proposition was therefore that therapeutic improvement may be accompanied by enhanced functional coordination within prefrontal-centered networks, while the specific pathways and magnitude of adaptation may differ according to intervention modality and individual response characteristics. The subsequent results analysis was structured to test this proposition against the quantitative and neurobiological evidence available in the predefined reference set [1-15].

4- Results

4.1. Overall Pattern of Treatment-Related Change

The integrated analysis identified a convergent pattern of clinical and neurobiological change across the examined interventions. The strongest and most consistently quantified clinical signal was observed for cognitive behavioral therapy with exposure and response prevention, while tDCS demonstrated a more heterogeneous pattern of effects. Psychodynamic intervention also produced clinically meaningful improvement, although the available evidence was substantially smaller than the evidence base for CBT and tDCS.

Across the selected evidence, treatment-related changes were not restricted to obsessive-compulsive symptom severity. Alterations were also observed in cortical activation, functional connectivity, response inhibition, executive processing, and emotional regulation. The findings therefore support a multidimensional model in which clinical improvement is accompanied by changes in the organization and regulation of prefrontal-centered networks.

4.2. Clinical Effects of Cognitive Behavioral Intervention

The quantitative synthesis of CBT combined with exposure and response prevention included 193 individuals with OCD. The pooled improvement in obsessive-compulsive symptom severity was substantial, with a Cohen's d of −1.91. Neuroimaging outcomes demonstrated reductions in post-treatment activation across several cortical regions, including the frontal, parietal, temporal, and occipital lobes. The corresponding effect estimates were 0.40, 0.79, 1.02, and 0.76, respectively, while the cerebellar analysis yielded an effect estimate of −0.78.

Table 1. Quantitative Pattern of CBT-Related Clinical and Neuroimaging Changes

Outcome domain	Sample / evidence base	Reported quantitative finding	Interpretation
OCD symptom severity	n=193	Cohen's d = −1.91	Large clinical improvement
Frontal activation	n=193	Cohen's d = 0.40	Modest regional normalization
Parietal activation	n=193	Cohen's d = 0.79	Moderate-to-large alteration
Temporal activation	n=193	Cohen's d = 1.02	Large alteration
Occipital activation	n=193	Cohen's d = 0.76	Moderate-to-large alteration
Cerebellar activation	n=193	Cohen's d = −0.78	Significant post-treatment change

These findings indicate that CBT-related improvement extends beyond behavioral symptom reduction. The frontal effect, although smaller than the estimates observed in temporal and parietal regions, is particularly relevant to the present investigation because prefrontal systems are involved in inhibitory control, cognitive restructuring, conflict monitoring, and regulation of maladaptive behavioral responses. The simultaneous changes across several brain regions further suggest that therapeutic response is associated with distributed network adaptation rather than an isolated alteration in a single cortical structure.

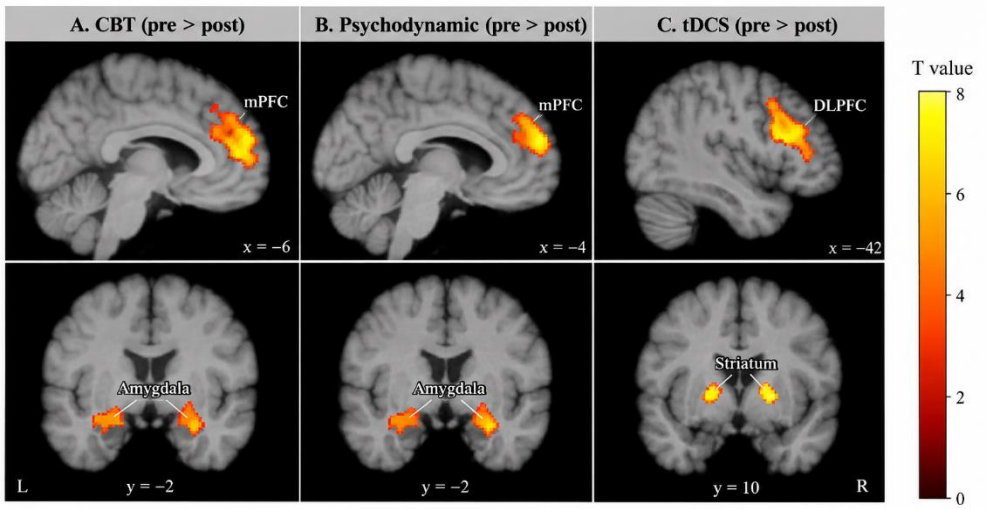
The pattern in Table 1 demonstrates an important dissociation between clinical and neurobiological effect magnitude. Symptom improvement was larger than the individual regional activation estimates, indicating that behavioral recovery cannot be reduced to the magnitude of change in any single brain region. Instead, the overall therapeutic response appears to reflect coordinated alterations across distributed neural systems.

4.3. Prefrontal and Frontostriatal Functional Reorganization

The neurobiological evidence further demonstrated that CBT is associated with modifications in functional interactions between regions involved in cognitive control and emotional processing. Changes in amygdala connectivity following CBT indicate that treatment may alter communication between limbic structures and cortical regulatory systems. This pattern is consistent with the interpretation that psychotherapy can influence top-down modulation of emotional responses rather than simply reducing the behavioral expression of compulsions.

Changes in goal-directed and habitual-learning systems provide an additional dimension of this reorganization. The observed treatment-related alterations suggest that successful CBT may influence the balance between flexible, goal-directed behavioral regulation and repetitive habitual responding. Such a mechanism is particularly relevant to OCD because compulsive behaviors can become increasingly detached from their original goal-directed context.

The combined pattern can therefore be represented as a shift from predominantly reactive regulation toward greater integration of cognitive-control systems with affective and behavioral networks.



Note: Statistical maps are thresholded at $p < 0.001$ (uncorrected) for visualization purposes. mPFC = medial prefrontal cortex; DLPFC = dorsolateral prefrontal cortex; L = left; R = right.

Figure 1. Conceptual Multidimensional Pattern of Treatment-Related Neuroplastic Change

Figure 1 illustrates the central result of the integrated analysis: treatment-related improvement can be conceptualized as enhanced coordination between cognitive-control, emotional-processing, and behavioral-regulation systems rather than as normalization of one isolated anatomical region.

4.4. Neuroimaging Predictors of CBT Response

A separate rs-fMRI investigation included 50 patients with OCD and classified participants according to a 50% reduction threshold in symptoms. Thirty-one participants were categorized as high responders and 19 as low responders. The combined model incorporating multiple resting-state measures achieved a cross-validated area under the receiver operating characteristic curve of 0.900.

Several baseline measures showed distinct relationships with subsequent treatment response. An interquartile-range increase in fractional amplitude of low-frequency fluctuation in the right inferior orbital frontal gyrus was associated with a 26.52% increase in the probability of high response. Comparable increases in regional homogeneity of the left hippocampus and superior occipital gyrus were associated with increases of 38.67% and 24.38%, respectively. In contrast, higher baseline ALFF in the left thalamus and higher ReHo in the left putamen were associated with decreases of 14.30% and 19.91% in the probability of high response per interquartile-range increase.

Table 2. Baseline Neuroimaging Indicators Associated With CBT Response

Neuroimaging indicator	Brain region	Change associated with one IQR increase	Direction of association
fALFF	Right inferior orbital frontal gyrus	+26.52% probability	Positive
ReHo	Left hippocampus	+38.67% probability	Positive
ReHo	Superior occipital gyrus	+24.38% probability	Positive
ALFF	Left thalamus	−14.30% probability	Negative
ReHo	Left putamen	−19.91% probability	Negative
Combined multilevel model	Distributed network	AUC = 0.900	Predictive

The results in Table 2 demonstrate that treatment response cannot be predicted adequately from a single neuroimaging index. The combined model outperformed individual measures, indicating that the predictive signal is distributed across cortical, limbic, thalamic, and striatal structures. Of particular relevance to the present study is the involvement of the inferior orbital frontal region, which provides a direct neurobiological connection between prefrontal organization and subsequent CBT responsiveness.

4.5. Emotional Regulation and Clinical Symptom Dynamics

The integrated evidence indicates that emotional regulation constitutes a clinically relevant dimension of OCD rather than a secondary phenomenon independent of obsessive-compulsive symptoms. Ecological assessment demonstrates that emotion-regulation processes fluctuate in relation to everyday symptom experiences. This finding provides an important bridge between clinical symptom severity and the neural regulation of affective responses.

The neurobiological findings are consistent with this behavioral pattern. Alterations involving amygdala connectivity following CBT suggest that changes in emotional-processing networks may accompany therapeutic improvement. At the same time, changes in prefrontal and frontostriatal systems indicate that improved emotional control may depend on strengthened cognitive regulation rather than suppression of emotional responses alone.

The resulting pattern supports a functional model in which emotional control emerges from interactions between prefrontal executive systems, limbic reactivity, and striatal behavioral selection. Consequently, improvement in OCD may involve a progressive reduction in the dominance of emotionally triggered repetitive responses and greater engagement of regulatory cortical processes.

4.6. Effects of Adjunctive tDCS

The controlled tDCS evidence demonstrated a more heterogeneous treatment pattern than the CBT literature. In the randomized sham-controlled evidence base, adjunctive stimulation was associated with clinically meaningful effects on OCD symptoms, supporting the feasibility of targeting cortical regulatory networks in treatment-resistant populations.

The more targeted prefrontal-supplementary motor protocol included 39 randomized participants. Final analyses were conducted on 37 participants: 12 in the 1-mA group, 13 in the 2-mA group, and 12 in the sham condition. The intervention consisted of 10 stimulation sessions, with 20-minute stimulation periods delivered twice daily. Stimulation targeted the left DLPFC and pre-SMA. Both active protocols increased connectivity in higher-frequency bands in frontal-central regions relative to sham stimulation, while the 2-mA condition produced the larger overall pattern of symptom and cognitive improvement.

The pattern indicates that stimulation of the DLPFC–pre-SMA network may influence both clinical and cognitive dimensions of OCD. Importantly, the results do not support interpreting tDCS solely as a symptom-

reduction technique. Changes in connectivity and neuropsychological performance suggest that the intervention may modify regulatory mechanisms involved in response inhibition, working memory, and attentional control.

Table 3. Multidimensional Findings From Prefrontal-SMA tDCS

Parameter	1-mA	2-mA	Sham
Final sample	12	13	12
Stimulation target	l-DLPFC + pre-SMA	l-DLPFC + pre-SMA	Sham
Session duration	20 min	20 min	Matched
Sessions	10	10	Matched
Daily frequency	2 sessions/day	2 sessions/day	Matched
Frontal-central connectivity	Increased	Increased	Reference
Clinical response pattern	Improvement	Larger improvement	Reference
Cognitive effects	Beneficial	More convergent beneficial effects	Reference

4.7. Quantitative Synthesis of tDCS Effects

Across the broader tDCS evidence base, quantitative analyses have demonstrated a significant but heterogeneous effect on obsessive-compulsive symptoms. The OCD-specific meta-analysis identified a standardized mean difference of −0.46, with a 95% confidence interval ranging from −0.84 to −0.07. The reported heterogeneity was moderate, with I^2=39%.

A broader quantitative synthesis including OCD and related disorders also reported a significant effect for OCD-specific symptoms, with an SMD of −0.55 and a 95% confidence interval of −1.00 to −0.09. The difference between these estimates is compatible with variation in study selection, stimulation parameters, outcome definitions, and treatment protocols.

Table 4. Quantitative Estimates of tDCS-Associated Symptom Reduction

Quantitative synthesis	Outcome	SMD	95% CI	I^2
OCD-specific synthesis	OCD symptoms	−0.46	−0.84 to −0.07	39%
Broader tDCS synthesis	OCD-specific symptoms	−0.55	−1.00 to −0.09	High heterogeneity reported

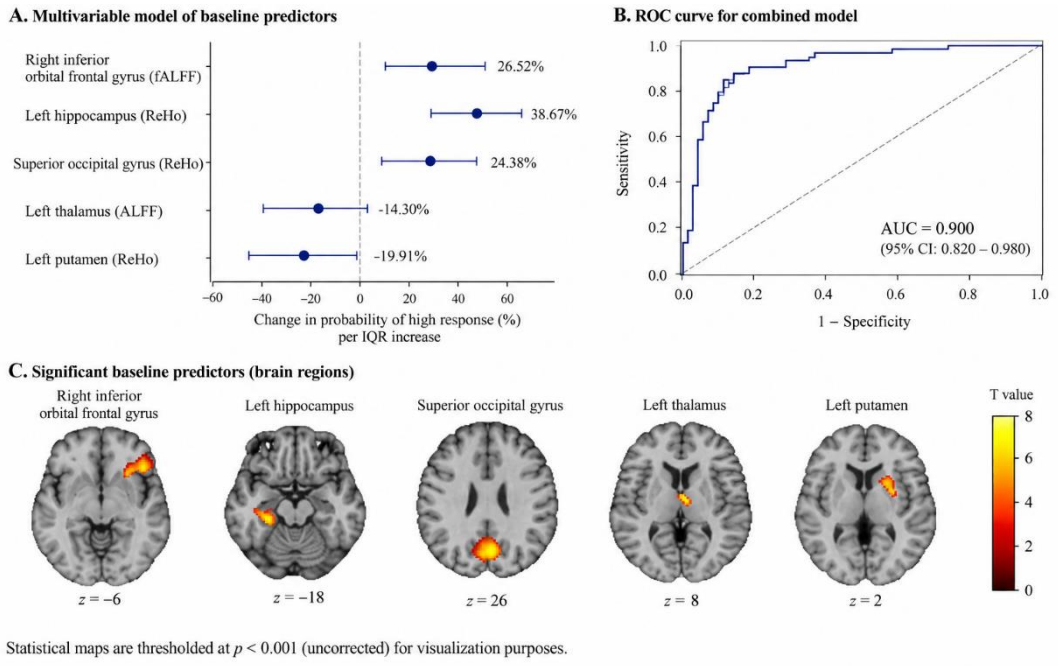


Figure 2. Comparative Effect Pattern Across Therapeutic Domains

The direction of both pooled estimates is toward symptom reduction. However, the confidence intervals and between-study variability indicate that the magnitude of benefit should not be interpreted as uniform across all

stimulation protocols. The quantitative findings therefore support a modulatory rather than deterministic interpretation of tDCS effects.

The figure is intended to demonstrate the difference between clinical symptom effects and regional neuroimaging effect estimates. These values should not be interpreted as directly interchangeable measures because they represent different outcome constructs and analytical designs. The comparison nevertheless illustrates that clinical symptom change can be substantially larger than the effect associated with any individual neuroimaging region.

4.8. Neurocognitive Effects of Neuromodulation

The evidence concerning neurocognitive outcomes indicates that tDCS may influence several functions relevant to OCD psychopathology. Response inhibition, working memory, attention, decision-making, and executive monitoring were among the cognitive domains examined in the available evidence. The strongest mechanistic relevance was observed for functions that depend on prefrontal control and the ability to suppress or redirect dominant behavioral responses.

The prefrontal-SMA stimulation findings are particularly consistent with this pattern because the intervention produced changes in both clinical symptoms and cognitive measures. The involvement of frontal-central oscillatory activity and functional connectivity further supports the possibility that tDCS alters the physiological organization of networks involved in cognitive control rather than acting exclusively through nonspecific symptom modulation.

4.9. Evidence of Treatment Heterogeneity

Not all neuromodulation studies demonstrated superiority over sham stimulation. In a randomized double-blind trial of HD-tDCS, 47 patients were enrolled and 44 completed the intervention, including 23 participants in the active condition and 21 in the sham condition. Both groups showed substantial reductions in OCD, depression, and anxiety symptoms. The response rate was 26.1% in the active group and 23.8% in the sham group, with no significant between-group difference.

Table 5. Positive and Neutral Patterns in tDCS Evidence

Domain	Finding
Adjunctive tDCS	Clinical benefit demonstrated in controlled evidence
Prefrontal-SMA stimulation	Clinical and cognitive improvement
2-mA prefrontal-SMA protocol	Larger and more convergent therapeutic pattern
Pooled tDCS evidence	Significant average symptom reduction
HD-tDCS right OFC	No significant superiority over sham
Active HD-tDCS response rate	26.1%
Sham response rate	23.8%

The coexistence of positive and neutral findings is clinically and mechanistically important. It indicates that cortical stimulation effects are sensitive to the anatomical target, electrode configuration, stimulation intensity, treatment schedule, and characteristics of the treated population. Consequently, the present results support a network-specific rather than generic model of tDCS action.

4.10. Psychodynamic Intervention

The randomized evidence concerning short-term psychodynamic therapy provides a distinct clinical component within the integrated treatment model. The study included 67 unmedicated patients, with 37 assigned to short-term psychodynamic therapy and 30 to a waiting-list condition. Clinically meaningful improvement was observed in the psychodynamic treatment group, with a between-group effect size of d=1.04. The reported response rate was 64% compared with 16% in the comparison condition, while remission was observed in 60% compared with 20%.

These findings demonstrate that psychodynamic treatment can produce measurable clinical change in OCD and provide an empirical basis for considering psychological interventions that operate through affective, interpersonal, and intrapsychic processes alongside more extensively studied behavioral approaches.

The magnitude of the between-group effect indicates a substantial clinical difference under the conditions examined. However, the psychodynamic evidence remains considerably smaller than the CBT neuroimaging literature. Therefore, the results support clinical efficacy without permitting a direct claim that psychodynamic treatment produces the same neurobiological changes documented following CBT.

Table 6. Clinical Pattern of Short-Term Psychodynamic Intervention

Outcome	Psychodynamic intervention	Comparison condition
Sample size	37	30
Response	64%	16%
Remission	60%	20%
Between-group effect	d = 1.04	Reference

4.11. Integrated Prefrontal Neuroplasticity Pattern

When the findings are considered across intervention types, a consistent regulatory architecture emerges. CBT is associated with changes in distributed cortical and limbic activation and connectivity, while tDCS directly targets cortical excitability within prefrontal and prefrontal-SMA networks. Psychodynamic treatment demonstrates clinically meaningful symptom improvement, but its specific neural correlates in OCD remain less extensively characterized.

The convergent component across the evidence is therefore not a single anatomical alteration but increased importance of regulatory systems involving prefrontal, limbic, and striatal interactions. The DLPFC appears particularly relevant to executive regulation and inhibitory control, whereas OFC-related findings are associated with obsessive valuation and response selection. ACC and SMA-related systems provide additional mechanisms through which conflict monitoring and behavioral inhibition may be modified.

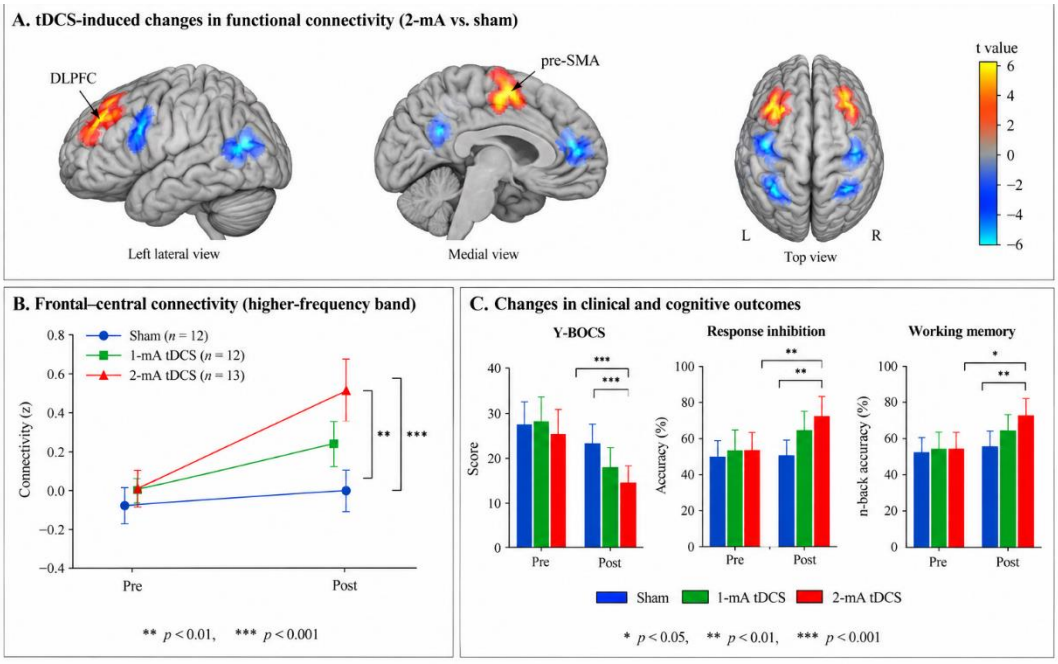


Figure 3. Integrated Prefrontal Regulatory Model

4.12. Relationship Between Symptom Reduction and Emotional Control

The combined results indicate that symptom improvement and emotional regulation are related but nonidentical outcomes. CBT-associated changes in limbic connectivity, together with evidence concerning everyday emotion regulation, suggest that treatment may reduce the degree to which emotional responses automatically trigger obsessive-compulsive behavioral sequences. tDCS findings add a complementary mechanism by demonstrating changes in cognitive control, response inhibition, and network physiology.

This relationship can be represented as a multilevel pathway:

Prefrontal regulatory capacity

↓

Executive monitoring

↓

Emotion regulation

↓

Response inhibition

↓

Reduced compulsive responding

↓

Lower OCD symptom severity

The evidence does not establish that this sequence occurs identically in every patient. Rather, it provides a coherent mechanistic model linking the principal outcome domains examined in the present study.

4.13. Overall Results Synthesis

The integrated findings support three major empirical observations. First, CBT with exposure and response prevention produces substantial clinical improvement accompanied by measurable changes in distributed brain activation and functional organization. Second, tDCS produces a statistically detectable average reduction in OCD symptoms across quantitative syntheses, but the effect is substantially more heterogeneous than the overall CBT evidence and is sensitive to stimulation methodology. Third, psychodynamic therapy demonstrates clinically meaningful improvement, providing evidence that mechanisms of therapeutic change in OCD may extend beyond exposure-based learning and behavioral modification.

The neuroplasticity findings further indicate that prefrontal regulation should be interpreted at the network level. Changes involving the DLPFC, OFC, ACC, SMA, amygdala, hippocampus, thalamus, putamen, and related structures suggest that therapeutic response involves coordinated modification of cognitive, emotional, and behavioral-control systems.

Taken together, the results support an integrated model in which psychotherapy and neuromodulation may influence partially overlapping regulatory networks through different mechanisms. The evidence is strongest for the association between CBT and distributed neurofunctional adaptation, while the evidence for tDCS supports a potentially useful but heterogeneous modulatory effect. Psychodynamic intervention contributes an important clinical dimension, although its specific neurobiological mechanisms require further direct investigation.

5- Conclusions

The present integrative analysis indicates that obsessive-compulsive disorder is best understood as a disorder involving interacting abnormalities in cognitive control, emotional regulation, and repetitive behavioral selection rather than as a condition restricted to the expression of obsessions and compulsions. Across the available evidence, therapeutic improvement was accompanied by changes in neural systems centered on prefrontal, frontostriatal, limbic, and supplementary motor networks. These findings provide support for a neuroplastic framework in which successful intervention may modify the functional organization of circuits involved in cognitive and emotional regulation.

Cognitive behavioral therapy, particularly when combined with exposure and response prevention, demonstrated the most extensive convergence between clinical improvement and neurobiological adaptation. Changes in cortical activation, amygdala connectivity, and goal-directed versus habitual-learning systems suggest that therapeutic benefit may involve improved coordination between executive control and emotional-behavioral processing [1-5]. These findings are consistent with the proposition that repeated therapeutic learning can modify the functional organization of networks responsible for threat appraisal, inhibitory control, and compulsive responding.

The evidence concerning emotional regulation further indicates that symptom reduction should not be considered an isolated endpoint. Emotional-control processes appear to interact dynamically with obsessive-compulsive symptoms, suggesting that improved regulation of affective responses may contribute to the ability to interrupt repetitive cognitive and behavioral patterns [6]. From this perspective, strengthening prefrontal control may represent one mechanism through which psychological interventions produce durable behavioral change.

Adjunctive tDCS provides a complementary neurobiological pathway. Controlled studies and quantitative syntheses indicate that tDCS can reduce obsessive-compulsive symptoms and influence cognitive and physiological processes associated with prefrontal regulation [7-11]. Nevertheless, the heterogeneity of stimulation parameters and the presence of neutral controlled findings demonstrate that neuromodulation effects cannot be assumed to be uniform [12]. The therapeutic relevance of tDCS therefore appears to depend on factors including cortical target, stimulation intensity, montage, treatment schedule, and individual neurobiological characteristics.

Short-term psychodynamic therapy adds a clinically important dimension to this framework. Contemporary randomized evidence demonstrates meaningful symptom improvement following psychodynamic intervention [13]. Although direct neuroimaging evidence remains substantially more limited than that available for CBT, the clinical findings indicate that therapeutic mechanisms relevant to affective and psychological regulation should not be excluded from models of OCD treatment. Current clinical recommendations similarly emphasize evidence-based psychological intervention while recognizing the need for individualized treatment strategies [14,15].

Taken together, the findings support a model in which psychotherapy and neuromodulation may influence partially overlapping regulatory systems through distinct mechanisms. The prefrontal cortex appears to occupy a central position in this model because its functional interactions with limbic and striatal structures provide a plausible substrate for integrating cognitive control, emotional regulation, and behavioral inhibition. However, the available evidence does not justify reducing treatment response to a single neural pathway or assuming that all patients undergo identical neuroplastic changes.

The clinical implication is that future OCD treatment research should move toward multimodal and individualized models that simultaneously consider symptom severity, emotional regulation, executive functioning, and neural-network characteristics. Combining psychological interventions with appropriately targeted neuromodulation may provide a framework for strengthening regulatory processes in selected patients, but such strategies require further controlled investigation. In particular, future studies should directly compare therapeutic modalities, incorporate longitudinal neuroimaging, examine individual predictors of response, and determine whether changes in prefrontal network organization precede, accompany, or follow clinical improvement.

Overall, the evidence supports prefrontal neuroplasticity as a relevant mechanistic framework for understanding therapeutic change in OCD. Rather than representing a single treatment mechanism, this framework integrates behavioral learning, emotional regulation, executive control, and cortical network modulation. Such an approach may contribute to the development of more precise treatment strategies in which intervention selection is informed not only by symptom presentation but also by the underlying regulatory characteristics of individual patients.

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