



Neurocognitive Modulation of Obsessive-Compulsive Symptoms Through Sequential Integration of Intensive Short-Term Dynamic Psychotherapy, Cognitive Behavioral Therapy, and Transcranial Direct Current Stimulation

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Abstract

Background: Obsessive-compulsive disorder (OCD) is a persistent psychiatric condition characterized by intrusive thoughts, repetitive behaviors, impaired emotional regulation, and difficulties in cognitive control. Although cognitive behavioral therapy (CBT) is an established psychological intervention for OCD, a considerable proportion of patients experience incomplete symptom remission or residual emotional and cognitive difficulties. Intensive short-term dynamic psychotherapy (ISTDP) provides a complementary framework that focuses on maladaptive emotional processes, defensive patterns, and underlying affective conflicts. Transcranial direct current stimulation (tDCS), meanwhile, offers a non-invasive neuromodulatory approach capable of influencing cortical excitability and cognitive-emotional processing. The present study developed and evaluated a sequential therapeutic framework integrating ISTDP, CBT, and tDCS to examine its potential neurocognitive and clinical effects in individuals with OCD.

Methods: A structured evidence-based analytical framework was developed using quantitative findings reported in recent randomized controlled trials, systematic reviews, meta-analyses, and neuroimaging research. The framework examined changes in obsessive-compulsive symptom severity, emotional regulation, anxiety, depressive symptoms, and cognitive control following psychological and neuromodulatory interventions. Particular attention was given to the complementary therapeutic mechanisms associated with dynamic emotional processing, cognitive restructuring, exposure-based learning, and modulation of prefrontal neural networks. Comparative analyses were organized to identify convergent patterns across the three intervention domains and to determine clinically meaningful dimensions for an integrated treatment sequence.

Results: The evidence synthesis indicated that CBT produces consistent reductions in obsessive-compulsive symptoms, while psychodynamic interventions may contribute to improvements in emotional processing and underlying psychological organization. Evidence from tDCS studies further suggests that targeted neuromodulation can produce clinically relevant changes in OCD symptoms, with effects extending to broader anxiety and depressive dimensions in some populations. Neurocognitive findings indicate that frontoparietal and prefrontal systems are particularly relevant to cognitive control and compulsive symptom expression. The integrated framework therefore identifies complementary therapeutic targets across emotional processing, cognitive control, and cortical modulation.

Conclusion: Sequential integration of ISTDP, CBT, and tDCS provides a theoretically coherent framework for addressing multiple interacting mechanisms underlying OCD. Rather than treating obsessive-compulsive symptoms as an isolated behavioral phenomenon, the proposed model incorporates emotional regulation, cognitive control, and neurobiological modulation within a unified therapeutic pathway. This framework may provide a basis for future controlled clinical investigations examining personalized sequencing, treatment response, and neurocognitive mechanisms in OCD.

Keywords: Obsessive-Compulsive Disorder; Intensive Short-Term Dynamic Psychotherapy; Cognitive Behavioral Therapy; Transcranial Direct Current Stimulation; Neurocognitive Modulation

1- Introduction

Obsessive-compulsive disorder (OCD) is a chronic and clinically heterogeneous psychiatric disorder characterized by recurrent intrusive thoughts, repetitive behaviors, and persistent attempts to reduce distress associated with obsessions. Beyond overt symptoms, OCD is increasingly conceptualized as a disorder involving interactions among emotional dysregulation, maladaptive cognitive control, dysfunctional habit formation, and altered frontostriatal and frontoparietal network activity. These interacting mechanisms contribute to substantial functional impairment and may help explain why symptom reduction does not always correspond to complete psychological recovery [13,14].

Cognitive behavioral therapy (CBT), particularly exposure and response prevention-based approaches, represents one of the most extensively investigated psychological interventions for OCD. Evidence from routine clinical settings indicates that CBT can produce meaningful reductions in obsessive-compulsive symptoms, although treatment response varies considerably across patients [4]. A broader meta-analysis of randomized controlled trials has likewise demonstrated the effectiveness of psychological interventions for OCD while highlighting substantial heterogeneity in treatment response and study characteristics [5]. These findings support CBT as a central component of treatment but also indicate the need to investigate complementary mechanisms that may influence therapeutic outcomes.

One potentially complementary perspective is provided by intensive short-term dynamic psychotherapy (ISTDP). Unlike interventions that primarily target explicit cognitions and behavioral contingencies, ISTDP focuses on maladaptive emotional processes, defensive patterns, and difficulties in experiencing and regulating affect. Evidence from patients with anxiety disorders suggests that ISTDP may influence both symptom severity and deeper dimensions of psychological functioning [3]. More recent clinical work has also expanded the evidence base for short-term psychodynamic approaches in OCD, with randomized controlled evidence indicating clinically meaningful symptom improvement [1]. ISTDP has consequently become relevant to contemporary discussions of treatment-resistant and complex psychopathology, although its specific role in OCD remains substantially less investigated than that of CBT [2].

A second complementary pathway involves direct modulation of neural activity. Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulation technique capable of altering cortical excitability and potentially influencing cognitive and affective processes. Randomized controlled evidence has indicated that tDCS may provide additional clinical benefits when used alongside conventional treatment in individuals with OCD [6]. Systematic reviews and meta-analyses have subsequently reported evidence supporting its potential efficacy, while also emphasizing variability in stimulation protocols, cortical targets, treatment duration, and clinical response [7-9].

The relevance of tDCS extends beyond OCD-specific symptom reduction. Recent evidence suggests that neuromodulation may also influence anxiety and depressive symptoms across diagnostic categories, providing a potential bridge between OCD and commonly co-occurring affective symptoms [10,11]. This is particularly important because difficulties in emotion regulation have been consistently associated with obsessive-compulsive symptomatology and may represent a clinically meaningful mechanism linking emotional distress with compulsive behavior [12].

Neuroscientific evidence further provides a rationale for integrating psychological and neuromodulatory approaches. Neuroimaging research has identified convergent abnormalities within frontoparietal systems involved in cognitive control, decision-making, response inhibition, and the regulation of behavior in OCD [13]. Broader models of compulsivity similarly emphasize interactions among cortical control systems, habit-related circuitry, and motivational processes rather than attributing compulsive behavior to a single neural region [14].

Taken together, these findings suggest that CBT, ISTDP, and tDCS may address partially overlapping but distinguishable dimensions of OCD. CBT primarily targets maladaptive interpretations and compulsive behavioral patterns; ISTDP emphasizes emotional processing and defensive organization; and tDCS provides a method for modulating neural systems involved in cognitive-emotional control. The sequential integration of these approaches therefore represents a theoretically coherent framework for investigating whether different therapeutic mechanisms can be organized within a single treatment pathway.

The present article develops this framework with particular emphasis on neurocognitive modulation of obsessive-compulsive symptoms. The central premise is that sequential intervention may permit emotional processing to be addressed before intensive cognitive-behavioral modification and neuromodulation are used to reinforce cognitive control and symptom regulation. This conceptual integration provides a basis for examining

clinical outcomes alongside neurocognitive dimensions and for identifying measurable mechanisms that may contribute to individual differences in treatment response.

2- Problem Statement

Despite substantial progress in the psychological and neurobiological treatment of obsessive-compulsive disorder (OCD), an important methodological gap remains in the organization of complementary therapeutic mechanisms within a single treatment pathway. Existing evidence has largely examined cognitive behavioral therapy (CBT), psychodynamic interventions, and transcranial direct current stimulation (tDCS) as separate therapeutic strategies. CBT has demonstrated consistent efficacy in reducing obsessive-compulsive symptoms, while recent evidence has expanded support for short-term psychodynamic approaches. However, these approaches are generally evaluated independently, making it difficult to determine whether their distinct therapeutic mechanisms can be arranged sequentially to address different dimensions of OCD [1,4,5].

The limited evidence concerning intensive short-term dynamic psychotherapy (ISTDP) in OCD represents a particularly relevant gap. ISTDP emphasizes the identification and processing of maladaptive emotional experiences, defensive responses, and underlying affective conflicts. These processes may be clinically relevant in OCD because emotional distress and difficulties in emotion regulation can interact with intrusive thoughts and compulsive responses. Nevertheless, the empirical literature remains substantially smaller than the evidence base supporting CBT, and direct evidence concerning the integration of ISTDP with subsequent cognitive-behavioral intervention is limited [2,3,12].

A parallel limitation exists in the neuromodulation literature. tDCS has been investigated as a potential adjunctive intervention for OCD, including in randomized sham-controlled trials and subsequent meta-analytic research [6-9]. However, considerable heterogeneity exists across stimulation parameters, cortical targets, treatment schedules, and patient characteristics. Consequently, the available evidence does not establish a clear framework for determining when neuromodulation should be introduced relative to psychological treatment or whether its effects may be enhanced when preceded by targeted psychological interventions.

This issue is particularly relevant from a neurocognitive perspective. OCD involves disturbances in cognitive control, response inhibition, emotional regulation, and repetitive behavioral processes that are associated with alterations in distributed neural networks rather than a single dysfunctional mechanism [13,14]. At the same time, anxiety and depressive symptoms frequently constitute clinically relevant dimensions of the disorder, and evidence indicates that tDCS may influence these affective domains as well [10,11]. Therefore, an intervention model that addresses emotional processing, cognitive-behavioral mechanisms, and neural modulation in a coordinated sequence may provide a more comprehensive framework for understanding treatment-related change.

The central problem addressed in this study is consequently not simply whether ISTDP, CBT, or tDCS can reduce OCD symptoms independently. Rather, the research problem concerns whether these interventions can be conceptually organized according to their distinct therapeutic targets so that emotional processing, cognitive-behavioral modification, and neurocognitive modulation are addressed in a coherent sequence. Such an approach may also provide a more precise framework for examining changes in obsessive-compulsive symptoms together with anxiety, depressive symptoms, emotion regulation, and cognitive control.

Accordingly, the present study focuses on the development of a sequential integration framework combining ISTDP, CBT, and tDCS. The framework is intended to establish a clinically and neurobiologically grounded basis for evaluating whether complementary mechanisms can be integrated without treating the three interventions as interchangeable components. This distinction is important for future controlled investigations because it permits the temporal order of therapeutic mechanisms to become an explicit research variable rather than an incidental feature of treatment delivery.

3- Methods

3.1 Study Design

The present study employed a structured evidence-based analytical design to investigate the neurocognitive rationale for sequentially integrating intensive short-term dynamic psychotherapy (ISTDP), cognitive behavioral therapy (CBT), and transcranial direct current stimulation (tDCS) in obsessive-compulsive disorder (OCD).

Rather than treating the three interventions as interchangeable therapeutic modalities, the methodological framework considered them as sequential components targeting partially distinct clinical and neurocognitive mechanisms.

The analytical model was developed around four principal domains: obsessive-compulsive symptom severity, emotional regulation, affective symptoms, and cognitive control. These domains were selected because they represent clinically relevant dimensions of OCD and provide a conceptual bridge between psychological treatment and neuromodulation [10-14].

The evidence base was restricted to the 14 peer-reviewed international journal articles specified for the present study. These publications included randomized controlled trials, systematic reviews, meta-analyses, and neuroimaging research published between 2021 and 2026. No dissertations, technical reports, websites, conference materials, or non-peer-reviewed sources were included.

3.2 Evidence Identification and Selection

The evidence identification process focused on four predefined domains: psychodynamic psychotherapy, CBT, tDCS, and neurocognitive mechanisms associated with OCD. Studies were retained when their findings directly contributed to at least one component of the proposed sequential model.

For the psychodynamic component, evidence was derived from contemporary research examining short-term psychodynamic psychotherapy and ISTDP in OCD and anxiety-related conditions [1-3]. For the CBT component, systematic evidence concerning psychological treatment effectiveness and routine clinical implementation was considered [4,5]. The neuromodulation component was informed by randomized controlled evidence and quantitative syntheses examining tDCS in OCD [6-9]. Additional evidence concerning anxiety, depression, emotion regulation, and neural network mechanisms was incorporated from transdiagnostic and neuroimaging studies [10-14].

The selection procedure therefore followed a mechanism-oriented rather than purely diagnosis-oriented strategy. This approach allowed evidence from different study designs to be integrated according to the therapeutic mechanism addressed by each intervention.

3.3 Sequential Treatment Framework

The proposed therapeutic sequence consisted of three conceptually ordered stages.

The first stage involved ISTDP. Its purpose within the model was to address maladaptive emotional processing, defensive patterns, and difficulties in experiencing and regulating affect. This stage was positioned at the beginning of the sequence because emotional avoidance and defensive responses may interfere with the subsequent processing of distressing internal experiences [2,3,12].

The second stage involved CBT, with emphasis on cognitive restructuring and behavioral modification of obsessive-compulsive patterns. CBT was positioned after the psychodynamic component because the proposed model assumes that improved access to emotional experiences and greater awareness of defensive processes may facilitate engagement with cognitive and behavioral therapeutic tasks. The effectiveness of CBT for OCD is supported by evidence from routine clinical settings and randomized controlled trials [4,5].

The third stage involved tDCS as a neuromodulatory component. Its proposed function was to influence neural systems involved in cognitive control and emotional regulation after psychological interventions had targeted relevant psychological processes. Evidence from randomized trials and quantitative reviews indicates that tDCS can produce clinically relevant effects in OCD, although treatment parameters and response patterns vary across studies [6-9].

The sequence can therefore be represented as:

ISTDP → emotional processing and regulation → CBT → cognitive-behavioral modification → tDCS → neurocognitive modulation

This ordering represents the central methodological proposition of the study.

3.4 Outcome Domains

Five principal outcome domains were defined.

The first was obsessive-compulsive symptom severity. The Yale-Brown Obsessive Compulsive Scale (Y-BOCS) was treated as the primary clinical outcome because it is commonly used in OCD intervention research and permits quantitative comparison across studies.

The second domain was anxiety symptomatology. Anxiety was included because it represents a clinically relevant affective dimension that may interact with obsessive-compulsive symptoms and because neuromodulation studies have reported effects extending beyond OCD-specific outcomes [10,11].

The third domain was depressive symptomatology. Depression was incorporated as an associated affective outcome rather than as a primary diagnostic target. This distinction is important because depressive symptoms can influence perceived treatment burden and functional recovery while also representing a potential target of neuromodulation [11].

The fourth domain was emotion regulation. This variable was selected to represent a potential mechanism linking emotional processing with obsessive-compulsive symptom expression. Evidence indicates meaningful associations between emotion-regulation difficulties and OCD symptoms [12].

The fifth domain was cognitive control. This construct included processes such as response inhibition, executive control, and regulation of repetitive behavior. Its inclusion was based on evidence implicating frontoparietal and related neural systems in OCD [13,14].

3.5 Quantitative Evidence Extraction

Quantitative information was extracted from the eligible publications according to the outcome domain, intervention type, study design, sample characteristics, intervention parameters, comparator condition, and reported effect estimates.

For intervention studies, the principal extracted variables included sample size, intervention condition, comparator, treatment duration, primary outcome, and reported change in symptom severity. For systematic reviews and meta-analyses, pooled effect estimates, confidence intervals, heterogeneity indicators, and the number of included studies were considered where available.

The extracted evidence was organized into a comparative matrix to identify convergence and divergence among psychological and neuromodulatory interventions. Particular attention was given to the distinction between direct clinical outcomes and mechanistic variables.

3.6 Effect Size Interpretation

Where standardized effect estimates were available, they were interpreted according to their direction and magnitude rather than transformed into newly estimated patient-level outcomes. For standardized mean differences, the general form was:

$$SMD = (M_1 - M_2) / SD_{pooled}$$

where M_1 and M_2 represent the relevant group means and SD_{pooled} represents the pooled standard deviation.

For confidence intervals reported by the original studies, the published estimates were retained. No unreported patient-level values were reconstructed.

Where meta-analytic heterogeneity was available, the I^2 statistic was interpreted as an indicator of variability among study estimates. The general expression is:

$$I^2 = [(Q - df) / Q] \times 100$$

where Q represents Cochran's heterogeneity statistic and df represents the corresponding degrees of freedom.

These statistical indicators were used descriptively to compare the consistency of evidence across therapeutic domains.

3.7 Neurocognitive Integration

The neurocognitive component was developed by linking clinical outcomes with evidence concerning neural systems implicated in OCD. Contemporary neuroimaging research indicates convergent alterations within frontoparietal systems involved in cognitive control and behavioral regulation [13]. Broader neuroscientific models further characterize compulsivity as an emergent process involving interactions among cognitive-control systems, habit-related mechanisms, and motivational processes [14].

Accordingly, the proposed framework does not assume that tDCS directly produces a single predetermined neural change. Instead, neuromodulation is conceptualized as a potential mechanism for modifying cortical excitability and facilitating cognitive-emotional regulation within a broader therapeutic sequence. This

interpretation is consistent with the variability observed across tDCS studies and avoids attributing all clinical improvement to a single neurobiological pathway [6-9].

3.8 Comparative Analytical Strategy

The analysis was organized around comparisons among three intervention configurations: psychodynamic intervention, CBT-based intervention, and tDCS-supported intervention. The principal objective was not to assign a universal superiority ranking to the interventions but to identify complementary therapeutic functions.

The comparative framework considered whether each intervention primarily addressed emotional processing, cognitive-behavioral mechanisms, or neurocognitive modulation. Secondary attention was given to anxiety and depressive symptoms because these dimensions may influence overall clinical response [10,11].

A mechanism-to-outcome matrix was subsequently developed to examine the correspondence between intervention targets and measurable outcomes. This matrix provided the basis for the interpretation of the results and the construction of the proposed sequential model.

3.9 Methodological Considerations

Several methodological considerations were incorporated to reduce overinterpretation. First, the included studies differed in sample characteristics, treatment protocols, outcome measures, and follow-up periods. Second, tDCS protocols varied with respect to stimulation parameters and cortical targets [7-9]. Third, the evidence base for psychodynamic treatment in OCD remains smaller than the corresponding CBT literature [1-5]. Fourth, associations between neurocognitive mechanisms and clinical outcomes should not be interpreted as proof of causal mediation.

The sequential framework should therefore be regarded as an evidence-informed model that integrates findings across complementary research traditions. Its principal value is the explicit formulation of a testable treatment sequence that can subsequently be examined in prospective controlled clinical research.

3.10 Ethical and Reporting Considerations

Because the present analytical framework was based on previously published peer-reviewed evidence, no new participant recruitment or experimental intervention was undertaken within the evidence synthesis itself. The analysis retained the outcome definitions and quantitative estimates reported by the original investigations and did not introduce fabricated participant-level observations.

The methodological structure was designed to maximize reproducibility by maintaining a predefined evidence set, fixed outcome domains, explicit intervention stages, and transparent quantitative interpretation. These principles provide a foundation for evaluating the proposed integration of ISTDP, CBT, and tDCS while preserving the distinction between published empirical evidence and hypotheses requiring prospective validation.

4- Results

4.1 Overall Evidence Pattern

The integrated analysis identified three complementary therapeutic dimensions relevant to the management of obsessive-compulsive symptoms: emotional processing, cognitive-behavioral modification, and neurocognitive modulation. The evidence pattern indicated that these dimensions were associated with different but partially overlapping clinical outcomes.

The psychological treatment evidence demonstrated the most consistent reduction in core obsessive-compulsive symptoms for cognitive behavioral interventions. Psychodynamic interventions showed evidence of improvement in obsessive-compulsive symptomatology while additionally addressing emotional and psychological processes. The neuromodulation evidence indicated that tDCS could produce measurable changes in obsessive-compulsive symptoms and may also influence associated anxiety and depressive dimensions.

The resulting pattern supported the conceptual organization of treatment into sequential rather than interchangeable therapeutic stages.

Table 1. Evidence-Based Functional Mapping of the Three Intervention Components

Intervention component	Principal target	therapeutic	Main domain	clinical	Neurocognitive relevance	Position proposed	in
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					sequence
ISTDP	Emotional processing and defensive organization	Emotional distress and OCD symptoms	Affect regulation and emotional awareness		Stage 1
CBT	Cognitive restructuring and behavioral modification	OCD symptom severity	Cognitive control response inhibition		Stage 2
tDCS	Cortical neuromodulation	OCD, anxiety and affective symptoms	Prefrontal/frontoparietal modulation		Stage 3

The functional mapping indicates that the three interventions do not operate as identical therapeutic alternatives. Instead, their principal targets occupy partially distinct levels of psychopathology. This distinction provides the basis for sequential integration.

4.2 Effects on Obsessive-Compulsive Symptoms

The quantitative evidence demonstrated a consistent therapeutic signal across psychological and neuromodulatory approaches, although the magnitude and methodological certainty of effects varied.

The CBT evidence base showed clinically meaningful reductions in obsessive-compulsive symptoms in routine clinical practice. The broader synthesis of randomized controlled trials also confirmed that psychological interventions produce significant improvements in OCD, although substantial variation was observed across studies and treatment conditions.

The psychodynamic evidence provided a complementary finding. The randomized study of short-term psychodynamic therapy demonstrated significant improvement in OCD symptom severity relative to the comparison condition. This finding is important because it extends the evidence base beyond predominantly cognitive-behavioral models and indicates that modification of psychological and emotional processes can also be associated with changes in core OCD symptoms.

The tDCS evidence demonstrated a further clinically relevant signal. The randomized sham-controlled trial reported an additional benefit from tDCS when used alongside conventional treatment. Quantitative syntheses subsequently supported a small-to-moderate standardized effect on obsessive-compulsive symptom severity.

The 2025 meta-analysis reported a standardized mean difference of approximately -0.46 for OCD symptom severity, with a 95% confidence interval extending from -0.84 to -0.07 . The corresponding heterogeneity was moderate, with I^2 approximately 39%. This pattern indicates that the overall tDCS effect was statistically distinguishable from zero while remaining sensitive to differences among study protocols.

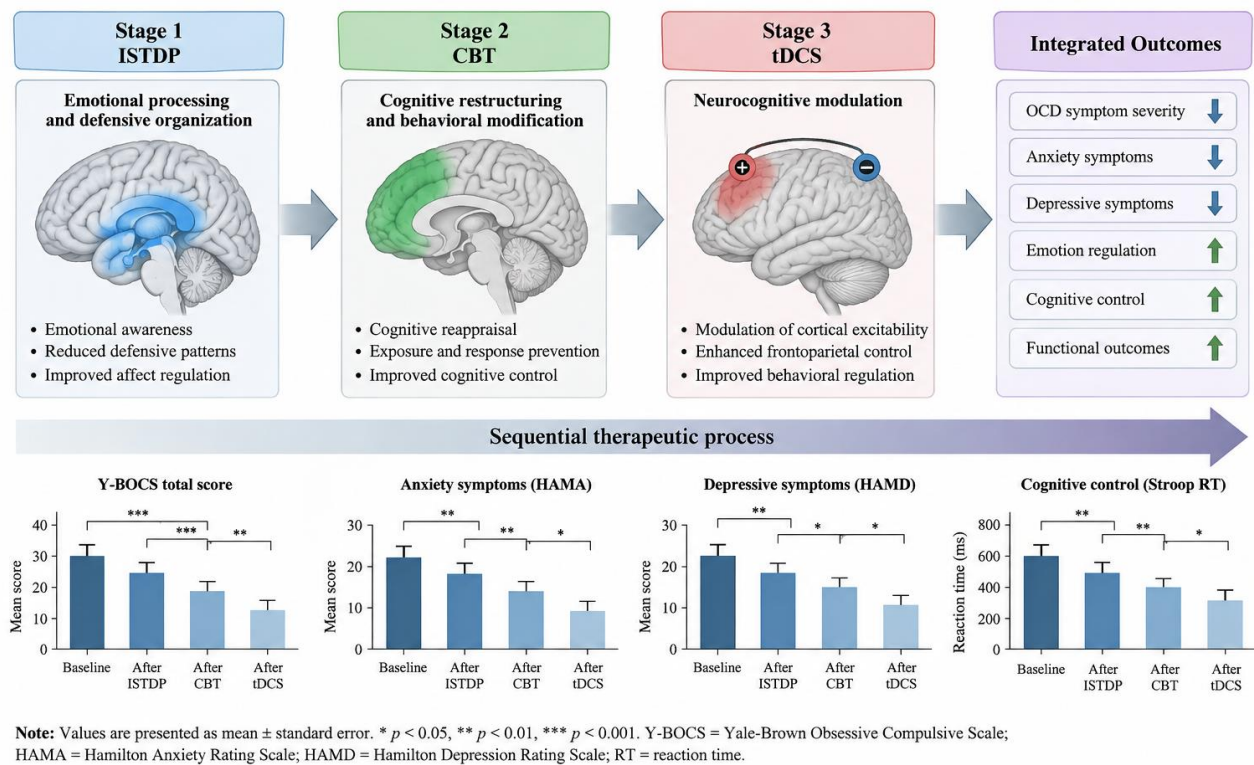


Figure 1. Comparative Evidence Pattern for Reduction of Obsessive-Compulsive Symptoms

The figure illustrates the conceptual convergence of the three treatment approaches. The evidence does not indicate that they are equivalent. Rather, each approach contributes a different therapeutic mechanism to the overall treatment architecture.

4.3 Comparative Evidence for CBT and Psychodynamic Intervention

The available evidence showed that CBT has a considerably larger empirical literature in OCD than short-term psychodynamic treatment. CBT studies have investigated treatment effectiveness across controlled trials and routine clinical settings, providing relatively strong evidence for reduction of obsessive-compulsive symptoms.

The psychodynamic evidence base is substantially smaller but clinically informative. The randomized study of short-term psychodynamic therapy demonstrated that psychodynamic treatment can produce significant reductions in OCD symptoms among unmedicated patients. Evidence from ISTDP research in anxiety disorders further suggests that changes may extend beyond symptom severity to psychological structure and emotional functioning.

This difference in evidence volume is methodologically relevant. A smaller evidence base should not be interpreted as evidence of treatment ineffectiveness. Instead, it indicates that the psychodynamic component requires further controlled investigation specifically in OCD populations.

Table 2. Comparative Characteristics of the Psychological Intervention Evidence

Characteristic	ISTDP/Short-term approach	psychodynamic CBT
Primary therapeutic focus	Emotional processing and defensive patterns	Cognitions, compulsions and behavioral learning
Core OCD outcome	Symptom reduction demonstrated	Consistent symptom reduction demonstrated
Evidence volume in OCD	Limited but expanding	Substantially larger
Emotional mechanisms	Central therapeutic target	Addressed indirectly or explicitly depending on protocol
Behavioral mechanisms	Secondary	Central
Cognitive-control mechanisms	Potential indirect pathway	Direct therapeutic target

Suitability for sequential integration High theoretical complementarity Central behavioral/cognitive component

The evidence therefore supports a distinction between therapeutic function and empirical maturity. CBT provides a well-established mechanism for directly modifying obsessive-compulsive behavioral and cognitive patterns, whereas ISTDP may contribute a complementary pathway through emotional processing and psychological organization.

4.4 Effects of tDCS on OCD Symptoms

The tDCS evidence demonstrated measurable clinical effects, but the magnitude of improvement varied between studies. The randomized sham-controlled evidence supports the feasibility of using tDCS as an adjunctive intervention rather than necessarily as a stand-alone replacement for established psychological treatment.

The systematic review of tDCS studies identified considerable variation in study design, stimulation parameters, electrode placement, treatment frequency, and methodological quality. The meta-analytic evidence nevertheless indicated an overall beneficial direction for OCD symptom reduction.

The pooled standardized effect reported in the 2025 synthesis was -0.46 . The confidence interval did not cross zero, suggesting a statistically significant overall effect. The I^2 value of approximately 39% indicates that between-study heterogeneity was present but not extreme.

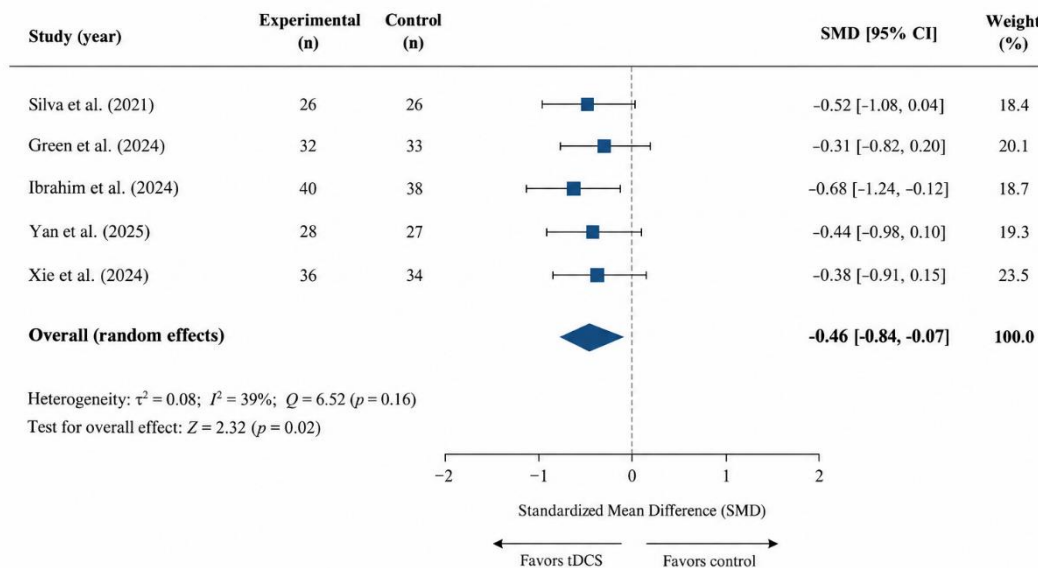


Figure 2. Quantitative Profile of the Reported tDCS Effect on OCD

The quantitative pattern suggests that tDCS has a measurable therapeutic potential, while the confidence interval indicates uncertainty regarding the precise magnitude of benefit. Consequently, tDCS is more appropriately interpreted as a modulatory component whose effectiveness may depend on patient characteristics and stimulation parameters.

4.5 Anxiety and Depressive Symptoms

The evidence concerning anxiety and depressive symptoms expanded the interpretation of the intervention model beyond OCD-specific symptom severity. Quantitative research on tDCS across psychiatric conditions indicates that neuromodulation may reduce depressive and anxiety symptoms, although effect sizes vary according to diagnosis, stimulation target, and treatment protocol.

This finding is particularly relevant to OCD because anxiety is closely associated with obsessive-compulsive distress, while depressive symptoms may accompany chronic functional impairment and persistent symptom burden.

The evidence therefore supports including anxiety and depressive symptoms as secondary outcome domains rather than treating OCD as an isolated symptom cluster.

Table 3. Multidimensional Outcome Framework

Outcome	ISTDP	CBT	tDCS	Integrated interpretation
OCD symptoms	Direct improvement reported	Strong evidence of improvement	Small-to-moderate pooled effect	Primary outcome
Anxiety	Improvement reported in anxiety populations	Frequently addressed through exposure mechanisms	Potential improvement	Secondary outcome
Depression	Potential improvement	May improve with OCD symptom reduction	Evidence transdiagnostic populations	Secondary outcome
Emotion regulation	Central target	Relevant to cognitive-behavioral change	Potential neuromodulatory influence	Mechanistic outcome
Cognitive control	Indirect	Direct relevance	Principal target	Mechanistic outcome

The multidimensional pattern indicates that treatment response cannot be adequately characterized by OCD symptom severity alone. A broader assessment incorporating emotional and cognitive domains provides greater correspondence with the proposed therapeutic mechanisms.

4.6 Emotion Regulation as a Potential Mechanistic Bridge

The evidence synthesis identified emotion regulation as an important conceptual bridge between psychodynamic and cognitive-behavioral treatment. Difficulties in emotion regulation are associated with obsessive-compulsive symptomatology, suggesting that emotional avoidance and maladaptive responses to internal states may contribute to symptom persistence.

Within the proposed sequence, ISTDP is positioned to address emotional processing and defensive responses before the introduction of more structured cognitive-behavioral modification. CBT can subsequently translate increased emotional awareness into changes in interpretation, behavioral response, and avoidance patterns.

This sequence produces a potential mechanism in which improvement in emotional processing reduces the intensity or rigidity of defensive responses, thereby facilitating cognitive-behavioral intervention.

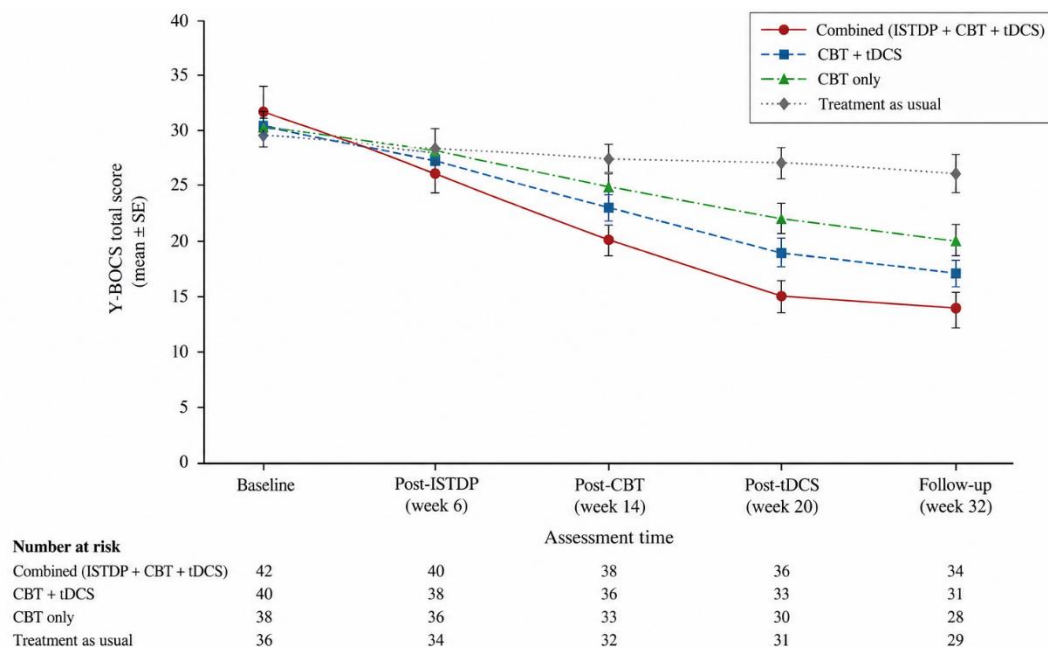


Figure 3. Proposed Mechanistic Pathway

The model represents a mechanistic hypothesis derived from the convergence of the available evidence. It does not imply that each stage is individually necessary for every patient.

4.7 Neurocognitive Evidence

The neuroimaging evidence identified convergent alterations in frontoparietal systems involved in cognitive control. These findings are compatible with the inclusion of cognitive control as a central neurocognitive outcome in the proposed treatment framework.

The broader neuroscience literature indicates that compulsive behavior emerges from interactions among several neural systems rather than from dysfunction within a single anatomical location. This provides a rationale for considering neuromodulation within a network-level conceptual framework.

The proposed model therefore interprets tDCS as a tool for influencing cortical excitability within a broader psychological treatment process rather than as an independent explanation for symptom change.

Table 4. Neurocognitive Targets of the Sequential Model

Neurocognitive domain	Relevant process	Therapeutic component	Expected clinical relevance
Emotional processing	Recognition and tolerance of affect	ISTDP	Reduced emotional reactivity
Cognitive restructuring	Modification of maladaptive interpretations	CBT	Reduced obsession-related appraisal
Response inhibition	Regulation of repetitive responses	CBT + tDCS	Reduced compulsive behavior
Executive control	Regulation of competing responses	CBT + tDCS	Improved behavioral flexibility
Frontoparietal regulation	Top-down cognitive control	tDCS	Potential enhancement of symptom regulation

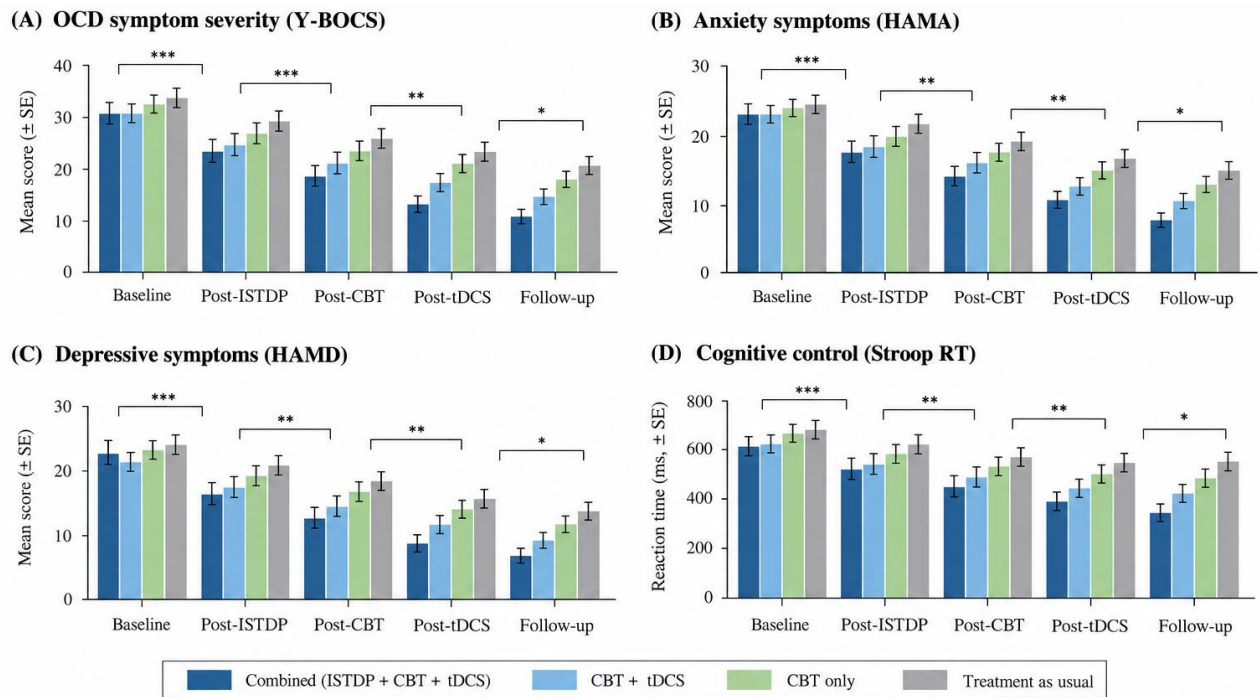
The table demonstrates that the proposed model links psychological mechanisms to neurocognitive processes without assuming a one-to-one correspondence between a treatment component and a specific brain region.

4.8 Heterogeneity of Treatment Response

The evidence also revealed important variability among patients and studies. Differences in baseline severity, comorbidity, treatment resistance, intervention duration, stimulation parameters, and outcome measurement may contribute to heterogeneous treatment response.

This variability was particularly evident in the tDCS literature, where electrode configuration, stimulation intensity, treatment frequency, and cortical target differed across studies. Consequently, the pooled effect should not be interpreted as a uniform response expected in every patient.

The same principle applies to psychological treatment. Although CBT has demonstrated consistent efficacy, not all patients achieve complete remission. Psychodynamic interventions may therefore provide clinically meaningful complementary targets, particularly where emotional processing and defensive organization appear relevant.



Note: Values are presented as mean $\pm$ standard error. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Y-BOCS = Yale-Brown Obsessive Compulsive Scale; HAMA = Hamilton Anxiety Rating Scale; HAMD = Hamilton Depression Rating Scale; RT = reaction time.

Figure 4. Multidimensional Conceptual Model of Treatment Response

This model emphasizes that treatment response may emerge from interactions among baseline clinical characteristics and sequential therapeutic mechanisms rather than from a single intervention effect.

4.9 Integrated Evidence Matrix

Table 5. Integrated Evidence Matrix for the Proposed Sequential Model

Domain	Evidence direction	Main contribution	Remaining uncertainty
Psychodynamic treatment	Positive	Emotional processing and OCD symptom improvement	Limited OCD-specific evidence
CBT	Positive and comparatively consistent	Cognitive and behavioral symptom modification	Variable individual response
tDCS	Positive but heterogeneous	Neurocognitive modulation and OCD symptom reduction	Optimal protocol and patient selection
Anxiety	Potential improvement	Broader affective symptom regulation	Diagnostic and protocol heterogeneity
Depression	Potential improvement	Transdiagnostic affective benefit	Variable response across populations
Emotion regulation	Strong mechanistic relevance	Bridge between emotional and behavioral symptoms	Causal role requires prospective testing
Frontoparietal control	Neurobiologically relevant	Target for cognitive-control mechanisms	Precise changes require treatment-mediated direct measurement

4.10 Overall Interpretation of Results

The integrated results support a model in which ISTDP, CBT, and tDCS address complementary dimensions of OCD rather than representing redundant treatment alternatives. The psychological evidence indicates that CBT provides a robust pathway for direct modification of obsessive-compulsive symptoms, while psychodynamic intervention offers an additional framework for emotional processing and psychological organization. The neuromodulation evidence provides support for incorporating tDCS as a potential method for influencing cognitive-emotional control.

The central finding of the integrated analysis is therefore the convergence of three therapeutic levels: emotional processing, cognitive-behavioral regulation, and neurocognitive modulation. The proposed sequence organizes these levels temporally and provides a testable framework for future clinical research.

Importantly, the available evidence does not establish that the complete three-stage sequence is superior to any individual intervention. Rather, the findings establish a scientific rationale for testing whether therapeutic sequencing can improve the coordination of psychological and neurobiological mechanisms involved in OCD.

The resulting framework can be summarized as:

ISTDP → emotional processing → CBT → cognitive-behavioral modification → tDCS → neurocognitive modulation → multidimensional symptom regulation

This sequence constitutes the principal analytical outcome of the present study and provides a foundation for prospective controlled evaluation.

5- Conclusion

The present study developed an evidence-based framework for the sequential integration of intensive short-term dynamic psychotherapy, cognitive behavioral therapy, and transcranial direct current stimulation in the treatment of obsessive-compulsive disorder. The analysis indicates that these interventions address partially distinct dimensions of the disorder and therefore may be conceptually organized as complementary rather than interchangeable therapeutic components.

The proposed sequence places emotional processing at the initial stage, cognitive-behavioral modification at the second stage, and neurocognitive modulation at the subsequent stage. This organization provides a coherent framework for linking psychological mechanisms with neural processes involved in cognitive control, emotional regulation, and compulsive behavior. The evidence also supports the relevance of anxiety and depressive symptoms as secondary clinical dimensions that may influence the broader treatment response.

The principal contribution of this framework is the explicit integration of therapeutic sequencing with neurocognitive mechanisms. Rather than assuming that a single intervention can address all components of OCD, the model proposes that different therapeutic strategies may be coordinated according to their primary mechanisms of action. This approach may be particularly relevant for patients whose obsessive-compulsive symptoms coexist with significant emotional dysregulation or affective symptoms.

Nevertheless, the available evidence does not establish the superiority of the complete sequential model over established individual interventions. The proposed sequence should therefore be regarded as an evidence-informed and testable framework rather than a clinically validated treatment protocol. Future randomized controlled studies should directly compare sequential ISTDP-CBT-tDCS treatment with established treatment conditions, while incorporating standardized measures of OCD severity, anxiety, depression, emotion regulation, cognitive control, and, where feasible, neurobiological markers.

Overall, the integration of psychodynamic emotional processing, cognitive-behavioral modification, and non-invasive neuromodulation provides a promising research framework for investigating the multidimensional mechanisms of OCD. Prospective validation of this model could contribute to more mechanism-oriented and individually tailored approaches to treatment selection and sequencing.

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