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NEUROMODULATION IN PEDIATRIC OBSESSIVE-COMPULSIVE DISORDER: A SYSTEMATIZED NARRATIVE REVIEW EMPHASIZING SEVERE AND TREATMENT-RESISTANT PRESENTATIONS

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ABSTRACT

Objective: Pediatric obsessive-compulsive disorder (OCD) is usually managed with cognitive-behavioral therapy with exposure and response prevention and selective serotonin reuptake inhibitors, yet a subset of patients remains severely symptomatic. This review evaluates the pediatric evidence for transcranial magnetic stimulation (TMS), transcranial direct current stimulation (tDCS) and deep brain stimulation (DBS), with emphasis on severe or treatment-resistant OCD.
Methods: We conducted a systematic narrative review of English-language literature published between 2015 and 2025. Searches were performed in PubMed/MEDLINE and Google Scholar and supplemented by reference-list screening. Eligible sources included pediatric or adolescent clinical studies, case reports, protocols and implementation-relevant ethical or developmental literature concerning TMS, tDCS, or DBS for OCD.
Results: Twenty-eight records were included. Pediatric evidence was sparse and heterogeneous, with few controlled data and limited follow-up. TMS was the most frequently described modality and was supported mainly by observational studies, case reports, and extrapolation from adult OCD literature. One sham-controlled adolescent tDCS trial reported improvement in both active and sham groups, without clear between-group superiority. No prospective pediatric DBS trials for primary OCD were identified; available evidence was limited to isolated cases, comorbid neuropsychiatric cohorts, and stakeholder or ethics studies.
Conclusions: Current evidence does not support routine neuromodulation for pediatric OCD. TMS appears closest to cautious clinical translation in specialist settings, whereas tDCS should remain research-focused. DBS should be restricted to exceptional, severe, treatment-refractory cases under multidisciplinary review, robust ethical oversight and developmentally appropriate consent or assent procedures.

KEYWORDS

Obsessive-Compulsive Disorder; Child; Adolescent; Transcranial Magnetic Stimulation; Transcranial Direct Current Stimulation; Deep Brain Stimulation

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1. Introduction

Obsessive-compulsive disorder (OCD) represents a significant public health burden affecting approximately 1-3% of the global population, with pediatric prevalence estimated at ~2-4%. It is characterized by persistent obsessions and compulsions that cause distress and functional impairment.[1,2]

Early-onset presentations are frequent: more than half of affected individuals report symptom onset before age 18, with peak incidence around ages 10-12.[1] These manifestations result in marked functional impairment across multiple life domains, with approximately 65% of cases involving severe impairment.[2] Despite this considerable clinical burden, pediatric OCD remains considerably underdiagnosed and undertreated. Epidemiological studies in population-based adolescent samples show that fewer than 10% of adolescents meeting diagnostic criteria receive a formal diagnosis, and only about 6-7% access adequate treatment.[1]

Beyond basic symptoms, pediatric OCD can disrupt psychosocial development and family functioning. Family accommodation is common and, although it may reduce distress in the short term, higher accommodation is associated with greater symptom severity, functional impairment and poorer treatment outcomes.[1,3]

Comorbidity is common in pediatric OCD.[1,3] Tic disorders/Tourette syndrome, ADHD and depressive/anxiety symptoms are frequent and can complicate assessment, treatment selection and monitoring during interventions.[1,3]

Current first-line treatment comprises cognitive-behavioral therapy with exposure and response prevention (CBT/ERP) combined with selective serotonin reuptake inhibitor (SSRI) medications, representing the evidence-based standard of care.[1,3] Meta-analytic evidence shows that approximately 68-70% of youth receiving CBT/ERP achieve treatment response.[1] However, about 20% of pediatric patients do not adequately respond even to combined behavioral and pharmacological treatment and may be considered treatment-resistant or refractory.[1,3] In such cases, clinicians have explored augmentation strategies (e.g., adding atypical antipsychotics to serotonergic medication), but pediatric evidence remains limited, relying largely on case series and open-label studies rather than robust controlled trials.[3]

Despite effective first-line care with CBT/ERP and SSRIs, a substantial minority of youth remain symptomatic, motivating interest in circuit-targeted neuromodulation. OCD has been consistently linked to dysfunction within cortico-striato-thalamo-cortical networks, and both non-invasive and invasive techniques aim to modify pathological activity within these circuits. In pediatric populations, neuromodulation is considered for severe impairment and family burden, while requiring developmentally informed dosing and safety and ethical oversight.[1,4,5,6]

1.1: Neurobiological Foundations and Circuit-Level Models

Contemporary models of OCD emphasize dysregulation within CSTC circuitry and interacting large-scale networks that shape habitual control, salience processing and affect regulation.[5]

The lateral orbitofrontal cortex (lOFC)-ventromedial caudate pathway is among the most extensively characterized circuits in OCD, underlying the persistence of habit-driven over goal-directed behavior.[5] This circuit comprises neurons projecting from lOFC through ventromedial caudate and globus pallidus internus (GPi) back to lOFC, forming a closed-loop that contributes to symptom heterogeneity (e.g., contamination vs symmetry obsessions).[5]

The limbic network, comprising hippocampus, anterior cingulate cortex (ACC), medial prefrontal cortex, amygdala and nucleus accumbens (NAcc), governs emotion, reward learning and memory.[5] In OCD, limbic dysregulation - manifested as altered reward learning and reduced frontal inhibition of striatal habit systems - implicated in OCD pathophysiology. SSRIs and neuromodulation have been reported to modulate connectivity measures in some studies.[5]

The salience network (dorsal ACC and anterior insula) *and default mode network* (self-referential processing) work together to direct attention. In OCD, aberrant connectivity within these networks contributes to excessive inward attention to intrusive thoughts and difficulty redirecting toward goal-directed behavior.[5]

Clinical and Targeting Implications

Circuit-based formulations inform neuromodulation target selection, particularly when stimulation is intended to differentially engage goal-directed vs habitual control systems.[4,5] Within neurocircuit taxonomy approaches, symptom content has been linked to differential activation patterns (e.g., contamination-related obsessions vs checking symptoms), supporting phenotype-informed targeting. However, these mappings remain an active area of research.[7]

1.2: Pediatric-Specific Neurodevelopmental Considerations

Pediatric OCD neuromodulation must be interpreted in a neurodevelopmental context, because the targeted circuits are still maturing throughout childhood and adolescence.[6,8] Unlike adults, children and adolescents experience ongoing developmental processes that shape neural circuit organization and may moderate both response and tolerability to brain stimulation.[6] The dorsolateral and medial prefrontal cortices support executive functions including response inhibition and cognitive control and show protracted maturation from early childhood through late adolescence - overlapping with the typical age range of OCD onset in youth.[1,6]

Synaptic Reorganization and Cortical Maturation

Across development, the prefrontal cortex undergoes dynamic remodeling, including synaptic pruning and changes in cortical thickness and myelination.[6] Cortical network topography changes substantially across development, characterized by greater short-range functional connectivity in early childhood and increased long-range functional connectivity with age.[6]

Heightened Cortical Reactivity and Parameter Adaptation

Compared with adults, developing brains may exhibit heightened cortical responsivity to electrical and magnetic stimulation, supporting developmentally sensitive parameter selection, conservative dosing and close monitoring rather than direct transfer of adult protocols.[6,9] In youth OCD, the NExT protocol specifies iTBS

at 70% RMT and cTBS at 90% RMT with clinician-guided adjustment.[4] Adult rTMS dosing is typically anchored to RMT and varies widely by protocol, limiting direct extrapolation to pediatric populations.[10]

For transcranial direct current stimulation (tDCS), most pediatric studies have used conventional adult intensities (1-2 mA) with acceptable short-term tolerability.[11,12] However, comprehensive long-term neurodevelopmental safety data and pediatric-specific dosing guidelines remain limited, supporting cautious interpretation and structured safety monitoring in pediatric trials.[6,8,11]

Distinct Neural Substrates in Pediatric OCD

Functional neuroimaging during symptom provocation in pediatric OCD demonstrates engagement of CSTC nodes together with additional cortical regions, consistent with developmentally shaped network recruitment rather than a purely "adult-like" pattern in all cases.[6,13] These developmental considerations have direct implications for target selection and parameter optimization, as interventions designed around mature adult circuits may not generalize straightforwardly to developing neural systems.[6,10]

Long-Term Developmental Considerations

Neurodevelopmental context necessitates heightened caution in pediatric neuromodulation compared to adult applications.[6,8,10] Myelination, synaptic pruning, white and gray matter volumes, and cortical thickness change at non-linear rates across development, raising unresolved questions about potential long-term effects of repeated stimulation on brain maturation, cognition, and behavior.[6] Extended longitudinal safety monitoring should therefore be considered a core element of pediatric neuromodulation trials.[6]

2. Methods and Study Selection

AI-assisted technology was used solely to improve readability and language. The authors reviewed and edited the output and take full responsibility for the content.

2.1: Review Type and Research Question

This review used a systematic search strategy with narrative synthesis to summarize evidence on the efficacy, safety, and clinical implementation of neuromodulation in pediatric OCD, with emphasis on severe and treatment-resistant presentations. The research question was: What is the current evidence for TMS, tDCS, and DBS in children and adolescents with OCD? Searches covered publications from 2015 through December 2025. One paper indexed in 2026 was included because it was accepted in 2025 and available ahead of print.

2.2: Information Sources and Search Strategy

PubMed and Google Scholar were searched systematically (final search: 12 December 2025) using combinations of terms related to pediatric OCD and neuromodulation (TMS/dTMS, tDCS, DBS, treatment-resistant/refractory). Reference lists of included articles were manually screened. Only English-language publications were included. In Google Scholar, results were screened to the end of the available results list.

2.3: Study Selection and Eligibility Criteria

Studies were included if they met at least one of the following: (A) primary pediatric OCD evidence - participants aged ≤21 years with OCD who received TMS/tDCS/DBS with reported efficacy (Y-BOCS/CY-BOCS) and/or safety outcomes; or (B) contextual evidence - trial protocols, systematic reviews/meta-analyses and ethical/developmental/implementation papers relevant to pediatric neuromodulation in OCD. Treatment resistance was variably defined. Therefore, treatment history was not an exclusion criterion and was extracted when explicitly reported. Studies were excluded if they evaluated adults only without separable pediatric data, were duplicates, off-topic, conference-only without sufficient methodological detail, or had unclear methods/outcomes. Case reports/series were retained when they represented the only available pediatric OCD-relevant clinical data.

2.4: Data Extraction and Quality Assessment

Data extracted included study design, sample characteristics, baseline severity, targets, stimulation parameters, concurrent treatments, and - when available - treatment history. Outcomes included percent Y-BOCS/CY-BOCS change, response rates, and adverse events. For systematic reviews and network meta-analyses, key summary findings were recorded for population-level context.

Study quality was appraised using design-appropriate criteria (risk of bias domains for RCTs; sample characterization, outcome rigor and follow-up completeness for observational studies and case series). Overall certainty of evidence by modality was summarized on a four-level scale GRADE (high, moderate, low, very low) considering limitations, consistency, directness and precision.

2.5: Data Synthesis

Findings were synthesized qualitatively and organized by modality (TMS, tDCS, DBS), with studies presented by design to enhance transparency. Pediatric-specific efficacy and safety signals were emphasized, while meta-analyses/network meta-analyses were used for broader context, acknowledging that much of the evidence derives from adult or mixed-age samples. Developmental considerations and ethical aspects (informed consent and clinical decision-making) were summarized from relevant conceptual and qualitative literature.

3. RESULTS

3.1: Study Selection Process

After screening 76 records, 46 full-text articles were assessed for eligibility. Twenty records were excluded (most commonly: conference abstracts without peer-reviewed full text, inability to extract pediatric-specific outcomes, or non-eligible study design). Twenty-six records met inclusion criteria. Two additional highly relevant papers were identified through reference-list screening, yielding 28 included records for qualitative narrative synthesis (2015-2025, including one article indexed as 2026 but available online ahead of print in 2025).

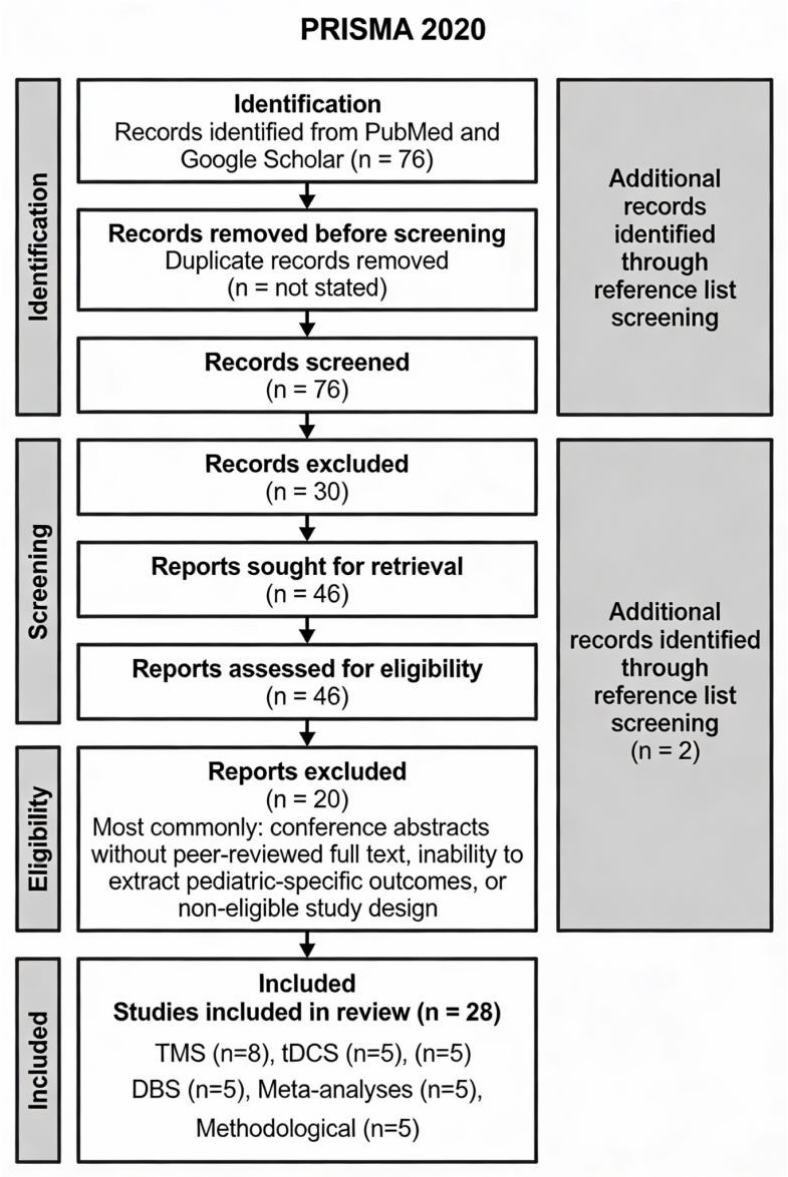

Fig. 1. *PRISMA 2020 flow diagram of the literature search and study selection process.*

Records were identified from PubMed and Google Scholar (n = 76) and from reference list screening (n = 2). After screening, 30 records were excluded. Forty-six reports were sought and assessed for eligibility; 20 were excluded (most commonly conference abstracts without peer-reviewed full text, inability to extract pediatric-specific outcomes, or non-eligible study design). Twenty-eight studies were included in the review.

3.2: Characteristics and Composition of Included Studies

The final dataset comprised 28 records: TMS (n=8), tDCS (n=5), DBS (n=5), and 10 contextual/meta-analytic or methodological papers retained to inform targeting, safety, and ethical interpretation. Across modalities, the primary pediatric OCD clinical evidence was dominated by small observational studies and case-based reports, reflecting the scarcity of randomized pediatric trials.

3.3: Neuromodulation Modalities - Mechanisms and Targets

3.3.1: Mechanistic Principles and Circuit-Informed Targeting

Neuromodulation comprises a spectrum of techniques that modulate abnormal neural activity patterns through electrical or magnetic stimulation, providing potential alternatives for treatment-resistant pediatric obsessive-compulsive disorder. The neurobiological rationale derives from detailed circuit mapping of cortico-striatal-thalamo-cortical (CSTC) dysfunction in OCD, as described in the previous section.

Transcranial Magnetic Stimulation (TMS) is a non-invasive technique in which a rapidly changing magnetic field induces an electric field in the cortex, modulating excitability.[5] In rTMS, higher frequencies (e.g., 5-20 Hz) are typically excitatory, whereas low-frequency stimulation (≤1 Hz) is typically inhibitory via LTP-/LTD-like mechanisms.[10]

Compared with conventional figure-8 coils, whose effective penetration is limited (approximately 0.5-1.5 cm), H-coil deep TMS is designed to engage deeper prefrontal-cingulate targets, including the mPFC and ACC.[14] In adult OCD, network meta-analytic evidence indicates that clinical effects of non-invasive brain stimulation vary across targets and protocols, with efficacy signals for selected prefrontal approaches - including high-frequency rTMS over bilateral DLPFC and high-frequency deep TMS targeting dmPFC/ACC - underscoring that target selection materially shapes outcomes and that extrapolation to youth requires caution.[15] The supplementary motor area (SMA) is a commonly investigated target in OCD, with meta-analytic signals supporting inhibitory protocols in this region.[16]

Transcranial Direct Current Stimulation (tDCS) applies a weak constant current (typically 1-2 mA) through scalp electrodes to bias cortical excitability. Practical advantages include low cost, portable equipment and the potential for supervised self-administration models in selected settings.[11,17] Key limitations are relatively diffuse current distribution, substantial inter-individual anatomical and physiological variability, and limited pediatric evidence - particularly regarding dose-response relationships and long-term neurodevelopmental safety.[9,11] In OCD, sham-controlled randomized trials have tested multiple montages (e.g., pre-SMA/SMA, mPFC, DLPFC and OFC) with heterogeneous efficacy across protocols.[5,17]

Deep Brain Stimulation (DBS) involves implantation of bilateral electrodes connected to an internal pulse generator, enabling continuous and programmable modulation of CSTC circuitry.[5,7] For context, in adults with severe, treatment-resistant OCD, commonly targeted nodes include the ventral capsule/ventral striatum (VC/VS) and anterior limb of the internal capsule (ALIC), nucleus accumbens/ventral striatum, anteromedial subthalamic nucleus (STN), globus pallidus internus, bed nucleus of the stria terminalis (BNST) and inferior thalamic peduncle (ITP).[5,7] Clinical benefit typically evolves over months and often requires iterative programming.[7]

3.4: Transcranial Magnetic Stimulation in Pediatric OCD

3.4.1: Clinical Evidence and Outcomes

Evidence for TMS in pediatric OCD remains preliminary, limited by small samples, heterogeneous protocols and a scarcity of well-controlled trials.[8,10] A narrative review of 32 child and adolescent TMS studies (2001-2025) identifies pediatric OCD as an area with early signals of potential benefit while emphasizing the need for more rigorous controlled research.[10]

Yang et al. (2025) conducted a single-center retrospective cohort study of 167 adolescents (12-18 years) with first-onset OCD, comparing low-frequency rTMS (1 Hz, 20 sessions) delivered over the right supplementary motor area (SMA), fluoxetine monotherapy, and combined treatment. The combined fluoxetine + LF-rTMS group achieved the highest response rate (85%, defined as ≥35% reduction in Y-BOCS), compared with 34% for rTMS alone and 64% for fluoxetine alone. The combined group also showed greater improvement on cognitive measures (Wisconsin Card Sorting Test) and quantitative electroencephalography patterns. No significant differences in adverse events emerged across groups.[18]

Das et al. (2023) reported a 17-year-old female with highly treatment-resistant OCD who received 20 sessions of H7 deep TMS targeting the mPFC/ACC as adjunctive treatment. The authors described a 66% improvement in obsession and compulsion symptom domains, alongside a 50% improvement in overall illness severity (CGI-S), with only mild transient headache.[14]

Prabha et al. (2024) described an individualized combined protocol in a 19-year-old with ultra-treatment-resistant OCD. Following insufficient response to previous stimulation approaches, the patient received a regimen combining ongoing deep TMS with low-frequency rTMS over the SMA, augmented by guided imagery to facilitate extinction learning. Progressive reduction in compulsive washing began at week 4, with cessation of foot washing by week 8.[19]

3.4.2: Implementation and Pediatric Considerations

SMA/pSMA remains the most commonly used inhibitory target in the available adolescent OCD TMS literature, consistent with its role in motor inhibition and habit circuitry.[4,5,18] Coil characteristics and deep TMS target considerations are summarized in Section 3.3.1.

In youth, stimulation dosing should follow age-adapted guidance with conservative thresholding and structured tolerability monitoring.[6,9] Across pediatric OCD studies, protocols vary widely (target, frequency, intensity, and session number), limiting cross-study comparability.[10,20] Standardized methodologies and pediatric-specific dosing frameworks are needed.[10] Combination strategies may be useful; in a large adolescent cohort, fluoxetine plus low-frequency rTMS over right SMA outperformed either monotherapy.[18]

3.4.3: Safety and Tolerability

Available pediatric TMS data suggest good short-term tolerability, with adverse effects typically mild and transient (e.g., scalp discomfort, headache). Serious adverse events appear rare but have been reported in youth TMS studies, underscoring the need for standardized screening and monitoring.[6,10]

Long-term developmental safety data remain limited, and follow-up in pediatric reports is typically short, often ≤6 months. (Section 1.2)[6]

3.5: Transcranial Direct Current Stimulation in Pediatric OCD

3.5.1: Evidence Base and Safety Profile

The evidence base for tDCS in adolescent OCD remains sparse and is largely limited to a single pilot randomized, sham-controlled trial. Agrawal et al. (2024) randomized 18 drug-naïve adolescents with first-episode OCD to active vs sham tDCS delivered alongside concurrently initiated fluoxetine; both arms improved over 12 weeks without significant between-group differences.[12] This design addresses early augmentation in treatment-naïve adolescents and does not directly inform efficacy in severe or treatment-resistant pediatric OCD.

Across available data, short-term tolerability appears acceptable. In Agrawal et al., no major adverse events were reported and side effects were mild and comparable between active and sham conditions.[12] In a larger mixed-diagnosis dataset of 2000+ sessions, adverse effects were predominantly mild and transient (burning ~16%, erythema ~12%, scalp pain ~10%, tingling ~10%).[21] Nevertheless, developmental neurophysiology suggests that adult-derived stimulation parameters may not translate directly to younger populations, supporting conservative dosing and longitudinal monitoring.[9,11]

Beyond symptom severity, a small case series of four adolescents reported improvements in shame, guilt and disgust sensitivity after 10 tDCS sessions, maintained at one-month follow-up (RCI ≥ 1.96).[22] Adult exploratory work also suggests that targeting L-OFC or SMA may influence cognitive inflexibility, but pediatric relevance remains uncertain.[17]

Overall, tDCS evidence in adolescent OCD remains preliminary, and clinical use should be restricted to research settings pending larger controlled trials.[11,12,21]

3.6: Deep Brain Stimulation In Pediatric OCD

3.6.1: Ethical Landscape and Clinical Experience

Muñoz et al. (2021) conducted semi-structured interviews with 25 clinicians experienced in treatment-resistant pediatric OCD. Five central themes emerged, three particularly salient in pediatric contexts. First, most clinicians (80%) raised concerns regarding adolescents' capacity to provide meaningful informed assent given developmental constraints. Second, 68% emphasized the limited evidence base for outcomes alongside uncertainty regarding long-term neurodevelopmental consequences. Third, 20% underscored the ethical imperative to exhaust conservative options before considering an invasive neurosurgical intervention involving implanted hardware.[23]

Two survey studies assessed acceptability of DBS for adolescents with severe, treatment-resistant conditions. Parents and other stakeholders reported moderate-to-high acceptability that increased with

expected symptom improvement and lower procedural risk, but declined when safety concerns, personality change, or long-term effects were emphasized. Familiarity with DBS and greater symptom severity were associated with greater willingness.[2,24]

Qualitative interviews with adolescents with OCD and their caregivers suggest that DBS is commonly expected to affect self-identity and agency, with some respondents anticipating positive identity exploration after symptom relief but others expressing concerns about authenticity and reduced agency. Views were also mixed regarding social impact, with some expecting improved social access and others anticipating increased stigma.[25]

3.6.2: Clinical Cases and Evidence

Currently, there are no randomized controlled trials evaluating DBS for primary pediatric OCD. Available clinical evidence remains restricted to isolated case reports and Tourette syndrome DBS cohorts in which obsessive-compulsive symptoms are comorbid rather than the primary indication.[26,27] Registry data in Tourette syndrome indicate that commonly targeted regions include the centromedian thalamic region and the globus pallidus internus (GPi), but obsessive-compulsive outcomes are not systematically reported as primary efficacy endpoints.[27] One such case illustrates both the potential and the limitations of this approach.

Srinivas et al. (2022) described a 14-year-old male with severe, treatment-refractory Tourette syndrome and prominent obsessive-compulsive symptoms who underwent bilateral anteromedial GPi DBS. At 6 months, tic severity and CY-BOCS both improved with maintained functional gains.[28] This case supports potential benefit in comorbid presentations, but it does not constitute evidence for DBS as a primary intervention for pediatric OCD.[26,28]

Ashkan et al. (2021) reviewed DBS across pediatric neuropsychiatric conditions and identified 73 studies. Critically, none evaluated pediatric OCD as the primary indication.[26] Overall, pediatric DBS applications most concerned Tourette syndrome and severe disruptive or aggressive/self-injurious presentations, reinforcing that pediatric OCD-specific DBS efficacy data remain anecdotal.[26,27]

3.6.3: Summary and Clinical Recommendations

This extreme scarcity of pediatric data contrasts with the adult OCD literature, where DBS is a clinically used option in selected refractory cases.[5,7] The evidence base for DBS in pediatric OCD remains virtually absent.[23,26] Key barriers include limited outcome data and unresolved concerns regarding long-term developmental impact and the risk-benefit balance of chronic implanted stimulation in youth.[23,26]

Accordingly, if considered at all, DBS should be restricted to exceptional, severely disabling cases within specialized multidisciplinary programs, with developmentally tailored consent/assent procedures adapted to adolescent decision-making capacity, including explicit discussion of potential impacts on identity, agency and stigma. [8,23,25]

Table 1. Comparative Overview of Neuromodulation Modalities in Pediatric Obsessive-Compulsive Disorder

Domain	TMS	tDCS	DBS
Invasiveness	Non-invasive[10,20]	Non-invasive[12]	Invasive (neurosurgical)[23,26]
Evidence Level	Moderate (multiple studies, RCTs emerging)[10,18,20]	Very limited (single pilot RCT)[12]	Virtually absent (ethical discussions only)[23,26]
Targets & Protocols	SMA (1 Hz); mPFC/ACC (dTMS H7, 20-30 sessions); pre-SMA (TBS)[5,10,20]	DLPFC anode + OFC cathode (2 mA, 20 min, 10-20 sessions); L-OFC/SMA[12,17]	ALIC/VC/VS, NAcc, STN, BNST (targets not pediatrically established)[7,26]
Clinical Availability	Growing at specialized centers; FDA approval for adult OCD[10,20]	Experimental only; limited regulatory clearance[12]	Highly specialized neurosurgical centers only[23,26]
Safety Profile	Mild-moderate: transient headache, low seizure risk (0.087%); no cognitive impairment[3,10]	Mild: erythema, itching; no serious AEs in 2,000 sessions; heightened pediatric sensitivity[9,11,21,22]	Significant: surgical risks (hemorrhage, infection); mood/autonomy effects; unknown long-term neurodevelopmental impact[23,25,26]
Algorithm Position	2nd-3rd line after failed SSRI + CBT/ERP[10,18,20]	2nd-3rd line (exploratory early augmentation)[12]	Last resort: exceptional TR-OCD with severe impairment, suicide risk, multidisciplinary consensus[23,25,26]
Clinical Summary	✓ Most evidence; ✓ Safe; ✓ Compatible with SSRIs; ! Limited long-term pediatric data[3,10,18,20]	✓ Cost-effective; ✓ Portable; ✗ Insufficient RCT evidence; ! Placebo response high[11,12]	✗ No primary pediatric OCD trials; ! Informed assent concerns; ✗ Irreversible[23,25,26]

Abbreviations:

Neuromodulation modalities: TMS, Transcranial Magnetic Stimulation; tDCS, Transcranial Direct Current Stimulation; DBS, Deep Brain Stimulation; dTMS, Deep Transcranial Magnetic Stimulation; H-coil/H7, H-shaped coil (H7 coil); RCT, Randomized Controlled Trial; FDA, Food and Drug Administration.

Brain regions and targets: SMA, Supplementary Motor Area; pre-SMA, pre-supplementary motor area; mPFC, Medial Prefrontal Cortex; ACC, Anterior Cingulate Cortex; DLPFC, Dorsolateral Prefrontal Cortex; OFC, Orbitofrontal Cortex; L-OFC, Lateral Orbitofrontal Cortex; ALIC, Anterior Limb of Internal Capsule; VC, Ventral Capsule; VS, Ventral Striatum; NAcc, Nucleus Accumbens; STN, Subthalamic Nucleus; BNST, Bed Nucleus of Stria Terminalis.

Clinical and treatment terms: OCD, Obsessive-Compulsive Disorder; TR-OCD, Treatment-Resistant OCD; SSRI, Selective Serotonin Reuptake Inhibitor; CBT/ERP, Cognitive-Behavioral Therapy with Exposure and Response Prevention; AE/AEs, Adverse Event(s).

4: Discussion

4.1: Key Findings and Evidence Synthesis

Overall certainty of evidence for pediatric OCD neuromodulation is low (TMS, tDCS) to very low (DBS) under GRADE, driven primarily by small samples and limited controlled pediatric trials.[8,10,20]

Among available neuromodulation approaches, TMS has the most developed adolescent/youth literature, supported by circuit-informed rationale and emerging clinical data, including observational cohorts and case-level reports.[10,18,20] Deep TMS in youth remains case-based at present and should be interpreted as an emerging, investigational extension rather than a mature evidence base.[10,14] In an adolescent retrospective cohort, combined fluoxetine + low-frequency rTMS over right SMA was associated with higher response than either monotherapy, supporting clinical plausibility of combination strategies.[18] Separately, isolated case reports describe marked symptom improvement following dTMS protocols, but these findings remain hypothesis-generating.[14,19]

For tDCS, practical advantages (cost, accessibility, feasibility of supervised delivery) are clear, yet current pediatric/adolescent OCD efficacy evidence does not demonstrate superiority over sham.[11,12] Importantly, the only sham-controlled adolescent OCD trial examined early augmentation in treatment-naïve, first-episode patients, limiting direct inference for severe or treatment-resistant pediatric OCD.[12] DBS, in contrast, remains experimental for pediatric OCD. Available pediatric evidence primarily reflects comorbid Tourette syndrome cohorts and isolated case reports rather than OCD as a primary indication.[26,27,28]

4.2: Interpretation Within The Context Of Existing Research and Neurodevelopment

Because targeted circuits remain under active maturation in youth (Section 1.2), pediatric neuromodulation cannot be treated as a direct downscaling of adult protocols and requires developmentally informed parameter selection and monitoring.[6,8,10]

Functional neuroimaging studies in pediatric OCD occasionally suggest that symptom provocation engages CSTC circuitry alongside additional cortical and limbic recruitment, including temporal poles and limbic regions, in some youth presentations.[13] This developmental heterogeneity supports pediatric-specific targeting strategies and careful parameter optimization rather than direct transfer of adult protocols.[4,6] As detailed in Sections 3.3.1 and 3.4.2, SMA/pSMA remains the most consistently represented inhibitory target across available youth TMS studies.[4,5,10,18]

Long-term neurodevelopmental safety remains incompletely characterized (Section 1.2), necessitating extended prospective follow-up with comprehensive neurocognitive assessments.[6,8]

4.3: Critical Evidence Gaps and Knowledge Deficiencies

Translation to routine care is limited by scarce and heterogeneous pediatric randomized evidence, particularly for tDCS and DBS.[8,10,20] For tDCS, data are confined to a small pilot sham-controlled trial, and DBS lacks pediatric OCD-specific trials; available reports largely involve comorbid Tourette cohorts and ethics-focused discussions.[11,12,23,26,27,28]

Predictive biomarkers and validated stratification tools are not yet available. At present, there are no validated neuroimaging or clinical markers enabling personalized intervention selection across modalities or optimize target choice and dosing in youth.[6,20] Similarly, pediatric age-stratified parameter guidance (e.g., across late childhood vs early vs late adolescence) remains largely conceptual and has not been empirically established in OCD-specific cohorts.[6,8]

4.4: Clinical Implications and Integration Into Treatment Algorithms

Pending stronger efficacy and long-term safety data, neuromodulation in pediatric OCD should be framed as a specialist, escalation-level intervention for youth with clearly documented non-response to evidence-based first-line care (adequate CBT/ERP and pharmacotherapy, with augmentation when clinically indicated).[10,20] Where neuromodulation is considered, implementation should occur in specialized multidisciplinary teams (child and adolescent psychiatry, neuromodulation expertise, neuropsychology, and ethics support), with structured monitoring and strong family engagement.[8,23]

Within currently available non-invasive options, TMS is the most clinically implementable modality, SMA/pSMA is the best-supported inhibitory target in adolescent frameworks and cohort data, with pediatric-adapted dosing and thresholding as outlined earlier.[4,6,18] Combination strategies are clinically plausible: observational adolescent data suggest improved outcomes when rTMS is combined with pharmacotherapy, and youth protocols are actively evaluating ERP-augmented approaches.[4,18] In contrast, tDCS should remain primarily a research modality in pediatric OCD until larger controlled trials clarify efficacy, dosing and long-term developmental safety.[9,11,12]

For DBS, the central issue is the combination of minimal pediatric OCD-specific efficacy evidence and unresolved developmental and governance questions.[8,23,26] Stakeholder work highlights the risk of overestimating benefit and underappreciating uncertainty in severe, refractory illness, and interviews with adolescents and caregivers raise additional concerns about potential impacts on identity, agency and stigma.[2,8,23,25] Accordingly, if DBS is considered at all, it should be restricted to exceptional cases within robust ethical oversight frameworks, with transparent risk communication, developmentally tailored consent/assent processes and long-term follow-up.[8,23]

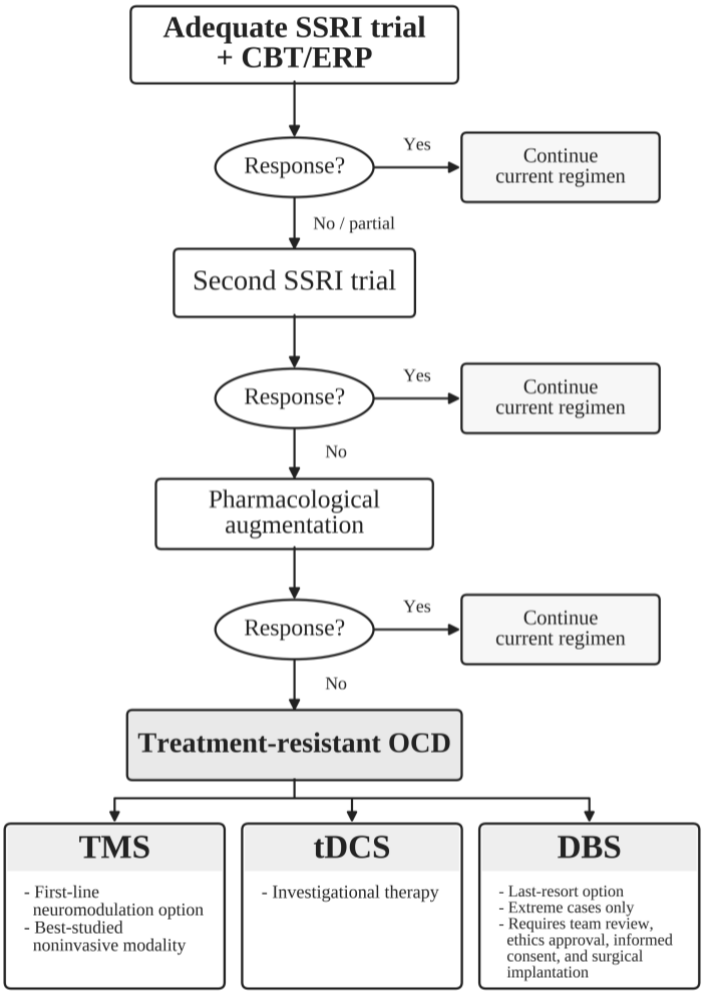

Fig. 2. *Neuromodulation decision pathway for pediatric treatment-resistant obsessive-compulsive disorder (OCD).*

Clinical algorithm outlining escalation from first-line treatment (selective serotonin reuptake inhibitor [SSRI] plus cognitive-behavioral therapy with exposure and response prevention [CBT/ERP]) to a second SSRI trial and pharmacological augmentation. In the absence of response, neuromodulation options include transcranial magnetic stimulation (TMS; first-line neuromodulation), transcranial direct current stimulation (tDCS; investigational), and deep brain stimulation (DBS; last resort). "Continue" indicates maintenance of the current effective treatment.

4.5: Research Priorities and Implementation Framework

Future pediatric trials should incorporate long-term follow-up of at least 12-24 months to evaluate durability of clinical response and neurocognitive safety.[4,10,20]

Within TMS research, key priorities include systematic evaluation of inhibitory SMA/pSMA protocols, comparison with alternative targets where clinically justified and formal testing of combined strategies (with pharmacotherapy/ERP) using designs capable of isolating additive benefit.[4,10,18] Mechanistic and biomarker work should accompany clinical trials. Priority areas include symptom provocation paradigms, resting-state connectivity approaches and multimodal predictors of response that may enable individualized targeting and dosing in developing brains.[6,13,20] Computational modeling that accounts for pediatric anatomy and network-level circuit engagement may further refine targeting and reduce off-target effects.[5,20]

Finally, implementation science is needed. Establishment of specialized pediatric neuromodulation programs and registries could improve safety surveillance, standardize reporting, and enable long-term tracking of developmental trajectories alongside clinical outcomes.[10,20,27] Education and competency frameworks for clinicians involved in pediatric neuromodulation - spanning patient selection, parameter optimization, adverse event monitoring, and ethical governance - should be treated as a core prerequisite for responsible expansion of clinical use.[8,10,23]

4.6: Limitations of This Review

This systematized narrative review has several limitations. Heterogeneity across pediatric neuromodulation studies (including variability in design, patient characteristics, stimulation parameters and outcome measurement) limits cross-study comparability and precludes formal meta-analytic synthesis.[10,20] Most available data derive from small single-center studies, case series, or case reports, restricting statistical power and limiting causal inference.[10,20]

The 2015-2025 search window may have missed earlier foundational work, although key concepts were incorporated via later syntheses. Exclusion of inaccessible full texts may have introduced selection bias.

Long-term developmental safety data remain limited, with pediatric follow-up often short.[6,8] Finally, important pediatric subgroups remain understudied, including youth with neurodevelopmental comorbidity (ASD/ADHD/tics) and sociodemographically underrepresented groups, limiting external validity and implementation generalizability.[10,20]

5. Conclusions

Neuromodulation is an emerging adjunct for severe pediatric OCD and must be interpreted within neurodevelopmental and ethical frameworks.[6,8,10,20] TMS is the best-studied modality and appears feasible and generally well tolerated in the short term; evidence for tDCS is limited and DBS remains investigational.[10,12,20] Future progress requires adequately powered pediatric trials with long-term follow-up, standardized protocols, and mechanistic work to guide individualized targeting.[4,10,20] Until stronger data are available, neuromodulation should be reserved for highly selected cases in specialized services with developmentally appropriate consent/assent and longitudinal safety monitoring.[6,8,23]

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