(2) synthesise outcomes for deep transcranial magnetic stimulation, transcranial direct current stimulation and deep brain stimulation; (3) identify optimisation signals (dose, duration, targets).
Methods: Systematic searches in MEDLINE/PubMed, Embase, PsycINFO and Web of Science (2020–2025) in adults with TR-OCD. Designs: RCTs, meta-analyses, observational safety cohorts. Comparators: placebo/treatment-as-usual (drugs), sham (devices). Outcomes: Y-BOCS change, response (≥35%), AE/SAE, discontinuations; metrics: RR, OR, NNT. Flow: 1,004 records; 742 post-deduplication; 114 full texts; 26 included; 88 excluded. Populations: mean age 30–45; women 45–55%; tertiary clinics. Mixed samples included only with TR-OCD subgroup data or ≥70% TR; other effects interpreted cautiously.
Results: Antipsychotic augmentation: response 33% in SSRI non-responders (NNT≈5); risperidone/aripiprazole consistent; quetiapine/olanzapine inconsistent. AE: akathisia 10–20%, sedation/weight gain 15–30%; discontinuations 10–25%. Glutamatergic: memantine (7 RCTs; n=315) large effect by ≥8 weeks (SMD≈−1.17; 20 mg/day); rash risk ↑ (RR≈7.00); discontinuations 0–3%. N-acetylcysteine (6 RCTs; n=195) modest improvement at 5–8 weeks (≈−3 Y-BOCS points); AE ~placebo (0–2%); durability >12 weeks not consistently reported. 5-HT3 antagonists (6 RCTs; n=334; ondansetron/granisetron/tropisetron) reduced Y-BOCS total/subscales; discontinuations 0–2%; AE mild, mainly gastrointestinal. Neuromodulation: dTMS (4 RCTs; n=252) RR 3.71 post-treatment and 2.60 at 1 month; absolute gain +20–30 pp; AE: headache 35–38%, local discomfort ~30%; seizures <0.01%/session. Real-world: 72.6% initial; 52.4% ≥1 month. tDCS (7 RCTs; n=201) small–moderate effect (SMD≈−0.46); AE ~sham; durability >1–3 months not consistently reported. DBS (9 RCTs; n=91) −5.1 Y-BOCS vs sham; OR≈4.7; NNT≈3.9; assessed ≥3–6 months; hardware ≈8%, infections 4–5%, hypomania/suicidality 3–4%.
Conclusions: Antipsychotic augmentation yields ~33% response with tolerability costs. Glutamatergic add-ons: memantine (large effect; rash risk), N-acetylcysteine (modest, time-dependent; safe), 5-HT3 antagonists (consistent; discontinuations 0–2%). Among devices, dTMS shows most consistent short-term evidence (RR 3.71; +20–30 pp; 52% sustained); tDCS gives small–moderate gains; durability uncertain; DBS effective for severe, selected cases (NNT≈3.9). Major limitation: between-study heterogeneity in TR-OCD definitions and stimulation/augmentation parameters limits interpretability and generalisability.

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EPP448

Inattention in Obsessive-Compulsive Disorder Depends on Anxiety Load

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Introduction: Obsessive-compulsive disorder (OCD) is diagnosed by clinically impairing obsessions and/or compulsions. A proportion of OCD is comorbid with attention-deficit/hyperactivity disorder (ADHD), especially with the inattentive subtype (ADHD-IA). Inattention in OCD has been explained due to a cognitive overload with anxiety and obsessions, impeding effortful concentration. This study explores whether a diagnosis of OCD with ADHD-IA is accompanied by excess anxiety, compared to OCD without ADHD-IA.
Objectives: 1) To test if anxiety disorders are in excess in OCD + ADHD-IA compared to OCD only; 2) to identify specific anxiety disorders that are in excess in OCD + ADHD-IA.
Methods: 989 probands from the OCGAS sample with a primary OCD diagnosis and ages 7-75 years comprised the sample. The Kiddie Schedules for Affective Disorders and Schizophrenia (K-SADS) was used to ascertain diagnoses of OCD and anxiety disorder in youth, and the Structured Clinical Interview for DSM Disorders (SCID) ascertained diagnoses in adults. Chi-square tests tested frequency data, and logistic regressions controlled for confounders.
Results: Of 989 participants, there were 806 OCD only (81 %) and 183 OCD + ADHD-IA (19 %). OCD + ADHD-IA was more male (55% vs. 42%, $p < 0.001$), and younger (24.6 ± 13.3 vs. 30.3 ± 14.6 years, $p = 0.002$). OCD + ADHD-IA had a higher proportion of any anxiety disorder (73% vs. 52%; ▫ ▫ 2=27.2; $p < 0.001$), with the highest differences present for generalized anxiety disorder (GAD) (38% vs. 27%; ▫ ▫ 2 = 8.21; $p = 0.004$), social anxiety disorder (SocAnx) (30% vs. 20%; ▫ ▫ 2 = 7.24; $p = 0.007$), separation anxiety disorder (SAD) (31% vs. 16%; ▫ ▫ 2 = 22.54; $p < 0.001$) and specific phobia (SPho) (22% vs. 13%; ▫ ▫ 2 = 8.54; $p = 0.003$), but not for agoraphobia/panic disorder.
On stratification, adults with OCD + ADHD-IA had significantly more 'any anxiety disorder', SocAnx, and SAD, while youth had significantly more 'any anxiety disorder', GAD and SPho. Males with OCD + ADHD-IA had more 'any anxiety disorder', GAD and SAD, while females with OCD + ADHD-IA had more 'any anxiety disorder', SocAnx, SAD. Results did not change when logistic regressions controlled for age and sex of participants. Across groups, SAD had the strongest association with OCD + ADHD-IA.
Conclusions: In this sample of OCD families recruited for genetic studies, probands with OCD with ADHD-IA had an excess of anxiety disorders with specific disorders linked to age and sex of participants. Whether the ADHD-IA phenotype is an alternate expression of anxiety in this OCD sample remains to be explored. In practice, both anxiety and ADHD-IA conflate the symptom of lack of concentration or inattention and may reflect the same mechanism of lack of effortful concentration.

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EPP449

Vortioxetine monotherapy for treatment-resistant OCD: evidence from a retrospective multicentre study

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