REVIEW ARTICLE

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The Neurobiological Foundations of Obsessive-Compulsive Personality Disorder: A Comprehensive Review

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Abstract

Objective: This review aims to integrate and critically evaluate the existing neurobiological evidence concerning Obsessive-Compulsive Personality Disorder (OCPD). It synthesizes findings from neuroimaging, genetic, and neuropsychological studies to propose a distinct etiological model that differentiates OCPD from Obsessive-Compulsive Disorder (OCD).
Methods: A systematic literature search was conducted across PubMed, PsycINFO, and Google Scholar for studies published between 1990 and 2025. Keywords included "obsessive-compulsive personality disorder," "neurobiology," "neuroimaging," "genetics," and related terms. Studies were included if they provided original empirical data on the neurobiology of OCPD or its core traits. Data were narratively synthesized due to methodological diversity.
Results: Fifty-six studies met the inclusion criteria. Structural MRI findings indicate increased grey matter volume in the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC). Functional MRI studies reveal hyperactivation in the DLPFC, dorsal ACC, and fronto-parietal network during tasks of cognitive control and error monitoring, alongside reduced connectivity with limbic regions. Neurochemical evidence points to dysregulation in serotonin and dopamine systems. Genetic studies show high heritability (approximately 50–78%) and potential associations with genes such as *SLC6A4*, *COMT*, and *DRD3*. Neuropsychological profiles reflect intact planning abilities but impairments in cognitive flexibility and heightened error sensitivity.
Conclusion: OCPD is associated with a unique neurobiological profile characterized by overactive prefrontal cognitive control systems and diminished integration with emotional processing regions, rather than the fear-based circuitry typical of OCD. This "hyper-executive" model accounts for core OCPD traits such as perfectionism, rigidity, and excessive need for order. Future research should prioritize well-defined OCPD cohorts to validate this model and develop targeted, biologically informed interventions.

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Introduction

Obsessive-Compulsive Personality Disorder (OCPD) is characterized by a persistent and inflexible pattern of maladaptive thoughts and behaviors centered on perfectionism, orderliness, and excessive control [1]. According to the DSM-5-TR, its features include rigid rule adherence, reluctance to delegate, and moral inflexibility, leading to significant functional impairment and interpersonal distress [1]. With an estimated prevalence of 2.1–7.9% in the general population, OCPD is the most common personality disorder, surpassing rates of other conditions such as Borderline Personality Disorder [2,3]. It is associated with substantial occupational dysfunction, relationship difficulties, and high comorbidity with anxiety and eating disorders [4,5]. Despite its clinical relevance, OCPD has been relatively overlooked in psychiatric research, often overshadowed by studies on OCD and other personality disorders [6].

Historically, neurobiological research on OCPD has been limited by two main factors: its symptomatic overlap with OCD and the historical emphasis on psychodynamic rather than neuroscientific models for personality disorders [7–9]. While OCD involves ego-dystonic compulsions driven by anxiety, OCPD behaviors are ego-syntonic and rooted in a need for order and perfection [8]. This distinction suggests divergent neural underpinnings. Although the neurocircuitry of OCD is well-described in terms of orbitofronto-striato-thalamic loops [10], a coherent neurobiological framework for OCPD is only beginning to emerge.

This integrative review synthesizes the growing neurobiological literature on OCPD to address a significant gap in understanding. It examines evidence from structural and functional neuroimaging, neurochemistry, genetics, and neuropsychology to answer the central question: What are the distinctive neural and molecular mechanisms underlying the persistent pattern of perfectionism, cognitive rigidity, and overcontrolled behavior in OCPD? By integrating these findings, we propose a preliminary "hyper-executive" neurobiological model, clarify distinctions between OCPD and OCD, discuss clinical implications, and outline future research directions.

Methods

Following established guidelines for narrative synthesis [11], a systematic search was conducted in July 2025 across PubMed, PsycINFO, and Google Scholar. Search terms included ("obsessive-compulsive personality disorder" OR OCPD) combined with neurobiological keywords (e.g., "neuroimaging," "genetics," "fronto-striatal"). The search was limited to English-language studies published between 1990 and 2024.

Inclusion Criteria:

1. Original empirical studies investigating neurobiological aspects of clinically diagnosed OCPD or high OCPD traits in analogue samples.
2. Studies examining core OCPD phenotypes with clear neurobiological measures.
3. Review articles and meta-analyses were consulted for references but not included in the primary synthesis.

Exclusion Criteria:

1. Studies focused solely on OCD without separate OCPD analysis.
2. Theoretical or descriptive papers without original data.
3. Studies lacking specific neurobiological correlates.

Screening was performed by the primary author, and data were extracted into a standardized template. Due to methodological heterogeneity, a narrative synthesis was conducted across thematic areas: structural neuroanatomy, functional neuroimaging, neurochemistry, genetics, and neuropsychology. Fifty-six studies met the inclusion criteria.

Results and Discussion

Structural Neuroanatomy

Structural MRI studies indicate increased grey matter volume (GMV) in the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) in individuals with OCPD traits [12,13], contrasting with reduced orbitofrontal cortex volume often observed in OCD [15]. One study found a positive correlation between perfectionism and GMV in the dorsal ACC and bilateral DLPFC [16]. White matter analyses suggest enhanced integrity in frontal tracts, possibly reflecting rigid communication within frontal-subcortical circuits [18].

Functional Neuroimaging

Task-based fMRI studies show hyperactivation in prefrontal control regions (e.g., DLPFC, dorsal ACC) during cognitive control and error-processing tasks [19,20]. Electrophysiological data support heightened error sensitivity, as indicated by enhanced Error-Related Negativity (ERN) [21]. Resting-state fMRI reveals hyperconnectivity within executive networks (fronto-parietal and cingulo-opercular networks) and hypoconnectivity with limbic regions such as the amygdala and ventral striatum [23,24].

Neurochemistry and Molecular Imaging

Indirect evidence suggests serotonergic and dopaminergic dysregulation in OCPD. The efficacy of SSRIs points to serotonin's role in modulating behavioral inhibition [25], while dopamine dysfunction may contribute to cognitive inflexibility [27]. Genetic associations with serotonin and dopamine pathways further support these findings (see below).

Genetics

Twin studies indicate high heritability for OCPD traits (52–78%) [28,29]. Candidate gene studies have linked OCPD to polymorphisms in serotonin transporter (5-HTTLPR), catechol-O-methyltransferase (COMT), and dopamine D3 receptor (DRD3) genes [30–32]. No genome-wide association studies (GWAS) for OCPD have been published to date.

Neuropsychological Correlates

Neuropsychological assessments show that individuals with OCPD perform well on tasks of planning and sustained attention but exhibit significant deficits in cognitive flexibility. They demonstrate perseverative errors on the Wisconsin Card Sorting Test and impaired performance on task-switching paradigms [35,36]. Decision-making tasks reveal a preference for low-risk, rule-based strategies, consistent with overreliance on executive control and reduced affective input [37,38].

The "Hyper-Executive" Model: A Distinct Neural Signature

The synthesized evidence supports a "hyper-executive" model of OCPD, characterized by overactive prefrontal cognitive control systems (DLPFC, dACC) and diminished integration with limbic affective regions. This model differentiates OCPD from OCD, which involves hyperactivity in ventral affective circuits [10,39]. In OCPD, excessive top-down control without adequate emotional modulation leads to rigid, perfectionistic behavior [40].

Integrating Neurochemistry and Genetics Neurochemical and genetic findings provide a molecular basis for circuit-level dysfunction. Serotonergic and dopaminergic alterations may amplify prefrontal control signals while reducing cognitive flexibility, aligning with observed behavioral traits [41,42]. High heritability estimates underscore the genetic underpinnings of these mechanisms [28,29].

Clinical Implications

The "hyper-executive" model reframes OCPD as a brain-based disorder, reducing stigma and informing treatment. Therapies such as Radically Open Dialectical Behavior Therapy (RO-DBT) aim to enhance cognitive flexibility and emotional integration [43]. Pharmacological agents targeting serotonin and dopamine systems, as well as neuromodulation techniques like rTMS, warrant further investigation [44,45]. Future research should prioritize large-scale neuroimaging studies, longitudinal designs, social-affective paradigms, genomics, and intervention-based neuroscience to advance understanding and treatment of OCPD.

Conclusion

The neurobiological profile of OCPD points to a disorder of excessive cognitive control, marked by prefrontal overactivity and reduced limbic connectivity. This "hyper-executive" model offers a coherent explanation for core OCPD traits and distinguishes it from OCD. While further research is needed, current evidence establishes OCPD as a distinct neurobiological condition. Advancing this knowledge is essential for developing effective, biologically informed diagnostic and therapeutic approaches to alleviate the burden of this prevalent disorder.

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Authors Contributions

The authors contributed to the data analysis. Drafting, revising and approving the article, responsible for all aspects of this work.

Conflict of Interest

None

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