Psychopathology

Systematic Review

Psychopathology
DOI: 10.1159/000526708

Received: November 23, 2021
Accepted: August 15, 2022
Published online: October 25, 2022

# Neurodevelopmental Antecedents and Sensory Phenomena in Obsessive Compulsive Disorder: A Systematic Review Supporting a Phenomenological-Developmental Model

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**Keywords**
Obsessive compulsive disorder · Sensory phenomena · Intrusive thoughts · Corollary discharge · Sense of agency · Schizophrenia spectrum disorders · Neurodevelopment · Phenomenology

**Abstract**
***Background:*** The majority of models on obsessive compulsive disorder (OCD) endorse a top-down perspective on the cognitive mechanisms underlying OCD functioning and maintenance, whereas a bottom-up perspective is rarely pursued. ***Objectives:*** The aim of the study was to review the empirical literature on sensory phenomena (SP) and neurodevelopmental antecedents of OCD, which could support the conceptualization of an alternative, bottom-up perspective integrating neurodevelopmental and phenomenological levels of analysis on OCD. ***Methods:*** A systematic review according to PRISMA guidelines was performed in PubMed/MEDLINE, PsycInfo, the Cochrane Library, and Excerpta Medica Database (EMBASE) and focused on SP and "neurodevelopmental antecedents" (operationalized in early risk factors, neuroimaging signs, neurological soft signs, and sensory responsivity). The time interval was from inception up to March 31, 2022. ***Results:*** From the search in electronic databases, 48 studies were retained and reviewed. SP are highly prevalent in OCD patients and overrepresented in comparison with healthy controls. Similarly, OCD patients also present a higher prevalence of early environmental adversities and sensorimotor alterations in terms of neurological soft signs and sensory over-responsivity in the tactile and acoustic domains; additional findings included hypogyrification signs at neuroimaging. Both sensorimotor alterations and SP are associated with tic-related manifestations and poorer insight in OCD patients. ***Conclusions:*** On the ground of established common subjective experience of SP and premorbid neurodevelopmental features, we hypothesized an explanatory model for OCD, which considers the possible pathophysiological role for altered corollary discharge and enhanced error detection in the neurodevelopment of SP and obsessions. SP may represent the subjective experiential resonance of an individual history of persistently inaccurate

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sensory predictions, whereas accompanying manifestations, such as the obsessive need for order and symmetry, may represent a compensatory attempt to mitigate SP. This neurodevelopmental-phenomenological bottom-up model, describing a dimensional gradient of sensorimotor alterations and related subjective experiences, may contribute to explain the dimensional affinity between OCD and schizophrenia spectrum disorders. Furthermore, this model could be useful for the early detection of subjects at higher risk of OCD.


## Introduction

The majority of theoretical models on obsessive compulsive disorder (OCD) are mostly top-down and focus on cognitive processes that may characterize OCD [1–3], whereas alternative, bottom-up approaches are far less represented in the contemporary scientific debate on OCD [4]. Furthermore, despite robust evidence on premorbid (i.e., neurodevelopmental antecedents) and prodromal features preceding the clinical onset [5], the ontogeny of OCD in youth is poorly investigated as compared to the massive attention to the cross-sectional study of OCD functioning.

In this paper, we provide an extended update on bottom-up theoretical models on the ontogeny of OCD [4, 6, 7], capitalizing on both neurodevelopmental and phenomenological levels of analysis. We recently proposed a similar approach for the ontogeny of the schizophrenic psychosis [8]. In this attempt, we hypothesized a pathophysiological role for early impairments in corollary discharges involved in sensory prediction, sensorimotor integration, and motor coordination [9–11]. The motor system continuously compares these internally predicted outcomes from self-generated actions with actual perceived outcomes and, if necessary, updates predictions based on received sensory feedbacks [12]. Being involved in the prediction of sensations from self-generated actions (and leading to sensory attenuation in case of congruent matching between sensory expectations and sensory consequences), corollary discharges contribute to the distinction between self-generated and externally generated actions; therefore, they are putatively involved in the emergence of an implicit awareness of being the voluntary agent of one's own actions [13–16].

Thus, from a neurocognitive perspective, corollary discharges may have a key physiological role in the ontogeny of embodiment, i.e., the implicit, immediate integration between conscious mental states and embodied correlates in terms of sense of agency (SoA) and Sense of ownership (SoO). In phenomenological terms, SoA corresponds to the tacit feeling of being the voluntary agent of one's own actions and SoO to the immanent feeling of mineness that accompanies our experiences [17–19].

We proposed a developmental pathogenic cascade from early childhood alterations of corollary discharge signals, endophenotypically expressed in childhood through motor dyscoordination, to distal psychotic symptoms as passivity delusions and auditory verbal hallucinations in adolescence or young adulthood, through intermediate phenomena represented by an altered SoA and anomalous subjective experiences of the basic self [20, 21]. Such experiences of self-alienation (e.g., feelings of derealization, perplexity, depersonalization, reduced self-presence, and alteration of the stream of thought), known as self-disorders, reflect a profound transformation of subjectivity antedating the onset of major clinical symptoms (i.e., positive, negative, disorganized), yet conferring a pervasive and painfully prolonged coloring to the entire experiential field [22, 23]. They meta-analytically co-aggregate in the schizophrenic spectrum, resulting in more pathognomonic than clinical symptoms [24].

The suitability of an analog progression for the ontogeny of OCD is suggested by certain affinities between OCD and schizophrenia spectrum disorders [25, 26], which involve epidemiological and symptomatological associations [27–30], partial polygenic overlap [31], and predisposing personality traits [32, 33]. In line with this aim, this paper provides a systematic and narrative review of two features of OCD, i.e., neurodevelopmental antecedents in the developmental anamnesis of OCD patients and sensory phenomena (SP), which could support a neurodevelopmental and phenomenological approach to the ontogeny of the OCD mind.

SP are disturbing feelings known as "just not right" experiences (hereafter NJRE; i.e., a subjective sense that something in the individual or in the world around is not as it should be) and feelings of incompleteness (hereafter FInc: i.e., a dissonant sense that one's actions, intentions, or experiences have not fully or properly been completed), which are increasingly acknowledged as highly prevalent in OCD patients [34–36]. The results of the systematic review are therefore combined in a neurodevelopmental and phenomenological model that attempt to explain 1) the ontogeny of the subjective experience leading to OCD symptoms in adolescents or young adults and 2) the affinity with the neurodevelopmental cascade hypothesized for schizophrenia [20, 21].

**Table 1.** Search results for features suggesting an altered neurodevelopment and for sensory phenomena in OCD patients

| Sub-domain | Studies identified (*n* = 48) | Key findings |
|---|---|---|
| Early risk factors | 4 | Increased prevalence of prenatal (i.e. during pregnancy) and perinatal hazards |
| Neuroimaging | 5 | Hypogyrification |
| Neurological soft signs | 19 | Increased prevalence in comparison with healthy controls, on both sides of the body and in multiple domains, associated with poorer insight, OCD severity, tics and psychosis risk |
| Sensory responsivity | 11 | Sensory over-responsivity in terms of enhanced startle reaction or impaired sensory gating, and in multiple sensory domains, as tactile and acoustic |
| Sensory phenomena | 9 | Increased prevalence of feelings of incompleteness and of not just right experiences |

## Methods

As this review aims at offering a focused overview of these two features of OCD (1. neurodevelopmental antecedents and features, 2. SP), we have screened the following scientific search engines for relevant literature: PubMed/MEDLINE, PsycInfo, the Cochrane Library, and Excerpta Medica database (EMBASE). Also, references lists of systematic reviews or meta-analyses on these topics were screened for additional references. This study has been set up according to the requirements of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [37, 38]. The time interval was from inception up to March 31, 2022. To optimize the literature search in databases, "neurodevelopmental antecedents" were operationalized through the subsequent key terms, matched with the term "OCD":

- "Early risk factors" as concerns potential triggers of an altered neurodevelopment
- "Neuroimaging" as concerns signs of an altered neurodevelopment (e.g., anomalous gyrification in adult OCD [39] or structural and/or functional alterations in pediatric OCD)
- "Neurological soft signs (NSS)" and "sensory responsivity" as concerns endophenotypic manifestations of an altered neurodevelopment in the sensorimotor domain [40, 41].

Sensory Phenomena (SP) were searched combining the term "OCD" AND "' SP" OR "NJRE" OR "FInc". The results of the searchers were inspected by two investigators with minimum 10 years of experience in the doing of systematic review and cross-checked by a third investigator expert in the field. The following criteria were applied to retain an article in the study, for both searches: the article was written in English; it included independent samples with people diagnosed as OCD based on a validated diagnostic procedure or help-seeking subjects presenting OCD features according to validated instruments. Moreover, for the first search we included only studies reporting features related to neurodevelopment, as indicated above; for the second search we included only cross-sectional studies aimed at investigating the prevalence of the subjective experience of OCD patients or of help-seeking individuals with OC features in terms of SP as NJRE or FInc; in light of these criteria, all studies assessing OC features in nonclinical samples were excluded. Finally, results of this search were exposed in a narrative review.

## Results

From the search in electronic databases, 48 studies were retained and reviewed (see online suppl. Fig. S1 for PRISMA procedure; for all online suppl. material, see www.karger.com/doi/10.1159/000526708 and Table 1 for results).

*Neurodevelopmental Antecedents*

4 studies reported that OCD patients present an increased prevalence of prenatal (i.e., during pregnancy) and perinatal hazards [42–45] indicating early environmental adversities that may trigger alterations in neurodevelopment. Five neuroimaging studies reported findings related to signs of an altered neurodevelopment or alterations in pediatric OCD. In particular, three studies described a neuroimaging sign of hypogyrification in adult OCD patients [46–48] and two studies described functional and structural alterations in children and adolescents OCD patients: one study reported significant clinical correlations between current age and age of onset of OCD and regional cerebral blood flow in the bilateral superior frontal and bilateral parietal cortical regions [49], and another study reported the involvement of genetic variants related to glutamatergic, dopaminergic, and neurodevelopmental pathways in determining the white matter microstructure of child and adolescent OCD patients [50].

19 studies with independent samples globally reported increased prevalence of NSS in comparison with healthy controls [51–69]. Studies up to 2013 were meta-analyzed in a study [64]: findings indicated large effect sizes (Hedges' $g = 1.27$, 95% confidence interval 0.80–1.75), suggesting that OCD patients have significantly higher rates of NSS than matched healthy controls on both sides of the body and in multiple domains (motor coordination, sensory integration, and primitive reflexes). After this meta-analysis, other 6 studies investigated NSS in OCD patients, reporting an association with poorer insight, OCD severity, and tics [65–70]. Finally, one study reported that poorer fine-motor and visuospatial skills in pediatric-onset OCD are predictive of OCD persistence in adulthood [70].

11 studies described a sensory over-responsivity in OCD patients [71–81], for example, in terms of enhanced startle reaction or impaired sensory gating, and in multiple sensory domains, such as tactile and acoustic.

*Sensory Phenomena*

Anomalous subjective experiences of OCD patients were formalized as SP in 2000 [34] and ante-litteram literature on SP published up to 2007 was systematically reviewed by a 2007 study [35]. This review found previous spurious and heterogeneous descriptions of anomalous subjective experiences in OCD patients that could be overall ascribed to SP. All those phenotypic features were more prominently associated with the tic-related OCD subtype and were more frequent in the early-onset OCD subtype.

Although the formalization of SP raised the interest on this topic, in the analysis of the empirical literature in electronic databases, we found several studies derived from the same sample and many studies performed non-clinical samples (typically undergraduate students). Indeed, most studies on SP derived from the same sample, mostly including Brazilian patients, that progressively increased along the years to the size of 1,366 patients. Several studies reported the association of SP with other clinical features such as tics and poorer insight [34–36, 82–87]; neuroimaging in a subsample revealed a structural gray matter volume increase in the left sensorimotor cortex in OCD patients with SP in the comparison to patients without SP and bilateral sensorimotor cortex gray matter volume increases in comparison to controls [85]. A recent factorial-network analysis of this sample [86] provided a significant step forward in the identification of a core structure below the phenotypic heterogeneity of obsessive compulsive disorder (OCD). Indeed, a dimension related to SP (i.e., FInc) constitutes a strong node in the network structure of OCD. Briefly, the authors reduced the heterogeneity of OCD (as covered and measured by the 87 distinct OCD symptoms of the Dimensional Yale-Brown Obsessive-Compulsive Scale) [87] through an exploratory and confirmatory factor analyses on a wide multinational cohort of children, adolescents, and adults diagnosed with OCD ($n = 1{,}366$); finally, interconnections between empirically-supported symptom dimensions were established using network analysis. Among the OCD dimensions emerging from factor analyses, the network analysis found FInc to be the most central dimension, i.e., with the most unique positive associations with other dimensions (except loss/separation and transformation) and was significantly more central than all other dimensions. On this empirical ground, the authors suggested that symptoms within the FInc dimension could represent a core phenotype in OCD. Such a dimension, consisting of three sub-dimensions (accuracy, perceptual phenomena, and NJRE), was strongly associated with a history of suicide attempts, psychosis spectrum disorder, and an earlier age at OCD symptom onset.

Few other cross-sectional studies investigated SP in independent clinical samples of OCD patients [88–95], reporting an increased prevalence or an association with core OCD symptoms. Main findings of these studies included the following:

- An increased prevalence of NJRE in OCD offspring in comparison with non-OCD offspring [91];
- NJRE as an important connecting link between specific OCD symptom dimensions, in particular ordering and checking, and accentuated traits OC personality disorder [92];
- A 22.8% prevalence of SP in terms of FInc in a wide sample of more than 300 OCD patients [93]; SP presence was associated with greater rates of symmetry/exactness obsessions and ordering/arranging compulsions and of comorbidity, poorer ratings of functioning, lower quality of life, and higher rates of unemployment and disability;
- A link between FInc and symmetry symptoms in pediatric OCD, and FInc being the feature most differentiating youth with OCD from those with social or generalized anxiety disorder [94];
- An association between SP in OCD patients and hyperactivation of the insula during a "body-focused" video task designed to elicit activation in sensorimotor brain regions [95].

## Discussion

*Neurodevelopmental-Sensory Phenomena Intertwine in OCD*

This review found a coherent pattern of empirical findings overall suggesting an altered neurodevelopment underlying the ontogeny of OCD manifestations. Preliminary supportive findings derive from neuroimaging (e.g., hypogyrification), while more robust findings involve a higher prevalence of 1) early environmental adversities that may trigger an altered neurodevelopment and 2) sensorimotor alterations in terms of NSS and of sensory over-responsivity (mostly investigated for tactile and acoustic modalities of stimulation). Interestingly, this pattern intertwines with empirical findings related to SP; indeed, both sensorimotor features and SP are associated with tic-related manifestations and poorer insight in OCD patients.

Given the crucial role of corollary discharges in sensorimotor integration [9–11], are these mechanisms somehow impaired also in OCD patients? Although less investigated than in schizophrenic patients [96], this hypothesis received preliminary support also in OCD patients: for example, an electroencephalographic study [97] measured the suppression of the N1 component of the event-related potential during active generation and passive observation of visual feedback. OCD patients showed decreased attenuation of sensory consequences of self-generated actions (i.e., reduced N1 suppression to actively generated feedback as compared with passively observed feedback). Furthermore, N1 component was not modulated by additional predictive motor cues (as observed in control subjects) and OCD patients reported enhanced ascriptions of agency experience, which correlated with the severity of SP (e.g., FInc). These findings suggested that OCD patients may partially fail to predict and suppress the sensory consequences of their own actions and the increased mismatch in sensory prediction between expected and actual outcomes may be involved in persistent FInc even after properly executed actions.

*Towards a Phenomenological Model*

OCD patients' descriptions indicate that SP are passively felt as sensorimotor, quasi-atmospheric perceptions perturbing the normal flow of consciousness. Similarly to obsessions, SP are felt by OCD patients with a reduced SoA [4, 6, 7], in particular, in relation to the feeling of agency, i.e., the pre-reflective, low-level, and non-conceptual feeling of control over one's own mental contents or actions and their consequences (in comparison with the conscious judgment of agency [13–16]). Overall, a variety of mental phenomena such as obsessive thoughts and SP precede, trigger, and accompany OCD compulsions (i.e., the ritualistic acts stereotypically performed to mitigate the distress engendered by obsessions). Considering that disturbing SP are related to the FInc [86], the core dimension of OCD seems to be characterized by a continuous alteration of subjectivity, with a flow of consciousness experienced and felt with a reduced SoA.

SP in OCD patients, such as NJRE and FInc, may represent the endpoint, in terms of subjective experiential resonance, of an individual history of persistent inaccurate sensory predictions, signaled by demonstrated sensory over-responsivity and NSS in OCD. Basing the ontogenetic emergence of SoA on the comparator model [14], the other needed piece in the mechanism at the basis of NJRE or FInc is the detection of the inaccuracy of sensory predictions. In this perspective, electrophysiological studies on OCD patients meta-analytically show a robust increase in comparison with healthy controls of the error-related negativity (ERN) [98], a frontocentral negative-going deflection following an erroneous response [99, 100]. Moreover, functional neuroimaging [101] and behavioral [102] evidence confirmed the presence in OCD patients of increased reward prediction errors, i.e., signals of a mismatch between expectations and outcomes driving reinforcement learning and goal-directed [103, 104].

Therefore, at least in the sensorimotor domain [11], the comparator model of OCD patients is inaccurate in predictions and too sensible in error detection; a persistence individual history of interaction with the surrounding world through this poorly calibrated comparator model may lead to the progressive subjective emergence of SP as NJRE and FInc. In a congruent perspective, it is possible to hypothesize that the obsessive need for order and symmetry (and the related ordering and arranging compulsions) could represent a compensatory attempt to mitigate SP by increasing the sensory predictability of the surrounding world, conferring it a rigidly structured spatial organization [7].

Such a phenomenological perspective points to a coherent view of the OCD subjective experience as an organized whole characterized by a predominant distortion of the SoA which does not fully saturate one's own mental phenomena, so that inner speech is felt in terms of intrusive (obsessive) thoughts and external or bodily somatosensory stimuli are felt in terms of SP. Therefore, it is possible to hypothesize that altered corollary discharges preliminary detected also in OCD patients [97] could be a plausible key pathophysiological mechanism, early ex-

pressed phenotypically by NSS and sensory over-responsivity, facilitating the emergence of the OCD symptoms along developmental years, especially if accompanied and matched with a too sensible system of error detection [98]. This would suggest that early severe motor deviances in childhood are a potential warning flag for an altered neurodevelopment headed to an obsessive mind.

Overall, this neurodevelopmental and phenomenological hypothesized account could provide a bottom-up perspective on the ontogeny of the obsessive mind, not fully in agreement with top-down cognitive models of adult OCD, more focused on how an obsessive mind works rather than how a mind may become obsessive [2, 3]. This partial neglect of the developmental component of OCD is surprising. Indeed, beyond the above described signs in the sensory domain (NSS and sensory over-responsivity), there is also meta-analytical evidence on neurocognitive endophenotypes in pediatric OCD emerging from experimental studies, for example, related to action monitoring [5].

The pheno-developmental model presented in this paper presents some affinities with recent models of OCD. Indeed, the reduced SoA ontogenetically emerging from an individual history of altered sensorimotor prediction due to altered CD mechanisms, matched with enhanced error detection, has some affinities with the core of a recent computational model of OCD [105], that is, an excessive uncertainty regarding state transitions, especially action-dependent transition. In this account, patients experience excessive uncertainty in the probabilistic predictive relationship between actions and their consequences, triggering a possible cascade of subjective and behavioral consequences that may longitudinally structure an OCD mind.

Moreover, our model has also affinities with the OCD model based on reduced SoA proposed by Szalai [4]. This model proposed that in OC patients, aberrant motor experiences and related NJRE and FInc of an action not reaching its goal prompt corrective repetition until the experience of the act being executed in the correct way is achieved. The adjustment nature of action repetition is mostly unconscious and the magical thinking regarding the power of certain acts, coupled with a sense of imperfect execution of these, may lead to a rationalization of acts in terms of rituals. This coheres with empirical findings of a study that employed video-telemetry to analyze 39 motor OCD rituals: the authors found that most OCD rituals, up to 75%, comprised a "tail," which is a section that follows the functional end of the task that the patients ascribed to their activity [106]. In this perspective, both sensorimotor deficits and dysfunctional beliefs (e.g., magical thinking, beliefs about personal responsibility) feed into the agent's motivation and self-understanding in action. Therefore, in such model, the reflective Self operates in a kind of rationalization of thoughts and behaviors triggered by sensorimotor alterations, resulting in a maintaining rather than a causal factor.

In comparison of both these models, our model gives more emphasis to clinical signs and endophenotypic features (in the sensorimotor domain) that are detectable along developmental years. Therefore, our model, even if probably more simplistic in the description of intertwined pathogenic mechanisms leading to habits, cognitive rationalizations, and overt and covert compulsions starting from an altered comparator model, could have more translational potentialities in the direction of an early detection of OCD risk.

*OCD-Psychosis Affinities*

The integration of phenomenological and neurodevelopmental perspectives delineated above on the ontogeny of OCD symptoms could contribute to explain the association of FInc with early-onset OCD and with psychotic manifestations [86]. Indeed, as reported in the introduction, a pathogenic cascade partly echoing the putative one of OCD has been proposed for the neurodevelopment of the psychotic mind [20, 21]. Early alterations in corollary discharges, endophenotypically signaled by childhood dyspraxia [107] (that is predictive of psychosis in offspring of schizophrenic patients [108]), may longitudinally facilitate the structuration of a schizophrenic vulnerability characterized by reduced feeling of agency and SD, representing the experiential basis for the development of clinical psychotic symptoms (the schizotaxic Self [109]).

The hypothesis of a neurodevelopmentally determined dimensional gradient of altered sensorimotor predictions may offer a pathophysiological bridge between OCD and psychosis, which are epidemiologically associated above chance [27–30]; such hypothesis is substantially confirmed by a higher severity of NSS in OCD patients with psychosis [61] and in OCD patients with poor insight [63]. Moreover, a trait-like feature of the schizophrenia spectrum such as schizotypy is higher in OCD patients in comparison with health controls [110], associated with obsessions rather than compulsions [111] and with NSS such as altered smooth pursuit of smooth movements [33]. According to this hypothesized perspective, schizophrenia spectrum disorders and OCD can be described along a neurodevelopmental continuum characterized by a dimensional gradient of sensorimotor deviances, with schizotypy representing a dimensional bridge between

these two categorically distinct disorders. At the same time, the long-term subjective effects of an individual's history of altered sensorimotor predictions, are presumably not reducible to all-or-none (i.e., categorical) phenomena, but could rather be described along a phenomenological continuum [112], modulated by associated characteristics of error detection (Table 2).

Milder alterations of sensorimotor predictions (and related attenuation of the feeling of agency and preservation of the feeling of mineness), associated with an enhanced (i.e., too sensible) mechanism of error detection [98], may be subjectively felt in terms of NJRE and FInc, representing an enabling condition for the development of the obsessive mind and its covert and overt compulsive rituals. More severe alterations of sensorimotor predictions, associated with a weak (i.e., poorly sensible) mechanism of error detection [113–116] and longitudinally conditioning the development of both altered feelings of agency and ownership, may be subjectively felt in terms of SD, representing an enabling condition for the development of the psychotic mind. In line with the hypothesis, a recent study reported a specific deficit in internal source monitoring (i.e., the ability to distinguish whether an internally generated event was expressed in the outer vs. inner space) in OCD patients, while an additive deficit in reality monitoring (i.e., the ability to determine whether information is perceived from environment or imagined) was present only in schizophrenic patients, suggesting a preservation of the SoO in OCD patients [117].

Such a developmental gradient may contribute to explaining the overlap between OCD and psychosis, in both terms of comorbidity and partial symptom affinity. Moreover, it coheres with established psychopathological transition sequences that lead to end-stage psychotic symptoms from more elementary transformations of the experiential field and, particularly, of the subjective experience of the stream of thought (e.g., thought block, thought intrusions, thought interferences, ruminations) [118]. For example, basic disturbances of the stream of thought have been described as intermediate experiential changes in the sequence of transition to auditory verbal hallucinations. Therefore, clinicians should be aware of the possibility that, along the psychopathological construction of psychotic states from early prodromal/clinically high-risk states, patients could feel and acknowledge the presence of disturbances in their stream of thought that might resemble OCD manifestations [119–121]. This is even more important when potential compatibility with OCD diagnosis is contextual with early-onset of symptoms and premorbid manifestations of putative neurobiological vulnerability [122].

**Table 2.** Cascade levels from the neural to the symptomatic one in OCD and schizophrenia spectrum disorder

| Level of analysis | Obsessive compulsive disorder | Schizophrenia spectrum disorders |
|---|---|---|
| Neural | Milder inaccuracy in sensory predictions | More severe inaccuracy in sensory predictions |
| | Enhanced ERN | Attenuated ERN |
| Endophenotypic | Milder NSS and sensory over-responsivity | Severe NSS |
| Phenomenological | Preserved sense of ownership and reduced sense of agency | Reduced sense of ownership and reduced sense of agency |
| | Sensory phenomena: NJRE and FInc | Self-disorders |
| Implicit Goal | Reduction of experiential dissonance: mitigation of NJRE and FInc | Maximization of sensory predictability |
| Symptomatic/clinical level | Repetitive behaviors and rituals | Need for order and symmetry | Hyper-reflexivity |

ERN, error-related negativity; FInc, feeling of incompleteness; NJRE, not just right experiences; NSS, neurological soft signs; OCD, obsessive compulsive disorder.

## Conclusions

We presented an integration of neurodevelopmental and phenomenological perspectives into a bottom-up model of OCD. In comparison with mainstream top-down cognitive models, this model focuses on the ontogeny of OCD and its putative endophenotypic manifestations:

- At the neurodevelopmental level, through premorbid and prodromal stages, especially in the sensorimotor domain, due to basic alterations of corollary discharge mechanisms;
- At the phenomenological level, through a progressively emergent conscious resonance of such alterations in the implicit level of functioning, in terms of NJRE and FInc;
- At the behavioral level, through the delineation of some compulsions as attempts to reduce FInc and more in general SP.

Being anchored to developmental manifestations that could appear in the premorbid stage (i.e., unrelated to strict OCD symptoms), this model could be useful for an early detection of subjects at higher risk of OCD manifestations [123–126]. Moreover, the integration of clinical data with a phenomenological perspective may add depth, nuance, and utility to clinical phenotyping [9, 127], also in the field of OCD, contributing to the differential diagnosis with partially overlapping conditions (as schizophrenia spectrum conditions).

## Statement of Ethics

The study is a review and only reports research already published. It does not include new data from humans or animals.

## Conflict of Interest Statement

The authors have no conflicts of interest to declare.


## Funding Sources

The authors have not received any funding.


## Author Contributions

Michele Poletti performed the systematic review, wrote the first draft and approved the final version of the manuscript. Eva Gebhardt and Lorenzo Pelizza wrote the first draft and approved the final version of the manuscript. Antonio Preti and Andrea Raballo performed the systematic review, critically revised the paper and approved the final version of the manuscript.

## Data Availability Statement

This research is a review article. All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

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