NÖRO PSİKİYATRİ ARŞİVİ
ARCHIVES OF NEUROPSYCHIATRY

REVIEW

Arch Neuropsychiatry 2022; 59: (Supplement 1): S50–S56
https://doi.org/10.29399/npa.28151

Obsessive-Compulsive Disorder from an Embodied Cognition Perspective

Orhan Murat Koçak, Selvi Ceran, Pelin Kutlutürk Üney, Ceyhun Hacıyev

Başkent University Faculty of Medicine, Department of Psychiatry, Ankara, Turkey

ABSTRACT

Obsessive Compulsive Disorder (OCD) is characterized by problems of control over behavior and cognition. Although almost all of the studies on pathogenesis of OCD point out fronto-striatal dysfunction, it is still not possible to reveal mechanisms to explain the entire clinical course of OCD through these circuits. A more holistic explanation can be given through the Embodied Cognition (EC) perspective, which suggests that the alteration/dysfunction of low-level sensory-motor process may appear as a multifarious extent of dysfunction of high-level cognitive processes. Fronto-striatal circuits play fundamental role in behavioral control. These circuits also have a central role for the feed-forward motor control (FFMC). In FFMC, the internal model of movement is driven by efference copies as templates for motor behavior, without being adjusted by sensory information. If impairment of low-level sensory-motor processing is crucial to occurrence of compulsions, one possible hypothesis about this impairment is the problem which emerges from occurrence of efference copy in FFMC. On the other hand, the efference copy has also pivotal role for subject's feeling of the agency of an action. Therefore, there may be role of failure in successfully reproduction of the efference copy in the background of subjects' experience of losing control on compulsive behaviors. In this paper, we will discuss how the embodied cognition (EC) perspective which can be one of the biological bases of computationalism, which brings neuroscientific explanations on the functioning of nervous system to a more symbolic perspective, may contribute to our understanding of etiopathogenesis of OCD. In this perspective, our method will be to integrate the theoretical basis provided by EC perspective to the current models for OCD, rather than falsifying them.

Keywords: Anterior cingulate cortex, efference copy, embodied cognition, obsessive-compulsive disorder, orbitofrontal cortex

Cite this article as: Koçak OM, Ceran S, Kutlutürk Üney P. Obsessive-Compulsive Disorder from an Embodied Cognition Perspective. Arch Neuropsychiatry 2022; 59: (Supplement 1):S50–S56.

INTRODUCTION

Obsessive compulsive disorder (OCD) is a mental disorder the main symptoms of which are obsessions and compulsions and which can progress with severe disability. While obsessions are identified as repetitive and intrusive thoughts, impulses, or images, which can cause significant distress or anxiety in the person, which cannot be prevented despite all the efforts of the person; compulsions, on the other hand, are repetitive movements or mental acts that the person feels compelled to do, often associated with the distress caused by the obsession. Many compulsions are quantitatively exaggerated forms of activities of daily life, such as washing hands and locking doors. Although intended to neutralize the anxiety created by the obsession, compulsions are either unrealistically associated with the situations they are designed to neutralize/protect, or are clearly excessive. In the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM 5), of the American Psychiatric Association, a diagnosis of OCD requires the presence of obsessions and/or compulsions, and these symptoms should keep the person occupied for a significant part of the day and cause a significant impairment in functionality. Additionally, obsessions and compulsions should not be related to another mental or medical illness or substance use (1). The prevalence of OCD, which is considered to be among the most common mental disorders, has been reported as 1%-3% in different studies (2, 3). Although the effectiveness of antidepressants, especially serotonin reuptake inhibitors, and cognitive behavioral therapies have been demonstrated in the treatment, the fact that 40%-60% of patients do not benefit from the treatment sufficiently increases their curiosity about the neurobiology of OCD (4–6).

Highlights

- **Dysfunctional fronto-striatal circuits has been consistently shown in OCD.**
- **Embodied cognition emphasizes relationship of sensori-motor processes with cognition.**
- **This theory can fill the gaps left by fronto-striatal dysfunction in OCD.**

Many models has been proposed to explain the neurobiology of OCD. Although all of these models have an important place in explaining the etiopathogenesis of OCD, in terms of current findings and clinical presentation, every theoretical approach based on the updated and expanding literature and enriched information has remained far from explaining the whole puzzle, even made the puzzle more complex. In this context, there is still a need to integrate the existing literature and clinics with a more holistic approach to explain the etiopathogenesis of OCD.

The idea of computationalism draws neuroscientific explanations of the functioning of the nervous system into a more symbolic perspective.

Correspondence Address: Orhan Murat Koçak, Baskent University Faculty of Medicine, Department of Psychiatry, Taşkent Cad. Şht. H. Temel Kuğuoğlu Sokak No: 30, 06490 Bahçelievler, Ankara, Turkey • **E-mail:** orhanmuratkocak@gmail.com
Received: 07.03.2022, **Accepted:** 27.05.2022, **Available Online Date:** 16.12.2022

The view that the 'embodied cognition' (EC) approach, which is on the biological basis of the idea of computationalism, can provide a significant advantage in illuminating the etiopathogenesis of OCD will be discussed in this article.

OCD NEUROBIOLOGY

OCD Neurobiology and Dysfunction in Fronto-Striatal Circuits

Models to explain the pathophysiology of OCD have mostly emphasized the role of abnormalities in the fronto-striatal circuits. In both structural and functional imaging studies, findings supporting the deterioration of these cycles have been obtained (7). In studies with task-dependent functional magnetic resonance imaging (fMRI), activity changes in corticostriatal circuits have been demonstrated with various paradigms and during symptom provocation (8). However, when we look at the literature, in correlation with methodological differences, it has been determined that many regions such as the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), dorsolateral prefrontal cortex (DLPFC), limbic system, amygdalo-cortical circuits, thalamus, caudate, putamen, supplementary motor area, and frontal eye area exhibit functional differences in OCD (9–12).

In support of these findings, numerous studies have also reported differences in resting-state functional connectivity for many of the above-mentioned regions (11). There are conflicting findings not only for regions but also in terms of increased or decreased activity and connectivity. Moreover, there are studies that did not detect the functional changes. Although the findings were inconsistent, a meta-analysis reported that OFC, ACC, and caudate dysfunctions are prominent (13).

There are studies which support dysfunction in fronto-striatal circuits beyond functional imaging. Methodologically, event-related potential studies, studies using magnetoencephalography, and even structural MRI studies exhibit accordance with fMRI studies. Comorbid OCD symptoms in neurodegenerative diseases with basal ganglia involvement such as Parkinson's and Huntington's and in lesions due to caudate nucleus infarction have clinical evidence that supports the role of frontostriatal circuits (14, 15). Similarly, in PANDAS (pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections) where autoimmune mechanisms are responsible for OCD symptoms observed in childhood, antibodies formed against group A beta-hemolytic streptococcal bacterial antigens are thought to cross-react with neuronal tissues in the basal ganglia (16).

In addition, it is possible to reach the same conclusion with imaging studies showing that activity in the striatum and related cortical regions decreases after pharmacological treatment (serotonin reuptake inhibitors) and cognitive behavioral therapy (17).

Finally, it is possible to draw attention to the role of the fronto-striatal circuits in the pathogenesis of OCD through the effectiveness of separating the frontal cortex-basal ganglia connections from each other with less frequently used surgical treatments such as capsulotomy or cingulotomy (18). What was particularly emphasized at the beginning regarding the role of fronto-striatal circuits in OCD is the disruption of the balance between direct and indirect pathways in these circuits. The 'direct' and 'indirect' pathways are thought to work antagonistically through gamma-aminobutyric acid (GABA) and glutamate neurotransmission to modulate thalamic and cortical activation. While the direct way ensures the initiation and maintenance of behavior through disinhibition of the thalamus and excitation of the cortex, indirect way contributes to the inhibition and modification of behavior by reducing the excitation of the cortex through the inhibition of the thalamus (19).

Roles of Fronto-Striatall Circuits in Cognitive Processes

Different fronto-striatal loops associated with the pathogenesis of OCD, each originating from specific frontal-cortical areas and associated with emotional, cognitive, motivational, or sensory-motor inputs, have been described up to the present. These neuronal circuits originate from the cortex, pass through the basal ganglia and extend to the thalamus with a final projection through either by 'direct' or 'indirect' pathways; from there, it connects again with the cortical region from which they originate, thus forming a loop. The first one, affective circuit, connects the OFC, with the nucleus accumbens, and thalamus; responsible for affective and reward-related processes. This loop plays a role in the selection of behavior and the evaluation of reward and error. The dorsal cognitive circuit connects the DLPFC, the caudate nucleus, and the thalamus; responsible for executive functioning like set-shifting, also planning emotional regulation. The ventral cognitive circuit provides the connection between the inferior frontal gyrus, the ventrolateral prefrontal cortex (VLPFC), the ventral caudate, and the thalamus, and is involved in motor preparation and response inhibition. The sensory-motor loop connects the premotor cortex, putamen, and thalamus. It plays a role in the initiation and control of motor behavior and integrates the necessary sensory information for this. It mediates the automatic response and therefore the shift from goal-directed behavior to habitual behavior. This increases stimulus response, such as habitual behavior. These parallel loops are interconnected and are thought to conduct complex behaviors together. Other circuits such as fronto-limbic (includes the ventromedial PFC and amygdala and is responsible for emotional responses such as anxiety and fear), frontoparietal, and cerebellar circuits play a role in the pathogenesis of OCD, apart from the fronto-striatal circuits (20, 21) (Figure 1).

Information from clinical and preclinical studies highlights the central role of the integrity of the fronto-striatal circuits and basal ganglia in providing the affective, cognitive, and motor flexibility necessary for goal-directed behavior. The direct path facilitates the behavior and the indirect path inhibits unrelated effects/actions. Additionally, there are two motor behavior control modes: feed-forward and feed-back motor control (22). In feed-back motor control, sensory information directly guides the action, and the action is carried out accordingly. In feed-forward motor control, the efference copy which is the template for motor behavior

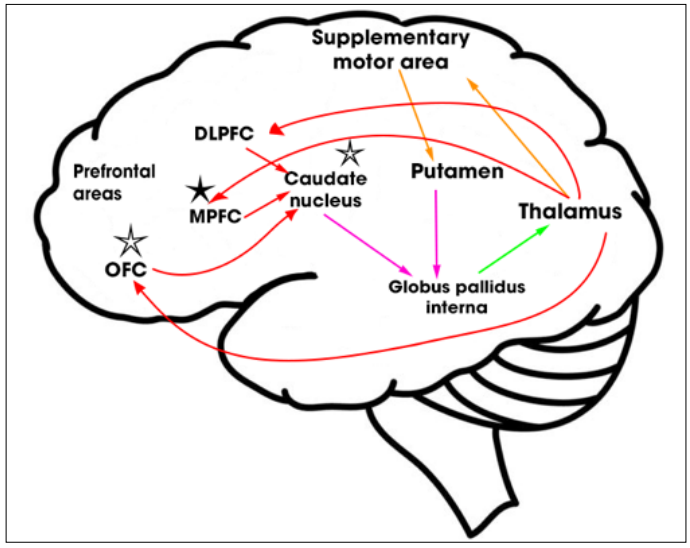

Figure 1. Schematic figure of the fronto-striatal circuits. The pathway that starts from each frontal region (DLPFC: dorsolateral prefrontal cortex, OFC: orbitofrontal cortex, MPFC: medial prefrontal cortex) forms a loop that returns to this point. Open-inside stars indicate regions where there is more evidence of its role in the etiopathogenesis of OCD. In the medial prefrontal cortex, especially the anterior cingulate cortex, may play a more important role (solid star).

without undergoing corrections generated by sensory information directly guides the internal model of action (23). These copies contain multi-sensory information related to the action (deep sense, joint position sense, visual and auditory sensory information), which also represents possible consequences of the action. It is assumed that daily actions that do not require high accuracy are carried out via a feed-forward motor control. In feed-forward motor control, sensory feed-back is not directly compared with the goal, but to it is compared with internal copies. The basal ganglia are the main regions where internal copies are represented. Additionally, motor association areas (frontal eye area, supplementary motor area and premotor area) are also involved in the representation of these copies.

Habit-Learning Theory in OCDs

The dominant view in the last 20 years in the development of compulsion is the excessive habit-learning theory, which claims an imbalance between goal-directed behavior and the habit-learning system in favor of the latter (24). Habits are rapidly occurring, automatic behavior that emerge with stimuli rather than carefully conceived responses to current demands and needs. Habits develop as they are practiced in the daily routine. Thus, we can perform many behaviors such as cooking, dressing and bathing in our daily practices without any need for concentration by combining action sequences (for example, hand washing consists of the actions as turning on the tap, soaping, rinsing, closing the tap, drying, etc.) (25).

It is proposed that the connections of the putamen with the premotor cortex are important in the development of habitual behavior. Goal-directed behavior, in contrast, considers organizing and updating according to outputs, not just impulses, to meet ever-changing needs in the best possible way, so they are slower and subject to executive control of the PFC. OFC and caudate nucleus are thought to play an important role in considering the consequences of behavior and using motivation, and therefore in the formation of flexible, goal-directed behavior.

The most important advantages of automatic behaviors are that they can be performed without the need for high-level cognitive processing. Automatic behavior gives the ability to multitask simultaneously, think while acting and learn more detailed tasks (for example, driving) by combining simple habits. However, habits also have some disadvantages. They function as a cognitive shortcut and therefore are also prone to errors or slips between habits, such as automatically typing an older automatic teller machine (ATM) password instead of the current one. Therefore, it is proposed that excessive habituation, which almost gains autonomy and deviates from the real goals and intentions of the individual, plays a key role in OCD.

Theorists who proposed that excessive habit formation may affect the formation of compulsion in OCD have based their opinion on three main reasons. The first is, although compulsion have negative clinical consequences, just like habits, they are rigid and repetitive and are experienced 'out of control' by an individual with OCD. The second is that fronto-striatal loops are involved in both compulsion and habit formation, habit expression, and 'goal-directed' habit control. The third reason is the clinical observation that the substance use behavior becomes 'compulsive' with the effect of continuous reinforcement over time. This situation resembles habits because it continues even after it loses its reinforcing effect. Therefore, it is proposed that the habit system may play a role in the formation or at least the maintenance of compulsions (26).

Fronto-striatal circuits play an important role in establishing the balance between habit learning and goal-directed behavior. OFC is the region responsible for attributing value to the consequences of a behavior. The

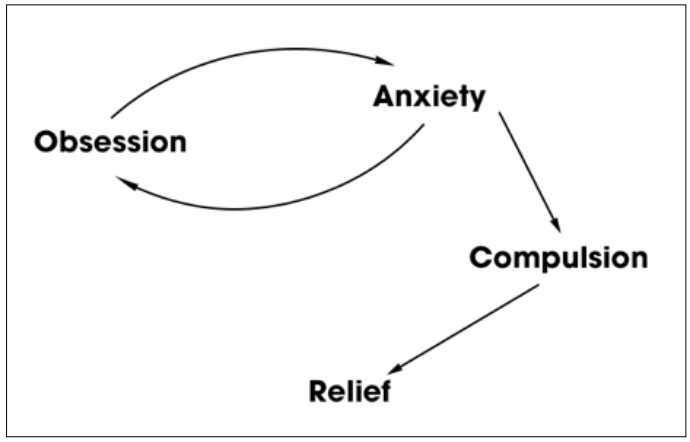

Figure 2. Model of the clinical situation of obsessive-compulsive disorder.

OFC is responsible for reinforcing behavior by determining its positive attribution. This can lead individuals with OCD to exaggerate the importance of the consequences of compulsive behavior and to prolong the effort towards the 'feeling of completion'. The relief that could be felt in parallel with the feeling of completion may become a positive reinforcer for the effort to complete the compulsion (27) (Figure 2).

Meanwhile, the ACC may have a disruptive effect on the completion of the action. It plays an important role in action, conflict, and error monitoring. A marker of these cognitive processes is "error-related negativity" (ERN) which is an event-related potential obtained from electroencephalogram (EEG) (28). It has been reported that, the ERN, which is thought to originate from ACC, accompanies 'not just right experiences' (29). Therefore, ACC hyperactivity can play a role in the inability to complete the compulsion (30). As a result, an increased basal ganglia activity can also serve to create this new form (this form is constituted from more repetition of washing, checking, etc.) of the behavior as a habit, even if it has deviated from its purpose (instead of aiming cleanness or safety, being relieved would be the aim). This whole pathophysiologic process of OCD has not been clearly proven, but is robustly supported by the findings of the vast majority of studies.

A Critical Look at the Theory of Habit Learning

A theory regarding the etiopathogenesis of OCD is expected to explain the neuropsychological findings found in almost all cognitive domains (31). These findings include impaired set shifting, increased action monitoring, over-investment in relaxation, impaired planning and executive functions.

Persistent and repetitive symptoms in OCD have been associated with impaired cognitive flexibility. Accordingly, it has been suggested that the inability to terminate thought or behavior even though it does not have any functionality may be associated with impaired flexibility in changing attention from one stimulus to another. A way to measure cognitive flexibility is to determine the 'set-shifting'. Set-shifting is the ability to shift attention from one stimulus dimension (e.g. color) to another dimension (e.g. shape). In a resting state FMRI study, this cognitive inflexibility was associated with decreased connectivity between the striatum and DLPFC (32). It has been shown that set shifting is impaired in both patients diagnosed with OCD and their relatives who do not meet the OCD diagnosis. In the pathophysiology of OCD, the conflict between the result of the action and the internal representation and purpose of this action has been associated with dysfunctional action monitoring (26) .

In the theory of excessive habit learning, it is generally emphasized that behavior that has not been in the habit repertoire would evolve into a

habit. It is reasonable to expect that uncontrolled circuits related to habit learning will also play a role in excessive habit learning. However, in OCD, acquired habit is mostly behavioral change, an increase in the frequency of behavior in the subject's habit repertoire. It is expected that there will be another system which will shape the transformation of acquired habits.

In addition to the aforementioned discordance of excessive habit learning with the phenomenology of compulsions, there are regions such as the posterior parietal, anterior insula, and supplementary motor area in OCD, which are frequently emphasized in the literature, apart from the central regions in explaining the etiopathogenesis of the disorder via excessive habit learning. It is becoming more and more difficult to obtain an explanation that includes the possible roles of these regions (33). For example, the posterior parietal cortex (PPC) involves many cognitive processes as a part of the multimodal association area. Among these, attention-centered executive processes are especially important and studies demonstrated their dysfunction in OCD. However, its exact role has not been revealed in the current etiopathogenetic explanations. The anterior insula plays a central role in the integration of physiological processes with cognition. As such, its clear role in the pathogenesis of OCD, as with PCC, has not been clarified, although it has been hypothesized to play a role in sensory phenomena (34,35). Therefore, just as the difficulty of a puzzle is proportional to the number of pieces that need to be put together, the vast accumulation of findings in the literature points to the difficulty of solving the etiopathogenesis of OCD.

EMBODIED COGNITION

Cognition is embodied when it is deeply connected to the characteristics of the physical body, that is, it becomes concrete when aspects of the body beyond the brain play a causal and physically constructive role in cognitive processing (36). According to the embodied cognition theory (also the terms grounded cognition, situation models and internal models intercept with each other and with embodied cognition conceptually), sensorial and motor functions that control and direct our motor, autonomic and sensorial processes of the body are used in cognition and social interaction. Contrary to the dualist approach, embodied cognition approach proposes the assumption that the neural infrastructure, which has been shown to specialize in sensory-motor functions, is also responsible for producing cognition. Embodied cognition approach also claims that cognitive processes result from having a "body" with certain sensory-motor abilities that interact with the environment. Linguist George Lakoff and philosopher Mark Johnson, for example, suggest that abstract concepts can be metaphors for physical and bodily concepts. According to Johnson, human understanding and rationality are outcomes associated with the embodied experiences of existing in the world. A good example of this can be given through language. Linguistically speaking, the concepts of up/down or good/bad seem to be related to gravity, the day-night cycle, and the sun being over the head. The mind, too, is formed as a product of the uninterrupted interaction between the body and the world (37). Therefore, what is hidden-dangerous-uncertain contains neural representations that are associated with the night/darkness and lower/underground.

In other words, the basic assumption of the embodied cognition approach is that the scope and character of an organism's experiences, and therefore of the concepts the organism has, is determined by the body. The relationship between the body and the world that the body is within also shapes the the body's relationship with cognition. In summary, the idea of grounded cognition, which can be evaluated within the conceptual framework of embodied cognition, accepts two main assumptions: cognition includes bodily interactions with the world and these interactions are represented in the brain. (38).

Sensory schemas corresponding to bodily interactions with the world are activated during the processing of abstract conceptual information. For example, when processing abstract concepts such as "power" or "god", a spatial image scheme of the concept is activated (39). Embodied cognition approach proposes that representations of abstract concepts are stored in the brain's "experiential system" by sensory-motor, proprioceptive, introceptive, and emotional structures. The bodily experiences represented in these systems are then reconstructed or "simulated" during the development of cognition. For example, judgments of importance are affected by the manipulation of weight, and heavier items are perceived to be more important than lighter ones (40). Similarly, it has been reported that the neural anatomical structures behind the mental representation of features related to an individual's position in the social hierarchy, such as dignity and efficiency, overlap with the regions (parietal lobe) involved in the comparison of the size of numbers (41).

Evidence for the idea that concepts are grounded in the fields of action and perception comes from neuro-imaging studies. Studies have shown that activation of the motor regions of the brain is accompanied by activated language-related brain regions during production and comprehension of action-oriented words (verbs), sentences and concepts (42,43). Similarly, a meta-analysis conducted in 2020 shows that brain circuits which are activated by reading action words are similar to circuits activated by watching action-related images (44).

UNDERSTANDING OCD FROM EMBODIED COGNITION PERSPECTIVE

As previously mentioned, according to embodied cognition theory, higher-level cognitive processes are based on the sensory and motor experiences of the organism (38, 45). The theory states that the primer and unimodal association regions responsible for motor and sensorial functions that take part in processes such as language, planning, decision-making, and judgment. For example, it has been shown in the literature that the sensory-motor system plays a role in creating metaphors, mental imagery, understanding the subject of the action (agency), and understanding and producing verbs and nouns (46, 47). Thus, a problem in the sensory-motor control of actions may cause dysfunctions in processes such as "disinhibition", "habit learning", "planning", "set shifting", and "action monitoring", which is also suggested in OCD phenomenology.

In the OCD literature, there are few studies which have tested the idea that impairment in low-level cognitive processes may also cause impairment in higher-level cognitive functions. For example, this idea was supported by Gentsch et al. (2012), who reported that feed-forward motor control is defective in OCD (48). In this study, they demonstrated that the relationship between motor response to a stimulus and subsequent agency experience was impaired in the OCD group and this was associated with impaired feed-forward motor control. They reported that the control of action occurs through the efference copy, which also shapes the attribution of being the agent of that action. This is also important in terms of revealing the role of low-level processes in patients with OCD who take on excessive responsibility. For further support, in the study by Rounis et al. (2016), it was observed that patients with OCD had motor deficiencies in extremity control compared with participants without OCD (49). Additionally, it has been reported that sensory phenomena experiences, which are frequently observed in patients with OCD, are associated with an increase in gray matter volume in the left sensory-motor areas (50).

Sensory phenomenon is a clinical condition that includes bodily sensations, a feeling of inner tension, an impression of "not just right", and emotions including incompleteness. In particular, the incompleteness and "not just right" experiences are more frequent in OCD (51).

The frequently observed need for completion and sensory phenomena in OCD and the accompaniment of difficulty in motor control with the difficulty of control of behavior together renders suggesting that OCD etiopathogenesis could be conceived from an EC perspective rational. Problems in low-level sensory-motor processes may be among the underlying factors for the state of incompleteness, set-shifting problems, difficulty in planning, and lack of flexibility because the prediction of outcome is important in all executive processes and it is expected that these predictions can be either kept in working memory (WM) or can be easily retrieved.

Two basic models for the control of action have been defined. These are feed-forward and feed-backward motor control. Feed-forward motor control is a control system in which information about the action is not updated according to sensory feedback in the execution of an action and its efference copy manages the execution of the action as a template. The efference copy or corollary discharge is the reproduction of the ideal state of action by structures that manage sensory and motor processes (52). The efference copy is also assumed to be a clear representation of the predicted sensory consequences of the action (52). Thus, representations of the response were activated just before the motor response occurred. Glutamatergic cortical neurons which are thought to encode the efference copy synapse on the indirect pathway's striatal medium-sized spiny neurons (MSN) on which the GABAergic dopamine 2 receptors are located (53-55) (Figure 3). It has been proposed that dopaminergic overstimulation here inhibits the activity of the indirect pathway (56). This may play a role in the disruption of the efference copy formation (56).

One of the electrophysiological markers for the formation of internal copy in studies is a phenomenon called motor-induced suppression (MIS) of sensory cortical feedback (57), in which the sensory responses to stimuli triggered by self-initiated motor movement are suppressed compared to the responses to the same externally triggered stimuli. This mechanism is associated with the fact that results of the motor response overlap with the efference copy when the process is executed in accordance with the expectation. This overlap ends with the suppression of the representation of sensory information by the efference copy. A frequently used method for observing the MIS phenomenon in studies is to determine, in the sensory regions, the difference in the evoked potential (N100 wave) amplitude between active and passive listening (58). The logic is that in situations where sensory information is expected (well-estimated), the activity of the motor response suppresses the sensory information (i.e. suppresses it during active listening) (58). However, Niziolek et al. (2013) suggested that less prototypical speech (when the produced speech is more distorted than expected) makes it difficult to estimate correctly, therefore, suppression through the efference copy reflects a sensory target, not a sensory prediction (52). In both cases, the function of efference copy is evident, during the execution of action under the feed-forward motor control.

Considering these factors, it seems appropriate to think that an impairment in the feed-forward motor control associated with the formation of the efference copy, which plays an important role in the regulation of habitual behavior, plays an important role in the emergence of compulsion. It is reasonable that the regions responsible for motor control, including sensorial and motor regions, basal ganglia and even brainstem, would also make it easier to theorize a consistent explanation of the endophenotypic features that comprise the vastly varied neuropsychological findings observed in OCD. The last point to be emphasized in relation to this is that the efference copy can also be important in terms of representing the action's state of being under the control of the individual, who is subject of the action. In other words, the inability to control compulsive actions may result from the failure in establishing the efferent copy (Figure 4).

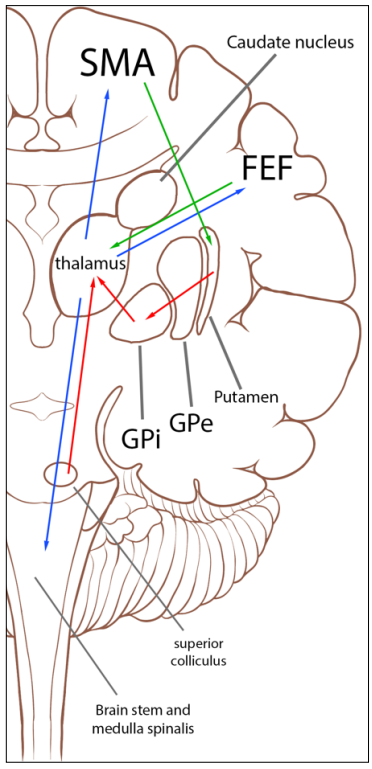

Figure 3. Brain regions involved in the representation of efference copy. Information about the efference copy which occurred in the supplementary motor area (SMA), the frontal eye field (FEF), and the globus pallidus interna (GPi) has been sent to the brain stem via the thalamus (dark blue arrow). Regions responsible for the movement of eyes are involved in the formation of the motion-related saccadic eye movements. The red arrows represent inhibitory and the green arrows represent excitatory connections. (GPe: The globus pallidus externa)

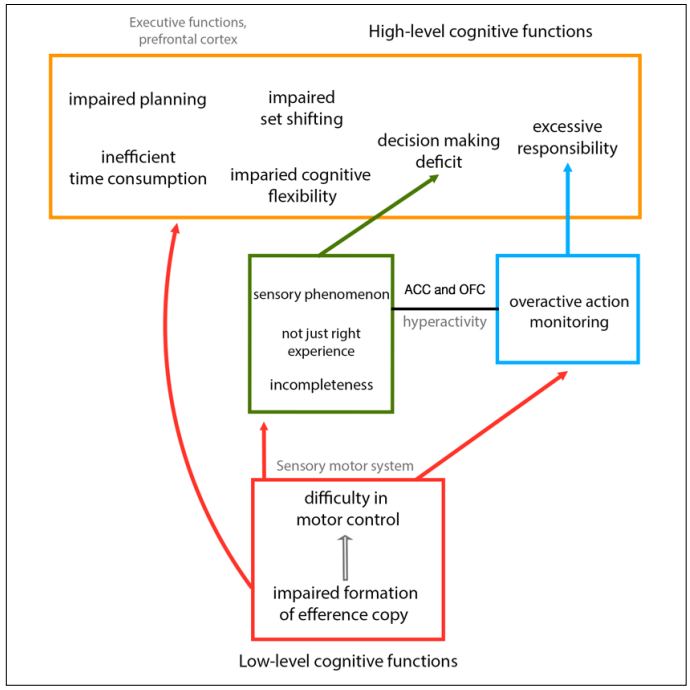

Figure 4. Modeling obsessive-compulsive disorder (OCD) from the perspective of embodied cognition (EC): The proposed integration of low-level functions with higher-order functions for OCD is shown in the EC perspective. The red rectangles represent low-level cognitive processes. The brown rectangle also symbolizes higher-order cognitive functions. In the figure, a hypothetical scheme is presented on how problems ordered from low level to higher order processes can be integrated with each other in phenomenological level where problems are defined. indirectly. ACC: Anterior Cingulate Cortex; OFC: Orbitofrontal cortex

CONCLUSION

The fact that impairment in low-level sensory-motor functions in OCD is associated with the establishment of the efference copy does not mean that dysfunction in fronto-striatal circuits can be excluded from the pathophysiology of OCD. According to the EC theory, neurons in the primary motor cortex that provide clapping are involved in the

production of the verb "to clap", but this does not mean that the neurons directly produce the word. Impairment in motor control in OCD may be involved in pathological processes related to dysfunctional fronto-striatal circuits. Problems in motor control may be accompanied by dysfunctional sensory phenomena, including OFC and ACC, and increased action monitoring.

For example, a problem in the establishment of the efference copy can be observed with hyperactivity of ACC because there can be a conflict between the expected state of action and its actual state associated with a discrepancy between what is expected and what is the real state of the action. This may, also, result in experiencing incompleteness. When a situation occurs in which OFC and ACC are prove dysfunctional, the individual with OCD may become unable to exhibit the required functionality due to the experience of conflict in decision-making processes and motor control problems.

The representation problems of being the subject of motor action, and a dysfunctional OFC and DLPFC together may explain the "inflexibility" and "exaggerated responsibility", which are mainly temperamental characteristics.

Such an explanation would shape our understanding of OCD as a clinical manifestation of different combinations of dysfunctional processes encompassing several brain areas and networks. Designing studies to understand the etiopathogenesis of OCD from this perspective can provide a wider scope to understand OCD and implement treatments.

Disclosure: *There are no financial conflicts of interest with any institution, organization, or person related to the publication and there is no conflict of interest between the authors.*

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – OMK, SC, PKÜ, CH; Design – OMK, SC, PKÜ, CH; Supervision – OMK, SC, PKÜ, CH; Resource – OMK, SC, PKÜ, CH; Literature Search – OMK, SC, PKÜ, CH; Writing – OMK, SC, PKÜ, CH; Critical Appraisal – OMK, SC, PKÜ, CH.

Conflict of Interests: The authors declared that there is no conflict of interest

Financial Disclosure: There is not any financial support for this study.

REFERENCES

1. American Psychiatric Association: Diagnostic and statistical manual of mental disorders, Fifth Ed. Arlington, VA, American Psychiatric Association, 2013. https://med-mu.com/wp-content/uploads/2018/08/American-Psychiatric-Association-Diagnostic-and-statistical-manual-of-mental-disorders-_-DSM-5-American-Psychiatric-Association-2013.pdf
2. Ruscio AM, Stein DJ, Chiu WT, Kessler RC. the epidemiology of obsessive-compulsive disorder in the national comorbidity survey replication. Mol Psychiatry. 2010;15(1):53–63. [Crossref]
3. Torres AR, Prince MJ, Bebbington PE, Bhugra D, Brugha TS, Farrell M, et al. obsessive-compulsive disorder: prevalence, comorbidity, impact, and help-seeking in the British national psychiatric morbidity survey of 2000. Am J Psychiatry. 2006;163:1978–1985. [Crossref]
4. Soomro GM, Altman D, Rajagopal S, Oakley-Browne M. Selective serotonin re-uptake inhibitors (SSRIs) versus placebo for obsessive compulsive disorder (OCD). Cochrane Database Sys Rev. 2008;2008(1):CD001765. [Crossref]
5. McKay D, Sookman D, Neziroglu F, Wilhelm S, Stein DJ, Kyrios M, et al. Efficacy of cognitive-behavioral therapy for obsessive-compulsive disorder. Psychiatry Res. 2015;227(1):104–113. [Crossref]
6. Pallanti S, Quercioli L. Treatment-refractory obsessive-compulsive disorder: methodological issues, operational definitions and therapeutic lines. Prog Neuropsychopharmacol Biol Psychiatry. 2006;30(3):400–412. [Crossref]
7. van den Heuvel OA, Veltman DJ, Groenewegen HJ, Cath DC, van Balkom AJ, van Hartskamp J, et al. Frontal-striatal dysfunction during planning in obsessive-compulsive disorder. Arch Gen Psychiatry. 2005;62(3):301–309. [Crossref]
8. Maltby N, Tolin DF, Worhunsky P, O'Keefe TM, Kiehl KA. Dysfunctional action monitoring hyperactivates frontal-striatal circuits in obsessive-compulsive disorder: an event-related fMRI study. Neuroimage. 2005;24(2):495–503. [Crossref]
9. Tang W, Zhu Q, Gong X, Zhu C, Wang Y, Chen S. Cortico-striato-thalamo-cortical circuit abnormalities in obsessive-compulsive disorder: A voxel-based morphometric and fMRI study of the whole brain. Behav Brain Res. 2016;313:17–22. [Crossref]
10. Peng Z, Shi F, Shi C, Miao G, Yang Q, Gao W, et al. Structural and diffusion property alterations in unaffected siblings of patients with obsessive-compulsive disorder. PLoS One. 2014;9(1):e85663. [Crossref]
11. Gürsel DA, Avram M, Sorg C, Brandl F, Koch K. Frontoparietal areas link impairments of large-scale intrinsic brain networks with aberrant fronto-striatal interactions in OCD: a meta-analysis of resting-state functional connectivity. Neurosci Biobehav Rev. 2018;87:151–160. [Crossref]
12. Robbins TW, Vaghi MM, Banca P. Obsessive-compulsive disorder: puzzles and prospects. Neuron. 2019;102(1):27–47. [Crossref]
13. Carlisi CO, Norman LJ, Lukito SS, Radua J, Mataix-Cols D, Rubia K. Comparative multimodal meta- analysis of structural and functional brain abnormalities in autism spectrum disorder and obsessive-compulsive disorder. Biol Psychiatry. 2017;82(2):83–102. [Crossref]
14. De Marchi N, Mennella R. Huntington's disease and its association with psychopathology. Harv Rev Psychiatry. 2000;7:278–289. [Crossref]
15. Maia AF, Pinto AS, Barbosa ER, Menezes PR, Miguel EC. Obsessive-compulsive symptoms, obsessive-compulsive disorder, and related disorders in Parkinson's disease. J Neuropsychiatry Clin Neurosci. 2003;15(3):371–374. [Crossref]
16. Pavone P, Bianchini R, Parano E, Incorpora G, Rizzo R, Mazzone L, et al. Anti-brain antibodies in PANDAS versus uncomplicated streptococcal infection. Pediatric Neurol. 2004;30(2):107–110. [Crossref]
17. Nakatani E, Nakgawa A, Ohara Y, Goto S, Uozumi N, Iwakiri M, et al. Effects of behavior therapy on regional cerebral blood flow in obsessive-compulsive disorder. Psychiatry Res. 2003;124(2):113–120. [Crossref]
18. Brown LT, Mikell CB, Youngerman BE, Zhang Y, McKhann GM 2nd, Sheth SA. Dorsal anterior cingulotomy and anterior capsulotomy for severe, refractory obsessive-compulsive disorder: a systematic review of observational studies. J Neurosurg. 2016 Jan;124(1):77-89. doi: 10.3171/2015.1.JNS14681. Epub 2015 Aug 7. PMID: 26252455.
19. van den Heuvel OA, van Wingen G, Soriano-Mas C, Alonso P, Chamberlain SR, Nakamae T, et al. Brain circuitry of compulsivity. Eur Neuropsychopharmacol. 2016;26(5):810–827. [Crossref]
20. Shephard E, Stern ER, van den Heuvel OA, Costa DL, Batistuzzo MC, Godoy PB, et al. Toward a neurocircuit-based taxonomy to guide treatment of obsessive-compulsive disorder. Mol Psychiatry. 2021;26(9):4583–4604. [Crossref]
21. Stein DJ, Costa DL, Lochner C, Miguel EC, Reddy YC, Shavitt RG, et al. Obsessive-compulsive disorder. Nat Rev Dis Primer. 2019;5(1):52. [Crossref]
22. Seidler RD, Noll DC, Thiers G. Feedforward and feedback processes in motor control. Neuroimage. 2004;22(4):1775–1783. [Crossref]
23. Ghez C, Hening W, Gordon J. Organization of voluntary movement. Curr Opin Neurobiol. 1991;1(4):664–671. [Crossref]
24. Gillan CM, Papmeyer M, Morein-Zamir S, Sahakian BJ, Fineberg NA, Robbins TW, et al. Disruption in the balance between goal-directed behavior and habit learning in obsessive-compulsive disorder. Am J Psychiatry. 2011;168(7):718–726. [Crossref]
25. Dickinson A. Actions and habits: the development of behavioral autonomy. Philos Trans R Soc Lond B Biol Sci. 1985;308(1135):67–78. [Crossref]
26. Fineberg NA, Robbins TW. The neurobiology and treatment of OCD: accelerating progress. Switzerland AG: Springer Cham; 2021. [Crossref]
27. Valentin VV, Dickinson A, O'Doherty JP. Determining the neural substrates of goal-directed learning in the human brain. J Neurosci. 2007;27(15):4019–4026. [Crossref]
28. Gehring WJ, Goss B, Coles MG, Meyer DE, Donchin E. A neural system for error detection and compensation. Psychol Sci. 1993;4(6):385–390. [Crossref]
29. Gillan CM, Fineberg NA, Robbins TW. A trans-diagnostic perspective on obsessive-compulsive disorder. Psychol Med. 2017;47(9):1528–1548. [Crossref]
30. Fitzgerald KD, Taylor SF. Error-processing abnormalities in pediatric anxiety and obsessive compulsive disorder. CNS Spectr. 2015;20(4):346–354. [Crossref]
31. Abramovitch A, Abramowitz JS, Mittelman A. The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis. Clin Psychol Rev. 2013;33(8):1163–1171. [Crossref]

32. Vaghi MM, Vértes PE, Kitzbichler MG, Apergis-Schoute AM, van der Flier FE, Fineberg NA, et al. Specific fronto-striatal circuits for impaired cognitive flexibility and goal-directed planning in obsessive-compulsive disorder: evidence from resting-state functional connectivity. Biol Psychiatry. 2017;81(8):708–717. [Crossref]
33. Rotge J-Y, Guehl D, Dilharreguy B, Cuny E, Tignol J, Bioulac B, et al. Provocation of obsessive-compulsive symptoms: a quantitative voxel-based meta- analysis of functional neuroimaging studies. J Psychiatry Neurosci. 2008;33(5):405–412. https://www.jpn.ca/content/33/5/405.long
34. Brown C, Shahab R, Collins K, Fleysher L, Goodman WK, Burdick KE, et al. Functional neural mechanisms of sensory phenomena in obsessive-compulsive disorder. J Psychiatr Res. 2019;109:68–75. [Crossref]
35. Koçak OM, Özpolat AY, Atbaşoğlu C, Çiçek M. Cognitive control of a simple mental image in patients with obsessive-compulsive disorder. Brain Cogn. 2011;76(3):390–399. [Crossref]
36. Foglia L, Wilson RA. Embodied cognition. Wiley Interdiscip Rev Cogn Sci. 2013;4(3):319–325. [Crossref]
37. Sowa JF. Review of "Lakoff G, Johnson M. Philosophy in the flesh: Embodied mind and its challenge to western thought. New York: Basic Books" Computational Linguistics. 1999;25(4):631–634.
38. Barsalou LW. Grounded cognition. Annu Rev Psychol. 2008;59:617–645. [Crossref]
39. Meier BP, Hauser DJ, Robinson MD, Friesen CK, Schjeldahl K. What's "up" with God? Vertical space as a representation of the divine. J Pers Soc Psychol. 2007;93(5):699–710. [Crossref]
40. Jostmann NB, Lakens D, Schubert TW. Weight as an implementation of importance. Psychol Sci. 2009;20(9):1169–1174. [Crossref]
41. Chiao JY, Harada T, Oby ER, Li Z, Parrish T, Bridge DJ. Neural representations of social status hierarchy in human inferior parietal cortex. Neuropsychologia. 2009;47(2):354–363. [Crossref]
42. Hauk O, Johnsrude I, Pulvermüller F. Somatotopic representation of action words in human motor and premotor cortex. Neuron. 2004;41(2):301–307. [Crossref]
43. Labruna L, Fernández-del-Olmo M, Landau A, Duqué J, Ivry RB. Modulation of the motor system during visual and auditory language processing. Exp Brain Res. 2011;211(2):243–250. [Crossref]
44. Courson M, Tremblay P. Neural correlates of manual action language: comparative review, ALE meta-analysis and ROI meta-analysis. Neurosci Biobehav Rev. 2020;116:221–238. [Crossref]
45. Wilson M. Six views of embodied cognition. Psychon Bull Rev. 2002;9(4):625–636. [Crossref]
46. Decety J, Grèzes J. The power of simulation one's own and other's behavior. Brain Res. 2006;1079(1):4–14. [Crossref]
47. Ross BH. The psychology of learning and motivation. London: Academic Press; 2011. https://co-mind.org/rdmaterials/pdfs/chapters/plm_59.pdf
48. Gentsch A, Schütz-Bosbach S, Endrass T, Kathmann N. Dysfunctional forward model mechanisms and aberrant sense of agency in obsessive-compulsive disorder. Biol Psychiatry. 2012;71(7):652–659. [Crossref]
49. Rounis E, Banca P, Voon V. Deficits in limb praxis in patients with obsessive-compulsive disorder. J Neuropsychiatry Clin Neurosci. 2016;28(3):232–235. [Crossref]
50. Subirà M, Sato JR, Alonso P, do Rosário MC, Segalàs C, Batistuzzo MC, et al. Brain structural correlates of sensory phenomena in patients with obsessive-compulsive disorder. J Psychiatry Neurosci. 2015;40(4):232–240. [Crossref]
51. Miguel EC, do Rosário-Campos MC, Prado HS, do Valle R, Rauch SL, Coffey BJ, et al. Sensory phenomena in obsessive-compulsive disorder and Tourette's disorder. J Clin Psychiatry. 2000;61(2):150–156. [Crossref]
52. Niziolek CA, Nagarajan SS, Houde JF. What does engine effect copy represent? Evidence from speech production. J Neurosci. 2013;33(41):16110–16116. [Crossref]
53. Reiner A, Hart NM, Lei W, Deng Y. Corticostriatal projection neurons - dichotomous types and dichotomous functions. Front Neuroanat. 2010;4:142. [Crossref]
54. Deng Y, Lanciego J, Kerkerian-Le-Goff L, Coulon P, Salin P, Kachidian P, et al. Differential organization of cortical inputs to striatal projection neurons of the matrix compartment in rats. Front Syst Neurosci. 2015;9:51. [Crossref]
55. Shipp S. The functional logic of corticostriatal connections. Brain Struct Funct. 2017;222(2):669–706. [Crossref]
56. McCutcheon RA, Abi-Dargham A, Howes OD. Schizophrenia, dopamine and the striatum: from biology to symptoms. Trends Neurosci. 2019;42(3):205–220. [Crossref]
57. Aliu SO, Houde JF, Nagarajan SS. Motor-induced suppression of the auditory cortex. J Cogn Neurosci. 2009;21:791–802. [Crossref]
58. Brumberg JS, Pitt KM. Motor- induced suppression of the N100 event-related potential during engine imagery control of a speech synthesizer brain-computer interface. J Speech Lang Hear Res. 2019;62(7):2133–2140. [Crossref]