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# Obsessive–compulsive disorder as a visual processing impairment

Óscar F. Gonçalves [a,*], Tiago Reis Marques [b,c], Nicolás F. Lori [d], Adriana Sampaio [a], Miguel Castelo Branco [d]

[a] Neuropsychophysiology Laboratory, CIPSI, School of Psychology, University of Minho, 4700 Braga, Portugal
[b] Institute of Psychiatry, University of London, UK
[c] University Hospital of Coimbra, Portugal
[d] Visual Neuroscience Laboratory, IBILI, Faculty of Medicine, University of Coimbra, Portugal

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SUMMARY

OCD has been hypothesized to involve the failures in both cognitive and behavioral inhibitory processes. There is evidence that the hyperactivation of cortical–subcortical pathways may be involved in the failure of these inhibitory systems associated with OCD. Despite this consensus on the role of frontal–subcortical pathways in OCD, recent studies have been showing that brain regions other than the frontal–subcortical loops may be needed to understand the different cognitive and emotional deficits in OCD. Some studies have been finding evidence for decreased metabolic activity in areas such as left inferior parietal and pari-eto-occipital junction suggesting the possible existence of visual processing deficits. While there has been inconsistent data regarding visual processing in OCD, recent studies have been claiming that these patients have abnormal patterns of visual processing social rich stimuli, particularly emotional arousing stimuli. Thus, in this article, we hypothesize that the fronto-subcortical activation consistently found in OCD may be due to a deactivation of occipital/parietal regions associated with visual-perceptual processing of incoming social rich stimuli. Additionally, this dissociation may be more evident as the emotional intensity of the social stimulus increases.



## Introduction

OCD is an anxiety disorder characterized by the presence of intrusive negative thoughts, images, or ideas, involuntarily entering consciousness (obsessions) which are often accompanied by repetitive, stereotyped or ritualized behaviors (compulsions).

OCD is probably one of the most disabling psychological disorders with a consistent cross cultural lifetime prevalence of about 2.5% [1,2].

In terms of neurocognitive processes, OCD has been hypothesized to involve the failures in two main inhibitory processes: (a) cognitive inhibitory process (responsible for obsessive symptoms); and (b) behavioral inhibitory process (responsible for compulsive symptoms) [3].

The failures of these two inhibitory systems may mediate not only OCD cognitive and behavioral symptoms but also most of the neuropsychological deficits found in this disorder in terms of attention, memory, planning and decision making [3–5].

Several distinct pathophysiological mechanisms have been associated with OCD at the neuroimmunological [6], neurochemical [7], and neuroanatomic levels [8]. A variety of neuroanatomic models have been proposed to explain the pathogenesis of OCD [9–11] but they tend to agree on the role of cortico-basal ganglia–thalamus interaction.

Contemporary models of OCD pathogenesis acknowledge that two cortical–subcortical pathways may be involved in the failure of the inhibitory systems: (a) the frontostriatal loop (dorsolateral-caudate–striatum–thalamus) responsible for failures of behavioral inhibition; (b) the orbitofrontal loop (orbitofrontal, medial prefrontal and cingulate) responsible for failures with cognitive inhibitory processes [12,13].

In fact, structural MRI studies confirm the existence of volumetric changes in these cortical–subcortical networks. A recent meta-analysis found evidence of significant volumetric reductions on the left anterior cingulated and bilateral orbitofrontal cortex, contrasting with an increase in left and right thalamic volumes [14].

These structural changes may be the consequence of the hyperactivation of frontal–subcortical circuits often found in neurofunctional studies. For example, a meta-analysis of the studies using PET found significant differences in radiotracer uptake between OCD and controls suggesting an increase in activity in the left orbital gyrus and the left and right head of the caudate nucleus [8]. Not differently, a recent review by Rotge et al. [15] on functional neuroimaging of provocation of OCD symptoms corroborates the consensus on the activation of the orbitofrontal and anterior cingulated loops.

* Corresponding author. Tel.: +351 253604261; fax: +351 253604221.
*E-mail address:* goncalves@iep.uminho.pt (Ó.F. Gonçalves).

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Despite this consensus on the role of frontal–subcortical activation in OCD, recent studies have been showing that brain regions other than the frontal–subcortical loops may be needed to understand the different cognitive and emotional deficits in OCD. In fact, a recent quantitative voxel-level meta-analysis of fMRI case–control OCD studies by Menzies et al. [16] brings evidence for the need to consider the role of other posterior brain regions in the pathogenesis of OCD. For example, Nordahl et al. [17] found a below-normal glucose metabolism in the occipital–parietal area contrasting with increased glucose metabolism in the left orbital frontal, right sensorimotor, and bilateral prefrontal and anterior cingulate regions. More recently, Kwon et al. [18] found a decreased metabolic activity in left inferior parietal and parieto-occipital junction contrasting with orbitofrontal hyper-metabolism in OCD patients.

In another study a whole brain voxel-based morphometry conducted by van den Heuvel [19] showed that multiple symptomatic dimensions of OCD may be associated with specific structural substrates both in terms of grey and white matter. Even though overall volume reductions were found for both white matter and grey matter in the cortical–subcortical networks, specific patterns of structural changes were found to be associated with different symptomatic configurations such as: symmetry/ordering (e.g., negatively correlated with right motor cortex grey matter; left insula and left parietal cortex); contamination/washing (e.g., negatively correlated with bilateral caudate nucleus and white matter volume in right parietal region); harm/checking (e.g., negatively correlated with bilateral temporal lobes white matter).

Not differently, diffusion tensor imaging (DTI) studies analyzing connectivity patterns in patients with OCD showed lower fractional anisotropy (FA) values in anterior and posterior cingulate gyrus, as well as bilaterally in parietal regions and in occipital lobe [20,21]. The lower FA values in the parietal region correlated significantly with higher Yale-Brown obsessive–compulsive scale scores [21]. Interestingly, these frontal and parietal white matter abnormalities were also exhibited by first relatives of patients with OCD [22].

Consistent with this, spectroscopy studies bring additional support to the involvement of parietal white matter abnormality in OCD in finding that choline-containing compounds and creatine/phosphocreatine were found to be significantly higher in the parietal cortex of OCD patients [23].

Altogether, there is initial evidence that areas involved in visual processing may be impaired in OCD. However, studies of visual processing results in OCD have provided inconsistent data with some authors claiming the existence of visual impairments [24] while others claim that visual processing deficits may be overestimated [25]. We believe that these inconsistencies are due to the abstract/conceptual nature of most of the stimuli used in the situation. We believe that moving visual stimuli closer to an ecological and emotional context will show evidence of visual processing impairment.

A recent study by Jung et al. [26] brings initial evidence for this hypothesis by showing that in a more social stimuli situation such as biological motion, OCD patients show an increased activation of the superior, middle, inferior temporal and fusiform gyrus as well as cerebellum and a hypoactivation in the postcentral region.

The need to study visual processing deficit in emotional contexts was recently demonstrated by Moritz et al. [27] finding that, in OCD patients, visual attention was affected both at early and late stages of processing when OCD relevant emotional stimuli are used.

Finally, in a clinical study, Kang et al. [28] found that improvement after SSRI psychopharmacological treatment was associated with improvement in performance of Rey–Osterrieth complex figure test. Additionally, this improvement was associated with decreased metabolism in frontal–subcortical and increased metabolism in parietal–occipital regions (e.g., lateral right postcentral gyrus; posterior region of the superior parietal lobe; medial portion of the superior occipital gyrus). Not differently, Nabeyama et al. [29] found that after effective treatment with behavior therapy, OCD patients showed, in a Stroop task, increased activation of the posterior regions such as parietal cortex and a decrease in the activation of frontal regions such as orbitofrontal cortex and middle frontal gyrus.

## The hypothesis

In sum, based on current findings, one may hypothesize that the fronto-subcortical activation consistently found in OCD may be due to a deactivation of occipital/parietal regions associated with visual-perceptual processing of incoming social rich stimuli.

Additionally, this dissociation may be more evident as the emotional intensity of the social stimulus increases. In fact studies have shown that the presentation of emotional pictures (e.g., IAPS) is responsible for increased activation of the visual cortex in healthy individuals when compared with non-emotional pictures [30] and that the level of picture affective arousal correlates positively with functional activity [30,31]. Additionally, the extent and strength of visual cortex activation was found to be greater for images with "erotic" and "mutilation" content [31,32]. These effects were particularly evident in visual secondary association areas BA [18,19] and fusyform gyrus.

Contrary to what is seen in normal healthy controls, OCD patients have a reversal pattern, with an increased activation in the frontal areas for the presentation of eliciting emotional arousal associated with their obsessions/compulsions [33].

Altogether, we believe there is initial evidence to advance the following hypothesis concerning the pathophysiology of OCD:

1. The fronto–subcortical activation consistently found in OCD may be due to a correlative of a deactivation of occipital/parietal regions associated with visual-perceptual processing.
2. The impairment of visual processing deficits may be restricted to social rich stimuli and as such be more evident on visual cortical association areas.
3. The level of visual processing deficit may be correlative on the level of emotional arousal of the social stimuli.

## Implications and further studies

If future research validates these hypotheses, important implications may be derived for the development of new therapeutic approaches. For instances, cognitive therapy methods should be aimed to increase the efficiency of visual processing for emotional laden stimuli rather than using distractive or thought-stopping techniques. In psychopharmacological treatments, all medications that might interfere with visual processing abilities must be avoided. Finally, more recent methods, such as transcranial magnetic stimulation, can be used to activate visual processing areas and deactivate frontal–subcortical circuits while the patient is confronted with emotional triggers.

## Conflicts of interest statement

There are not any financial, relationship and organizational conflict of interests that may bias any of the authors in the establishment of the hypotheses discussed in this article.

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