Degree project

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# OCD as Behavioral Addiction and the Reward Process: A Systematic Review

Bachelor Degree Project in Cognitive Neuroscience

First Cycle 22.5 credits

Autumn term Year 2021

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## Abstract

Studies have shown that aberrant activity in some brain regions involved in the pathology of OCD overlaps similarly with individuals with addiction disorders. The reduced anxiety following a compulsion together with findings of diminished activation in the striatum during reward anticipation proposes a view of OCD being a behavior addiction. To investigate if there are consistent results across studies that support this view a systematic search of the literature was conducted. The keywords in the final search string used were: Obsessive-compulsive disorder, OCD, reward, risk, functional MRI, MRI, fMRI. Databases used for the search were Web of Science and PubMed. The inclusion criteria were studies that compared the neural activity during the anticipation phase of reward between OCD patients and healthy controls. The intervention and brain imaging used in the included studies were the monetary incentive delay task and fMRI. The main data extracted were the alterations in the striatum. Four studies were included in this review with inconsistent results. Three studies did not find any significant difference between OCD and healthy controls and therefore the findings in principle did not support the view of OCD being a behavior addiction. However, differences in study design between studies could be an explanation for the conflicting findings.

*Keywords*: obsessive-compulsive disorder, reward, addiction.

# OCD as Behavioral Addiction and the Reward Process: A Systematic Review

Obsessive-compulsive disorder (OCD) is a neuropsychiatric disorder that affects more than 1% of the world's population. OCD can have an onset throughout life. The cause is not determined, though different theories exist such as genetic predisposition, behavioral addiction, and streptococcal infection (Figee et al., 2011; Grassi et al., 2015; Mell et al., 2005; Pauls et al., 2014). The comorbidity of other psychiatric disorders is common and the study of Hasler et al. (2005) showed that 92% of the participants (n=317) with OCD were diagnosed with at least one additional disorder (e.g., depression, mood, and eating disorders). Symptoms in people with OCD include obsessions in the form of intrusive unwanted thoughts which give rise to anxiety and are followed by compulsions in anticipation to reduce anxiety. Because the compulsions only reduce the anxiety short-term, the behavior is characterized by repetitive actions expressed in for example checking, controlling, or reassurance seeking (e.g., asking someone if they think it is possible that their thought is true, checking one's own bodily reactions for cues of physiological arousal or evaluating how the thought is making one feel, internet searching for answers, confessing their thoughts; Figee et al., 2011; Samuels et al., 2017).

The intrusive thoughts are disturbing, often involving catastrophic consequences (e.g., "If I touched something contaminated, I will die or pass it on to someone else who will die"), and individuals with OCD have a hard time accepting the possibility of a negative outcome in the future even if the possibility is low (Samuels et al., 2017). The most common measure used for mapping the severity of the disorder is the Yale-Brown Obsessive Compulsive Scale (Y-BOCS). The scale consists of 10-items where each item is rated from 0 to 4 with 0 being "no symptoms", measuring obsessions and compulsions separately. The score ranges between 0-40 (0-7 subclinical, 8-15 mild, 16-23 moderate, 24-31 severe, 32-40 extreme; Goodman et al., 1989). The treatment with the greatest empirical support is cognitive-behavioral therapy with exposure response prevention. It has a dropout rate of

30%. Of that 70% who complete the treatment 60% achieve some recovery, and only 25% get symptom-free (Melchior et al., 2019).

OCD has consistently shown dysfunctional connectivity in the corticostriato-thalamo-cortical circuitry and has generally been seen as an anxiety disorder (Luigjes et al., 2016). The main view is that the core dimension of OCD is harm avoidance behavior which is expressed in excessive avoidance of possible harm (Summerfeldt et al., 2014). In relation to risk aversion, OCD individuals had higher activation of the insula (which is for instance involved in risky decision making) in comparison with control (Luigjes et al., 2016). Studies have shown that those with OCD are more unwilling to take risky decisions compared to controls, which could be because they over-exaggerate the negative outcomes (Admon et al., 2012). Also, OCD patients have a quicker response to negative stimuli and slower to positive (Admon et al., 2012). OCD is often referred to as the "sickness of doubt", which is expressed in uncertainty and an experience of not being able to identify the advantageous outcome resulting in difficulties to make a choice (Cavedini et al., 2006). The executive function of decision-making involves various brain structures prominent in the pathology of OCD, such as the prefrontal cortex (PFC; Cavedini et al., 2006). The orbitofrontal cortex (OFC) has consistently been shown to be overactive in the resting state in people with OCD, which is associated with rumination about unpleasant future events. After successful treatment, this alteration in OFC reversed towards the activity level of control participants (Ursu & Carter, 2009). It has been stated in numerous studies that those with OCD have impaired decision-making in comparison to healthy controls (Cavedini et al., 2006).

Value and reward are important in the decision-making process (Cavedini et al., 2006). The process of reward involves phases of anticipation and outcome. The anticipation of reward is salient for the motivational process, it is guided by the subjective value of a cue that is either inborn or learned. For the learning of valuable cues, the outcome phase of reward is important (Luijten et al., 2017). Recent research on OCD has revolved around the disorder-specific dysfunction in the reward system. Individuals with OCD have shown

abnormalities in OFC and striatum, brain regions that are involved in the reward process (Figee et al., 2011). The caudate nucleus and the putamen constitute the dorsal striatum and are involved in implicit learning. The meta-analysis of Del Casale et al. (2011) showed that patients with OCD had diminished activity in the caudate nucleus during an implicit learning task and reward outcome. Deviant activation of the striatum has consistently been reported in those with addictive behaviors. The ventral striatum contains the nucleus accumbens (NAc) and the olfactory tubercle. Studies have shown conflicting neural activity of the NAc in individuals with an addiction disorder, with either higher activation or lower activation than healthy controls (Luijten et al., 2017). To measure the neural correlates of the striatum during the different stages of the reward process different tasks can be used in combination with brain imaging measures. The most commonly used (e.g., due to the reason for not requiring the involvement of complex cognitive functions) is the monetary incentive delay task (MIDT), which involves an imaging paradigm. The MIDT consists of three trial types, reward, punishment, and neutral. In the reward trial participants can win money, in the punishment/loss trial they can lose money, and in the neutral trial, there is no monetary consequence. Depending on the initial cue that indicates which trial type will follow, the anticipation of reward or loss can be measured during the following delay phase. After the delay, a target is presented which the participants should press as fast as possible. If the target is pressed within the time limit, in the reward trial participants gain money, and in the punishment trial they avoid losing money. The reward outcome is measured during the last phase when the result of either gain or loss is presented to the participant (Oldham et al., 2018). There are different methods available to measure brain activation (e.g., positron emission tomography, electroencephalogram, functional magnetic resonance imaging). The most common method is functional magnetic resonance imaging (fMRI).

Compulsions in OCD are experienced as rewarding due to the reduction of anxiety following a compulsion. This rewarding experience underlies the new possible view of OCD besides being anxiety-driven also being a behavior addiction (Figee et al., 2011). Figee et al. (2011) investigated the reward anticipation process where participants performed MIDT

while measuring the neural activity with fMRI. Decreased activity in NAc was observed in the group with OCD compared to controls. Interestingly, increased activation in NAc has been seen in previous studies when individuals with OCD responded to obsessions. This finding is similar to the process in those with an addiction disorder where the activation of the NAc is blunted to neutral rewards in the anticipation phase and enhanced when the reward is the particular substance of addiction (Figee et al., 2011).

The interest of this systematic review is to explore the view of OCD being a behavior addiction. This will be done by a comparison of literature results of studies that have investigated the neural correlates of the striatum due to its involvement in addiction and its dysfunction in the pathology of OCD. Because of the motivational aspect of the reward anticipation, this phase will be the focus rather than the outcome phase which is related to the process of learning. Studies using fMRI in combination with MIDT are the ones of focus in this review. The purpose is to answer the question of whether there is a consistency in the findings of neural activity across studies that investigated reward anticipation in people with OCD in comparison with healthy control. Consistent results of aberrant activity in the striatum in relation to the reward process would give support to the view of OCD being a behavior addiction which also would help to direct future studies to investigate this view and develop new relevant treatments.

## Method

### Search Strategy

The initial search started by trying out different combinations of keywords (obsessive compulsive disorder, ocd, reward, decision making, frontal cortex, orbitofrontal cortex, neuroimaging, striatum, nucleus accumbens, fronto-striatal circuit, value, risk, choice, brain regions, areas, activity, fMRI, functional MRI, MRI). The final search string used was "(obsessive compulsive disorder OR OCD) AND (reward OR risk) AND (functional MRI OR MRI OR fMRI)". The end date for the search was March 15th, 2021. The search string was required to be present in the title or abstract. A total of 322 records were generated (n=170 Web of Science, n=152 PubMed). These records were extracted, saved, and imported into

EndNote where 38 duplicates were removed and 284 records remained. The remaining records were screened for the title and abstract where 278 records were excluded due to not meeting the inclusion criteria (see inclusion and exclusion criteria). The full-text of the 6 articles that remained were screened. Two articles were excluded, one was excluded due to not measuring neural activity in the anticipation phase and the other not using MIDT as intervention. A total of 4 remaining studies were included in the systematic review (see Figure 1).

## Inclusion and Exclusion Criteria

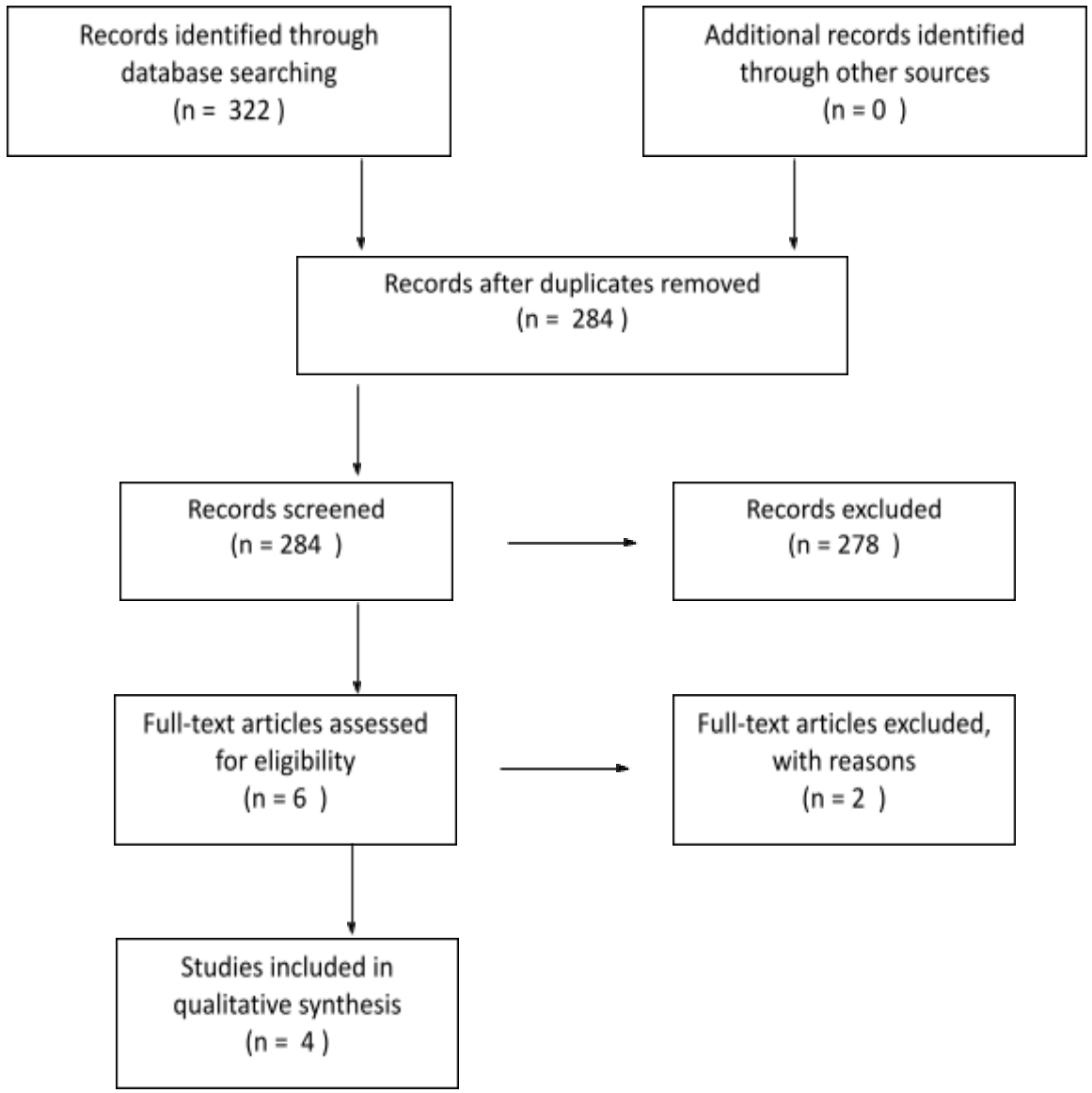
Inclusion criteria of the records are published peer-reviewed articles, and pre-publication papers with the design of an experimental study carried out between 1995 - 2021, and written in English. Only studies that are available through open access or the university database were included. Participants inclusion criteria are individuals diagnosed with OCD and compared to healthy controls. Therefore, studies that compare the neural correlates of patients with OCD with other disorders such as autism spectrum disorder, and attention-deficit/hyperactivity disorder are not of interest. Only studies using MIDT in combination with fMRI as a neuroimaging measure were included. Studies had to include evaluation of the anticipation phase of reward. The different processes in the reward circuit such as loss and avoidance are beyond the scope of this article.

## Data Extraction

To answer if the activation of the striatum in the anticipation phase of reward is a consistent finding in people with OCD across the literature. The findings that will be extracted and presented in a table in the result section are brain alterations in the reward-related circuitry while performing a MIDT measured with fMRI. The area of interest is the striatum (NAc, putamen, and caudate nucleus). Scores of the severity of OCD and depression will also be extracted if available. Studies have shown that the activation in the striatum is reduced in the reward anticipation phase in patients with major depression and is therefore of interest (Figee et al., 2011).

**Figure 1**

*PRISMA 2009 Flow Diagram*




*Note.* The literature search process, illustrated in a PRISMA Flow Diagram (Moher et al., 2009).

## Results

The search process and the final search results can be seen in Figure 1. All included studies (n=4) tested the anticipation phase of reward in patients with OCD compared to healthy controls, using the MIDT with fMRI for measuring neural activity. The clinical and behavioral data as well as the neural activity findings presented below are summarized in

Table 1.

## Clinical and behavioral data

The Y-BOCS mean score in the study of Figee et al. (2011) was 29.6 (SD = 7.3) indicated severe OCD symptoms in the participants, while in the remaining three studies the mean score ranged between 19.54 to 20.45 and according to the criteria of Goodman et al. (1989) are considered as moderate. In all four studies, the depression scores of the Hamilton depression rating scale (HAM-D) and Beck's depression inventory (BID) indicated mild to moderate symptoms in patients with OCD. Though only in the study of Figee et al. (2011) two participants were diagnosed with major depression, assessed with structured clinical interviewing. Overall, the comorbidity of other psychiatric disorders across studies ranged between 20% to 52%. All studies sex-matched the participants. In three studies the participants were matched by age (Choi et al., 2012; Jung et al., 2011; Kaufmann et al., 2013). The participants were IQ matched in the study of Choi et al. (2012) and Jung et al. (2011). The study of Kaufmann et al. (2013) matched the participants by verbal intelligence. The total number of OCD participants in all studies combined was 70, with more males than females (n=31).

## Neural activity during reward anticipation

Figee et al. (2011) had 18 participants with OCD, 9 of them were unmedicated. The MIDT used in this study had two cues indicating either neutral or reward trial. The time the cue was presented was 500ms. The reward anticipation was measured during the delay between the cue and the target, which varied between 3000-10000ms. A total of 72 trials were performed during the task. During the anticipation phase, attenuated activation of the NAc was found (which is the region of interest) in OCD participants. Also, diminished activation of the insula was seen in patients. Both the reduction of the NAc and insula activation were more prominent in a subgroup that had contamination symptoms when compared to patients with checking symptoms.

In the study of Jung et al. (2011), there were 20 OCD patients and in the study of Choi et al. (2012) the sample size was 13. The MIDT differed from the study of Figee et al. (2011) in

the time duration the cue was presented (350ms) and the delay time (4180-4480ms). Jung et al. (2011) found activation in the ventral striatum which matched the observation of Choi et al. (2012). Additionally, Choi et al. (2012) observed activation of the frontal cortex and occipital cortex but no significant difference of activation in either of these brain areas between control and OCD was found. The study of Kaufmann et al. (2013) had a sample size of 19 participants in the OCD group. The MIDT task in this study had three different cues in the reward trial with a duration time of 250ms. The cues differed in the amount of money that was possible to win in the reward trial. No significant differences in activation between control and OCD were found in the striatum.

**Table 1**

Summary of Participants Characteristics and Study Findings

| AUTHOR | PARTICIPANTS | COMPARISON | Y-BOCS Mean (Standard Deviation) | HAM-D/BDI Mean (Standard Deviation) | COMORBIDITY | TASK | REWARD ANTICIPATION FINDINGS |
|---|---|---|---|---|---|---|---|
| Figee et al. (2011) | N=18 OCD N=9 Unmedicated N=13 Females | N=19 Healthy control N=13 Females | 29.6 (7.3) | HAM-D 16.8 (6.6) | Major depressive disorder (N=2) Obsessive compulsive personality disorder (N=2) Additional disorders (N=4) | Monetary incentive delay task | Bilateral Nucleus Accumbens: Reduced activity |
| Jung et al. (2011) | N=20 OCD unmedicated N=7 Females | N=20 Healthy control Age, sex and IQ matched | 20.45 (6.46) | BDI 16.50 (11.40) | Tic disorder (N=1) Obsessive compulsive personality disorder (N=2) Schizotypal personality disorder (N=1) | Monetary incentive delay task | Ventral Striatum: Not significantly different from control |
| Choi et al. (2012) | N=13 OCD Males unmedicated | N=15 Healthy control Age, sex and IQ matched | 19.54 (5.94) | BDI 15.08 (10.42) | Tic disorder (N=1) Obsessive compulsive personality disorder (N=1) Schizotypal personality disorder (N=1) | Monetary incentive delay task | Ventral Striatum: Not significantly different from control |
| Kaufmann et al. (2013) | N=19 OCD N=16 Unmedicated N=11 Females | N=19 Healthy control Age, sex and verbal intelligence matched | 20.7 (7.9) | BDI 17 (11) | Affective disorder (N=3) Phobic disorder (N=3) Personality disorder (N=3) Impulse control disorder (N=1) | Monetary incentive delay task | Ventral Striatum: Not significantly different from control |

## Discussion

The aim of this systematic review was to investigate if there are consistent results across studies that support the view of OCD being a behavior addiction. The findings in this report are conflicting and accordingly, they do not mirror the dysfunction of neural activation in the striatum in the reward anticipation phase in those with OCD as in individuals with an addiction disorder. Studies included in this review used MIDT in combination with fMRI to look into the neural correlates of the striatum in OCD patients during reward anticipation. The report of Figee et al. (2011) found the activation of the ventral striatum in individuals with OCD to be diminished during the anticipation phase of reward, this is in line with the activation pattern seen in those with an addiction disorder. The results of the remaining three studies were not in line with the findings of Figee et al. (2011), as they did not observe any significant difference in the activation of the striatum between OCD and control (Choi et al., 2012; Jung et al., 2011; Kaufmann et al., 2013). The conflicting findings may depend on a couple of reasons. Firstly, Figee et al. (2011) was the only study where the Y-BOCS scores indicated severe OCD in participants. Choi et al. (2012) discussed the possibility of diminished activation of the ventral striatum to be specifically related to severe symptoms.

Secondly, all three studies which did not show any significant difference in striatum activation used a slightly different scheme of the MIDT, where they also tested the anticipation and outcome phase of loss (Choi et al., 2012; Jung et al., 2011; Kaufmann et al., 2013). Those with OCD react faster to negative stimuli (Admon et al., 2012) and during loss outcome phase trials results showed higher activation in the ventral striatum in OCD compared to control (Jung et al., 2011). This could mean that the studies where the participants had to switch between reward and loss trials, still had the elevated activity in the NAc as a response to the negative stimuli of loss during reward trials and therefore not matching the diminished activation of NAc observed in the anticipation phase of reward in the study of Figee et al. (2011) where no trials of loss were present.

Thirdly, in the study of Figee et al. (2011) OCD patients were measured for group difference in symptoms (contamination vs checking), those with contamination OCD had significantly more prominent diminished activation in the NAc than those with checking symptoms. Rauch et al. (2007) observed a different pattern of activation of the striatum in those with contamination symptoms compared to checking symptoms. The study reported an inverse correlation between contamination symptoms and striatal activation. The three studies included in this review that did not find any abnormal striatal activation between OCD and control did not divide the patients into subgroups. Therefore there is a possibility that the samples did not include an adequate number of participants with contamination symptoms to show for aberrant striatal activation. This raises the overall question if the dysfunction in the reward circuitry can be the core in the pathology of OCD, or if the heterogeneity of OCD symptoms and the different brain regions that seem to be involved respectively to symptoms are indicating the opposite. Future research should include neural activity comparison of subgroups mentioned in Summerfeldt et al. (2014) of symptoms such as contamination, checking, obsessions, and order to look for overlapping regions.

The view of OCD primarily being an anxiety-driven disorder with harm avoidance as a core dimension (Summerfeldt et al., 2014) can not be dismissed with the findings of this review. Even though the findings do not indicate a dysfunction in the striatum during reward anticipation I would argue that this does not exclude the possibility of a dysfunction in the reward process overall. Previous studies have shown that the activation in the caudate nucleus is diminished during reward outcome in people with OCD (Remijnse et al., 2006), and also diminished activation in the striatum compared to controls during an implicit learning task has been observed (Del Casale et al., 2011). The reward outcome is important in the learning of new rewarding cues (NAc reacts to these cues during reward anticipation), and the caudate nucleus is thought to react and process this information (Del Casale et al., 2011). An alteration in the anticipation phase would not necessarily be seen as the NAc is reacting to the cues that are already learned. The problem of those with OCD might therefore not depend on the motivational process toward already learned rewards but the learning of

rewards. According to the article of Summerfeldt et al. (2014), no dysfunction of the caudate nucleus was observed in the loss outcome phase. People with OCD can therefore be biased towards negative stimuli due to the dysfunction in the learning process of the rewarding cues and resulting in a faster response to the negative cues. Future research should continue to investigate the learning process of rewarding cues in OCD as it could explain why patients with OCD react faster to negative stimuli and slower to positive and give more insight into the behavior of over-excessive avoidance of possible harm.

## Limitations

The main limitation of this review is the few included studies. The reason is that the hypothesis of OCD depending on the dysfunction in the reward process has been recently proposed. By including studies that used tasks other than the MIDT, the search could have generated additional studies and offered a more reliable answer. Though the MIDT was favorable in this review because it specifically targets the activation of the striatum and does not involve complex cognition (which could influence the striatum) in comparison to reward learning tasks (Oldham et al., 2018). Admon et al. (2012) found lowered functional connectivity between NAc and OFC in the outcome phase of reward in those with OCD compared to healthy control. Lowered functional connectivity indicates an inadequate information process. Similar results of lowered functional connectivity between the NAc and frontal regions were found in alcohol-dependent adults during the reward outcome (Forbes et al., 2014). This review did not include studies that investigated the functional connectivity between regions in the anticipation phase. The dysfunction of the reward system in OCD could depend on the functional connectivity rather than neural activation in single regions.

One limitation of the included studies is the small sample sizes, ranging from 13 to 20 participants while a recommended sample size according to Cohen (1988) for group comparisons is at least 30. Also, the depression scores of individuals with OCD indicated mild to moderate symptoms and the comorbidity of psychological disorders were up to 52%. Although high comorbidity is a general observation in those with OCD (Hasler et al., 2005), it is still an issue that might raise the question if the results reflect the pathology of other

disorders than OCD. The high percentage of comorbid diagnoses in OCD makes it difficult to conduct research with pure samples. The combination of a larger sample size and group comparison is needed to get more valid results.

## Ethical and Societal Aspects

The implication of this review and included studies are the progression towards a broader understanding of the neural correlates involved in the pathology of OCD. OCD is the fourth most prevalent disorder in the world (Reddy et al., 2017). Being a common disorder with a high comorbidity with other psychological disorders (Hasler et al., 2005) it probably has a great distressing impact on the life of many individuals. Mental illness also might affect some societies that take economic responsibility for those individuals whose suffering hinders their functioning in society (e.g., paying long-term sick leave). The conduction of studies using brain imaging measures such as fMRI are expensive, though the broad negative impact the disorder has on the societal level is arguably bigger. Also, because the rate of those who get asymptomatic after the most commonly available treatment is low (25%; Melchior et al., 2019), the costs of therapy raise ethical concerns where the patients that do not gain full benefit need to repeatedly seek therapy for alleviation of symptoms. The possibility of which depends on the person's socioeconomic status. Research about OCD is crucial for the development of new or improved treatments.

**Conclusion.** Conclusively there is no consistency in the results in the literature that would support the view of OCD being a behavior addiction. The conclusion is though based on a limited amount of studies that have their own limitations. Even though the findings dismiss the new view, more research is needed to be able to reject the possibility of reward circuitry being the main issue in the pathology of OCD. Future studies should focus on bigger sample sizes, looking at the functional connectivity between regions during reward processing, observing the neural activity of striatum during implicit learning of rewards, and investigating if the exclusion of loss trials in MIDT results in different outcomes. Also, testing for group differences between subgroups of symptoms and between OCD with, and without comorbidity of other psychological disorders.

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