FACULTY OF PSYCHOLOGY
AND EDUCATIONAL SCIENCES

The relationship between obsessive-compulsive symptoms and information processing

Laura de Putter

Supervisor: Prof. Dr. Ernst H. W. Koster

A dissertation submitted to Ghent University in partial fulfilment of the requirements for the degree of Doctor of Psychology

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CONTENTS

CHAPTER 1

GENERAL INTRODUCTION

Obsessive compulsive disorder (OCD) is characterized by obsessions and/or compulsions. Obsessions are defined as intrusive and recurrent thoughts, impulses or images. These obsessions are typically suppressed or neutralized by compulsions. Compulsions are defined as ritualized, repetitive behaviors (e.g., checking or washing) or mental acts (e.g., counting or praying) intended to reduce distress or prevent a feared event. These obsessions and compulsions take at least one hour per day, cause significant distress and typically impairments in social functioning and work (American Psychiatric Association, 2013; Ruscio, Stein, Chiu, & Kessler, 2010). Its lifetime prevalence is 2.3 – 3.5%, however more than a quarter of people experience obsessions or compulsions at some time in their lives (Angst et al., 2004; Ruscio et al., 2010). Moreover, Rachman and de Silva (1978) demonstrated that nonclinical participants experience similar intrusive thoughts as clinical OCD patients. Indeed, Langlois, Freeston, and Ladouceur (2000) reported a prevalence of intrusive thoughts of 74% in nonclinical participants. OCD is more prevalent in females and the average age of onset is under age 20 (Angst et al., 2004; Ruscio et al., 2010). Although there are different efficacious treatments available (see Skapinakis et al., 2016), only 41.7% achieves remission (Farris, McLean, Van Meter, Simpson, & Foa, 2013). In order to improve treatments, in-depth understanding of the mechanisms of OCD is warranted.

OCD is a clinically heterogeneous disorder. Bloch, Landeros-Weisenberger, Rosario, Pittenger, and Leckman (2008) subdivided the symptoms of OCD in a meta-analysis in order to reduce heterogeneity and found the following symptom dimensions: (1) symmetry obsessions neutralized by repeating, ordering and counting. This symptom dimension differentiates itself by the need to resolve a not-just-right feeling rather than anxiety or fear (McKay et al., 2004). (2) Aggressive, sexual, religious or somatic obsessions neutralized by checking. Salkovskis (1985) and Rachman (1997;

1998) pose that the urge to check is elicited by the belief patients are responsible for these thoughts and their potential consequences. These consequences can be in real life (e.g., if someone has an image of stabbing someone, they fear they will actually do so) or for one's moral character (e.g., the fact that someone has an image of stabbing someone, means they are morally repugnant). Moreover, Salkovskis (1985) argues that some patients overestimate their responsibility by considering it their duty to prevent any possible harm, no matter how improbable. (3) Contamination obsessions neutralized by cleaning or washing. This symptom dimension consists of the fear of being contaminated or contaminating someone else (Markarian et al., 2010). Contamination fear is one of the most common symptom dimensions in OCD (Ball, Baer, & Otto, 1996). These symptom dimensions are associated with different patterns of neural substrates, genetic transmission, comorbidity, response to treatment, and neuropsychological functioning (Mataix-Cols, do Rosario-Campos, & Leckman, 2005). For instance, the symmetry and checking dimensions are more strongly associated with tic disorder (Leckman et al., 1997) and sexual/religious obsessions are associated with a lower treatment response to behavior therapy (Ball et al., 1996). Furthermore, the contamination symptom dimension is associated with better performance compared to the checking dimension on most cognitive tasks (Leopold & Backenstrass, 2015). To date, a wealth of research has focused on the relationship between the checking symptom dimension and executive functioning. As the contamination fear symptom dimension is also one of the most prevalent symptom dimensions, this dissertation will focus on the contamination fear symptom dimension.

Numerous studies have investigated the etiological and maintaining factors of this disorder. One of the main neurological models in OCD consists of abnormal functioning of the frontostriatal circuit. This model entails hyperactivation of the orbitofrontal cortex, anterior cingulate cortex, basal ganglia, and decreased activity of the dorsolateral prefrontal and parietal network. The frontostriatal circuit underlies executive functioning, a set of general-purpose control mechanisms that regulate our thoughts and behaviors (Miyake & Friedman, 2012). Therefore executive functioning is of particular interest in OCD (Melloni et al., 2012).

Executive Functioning in OCD

The literature on executive functioning in OCD is characterized by contrasting findings. For instance, Abramovitch, Abramowitz, and Mittelman (2013) reported substantial heterogeneity in findings across studies in different cognitive domains within their meta-analysis. However, overall meta-analyses have demonstrated differences between OCD patients and healthy controls in inhibition, set shifting, updating, verbal working memory, visuospatial working memory, planning, processing speed, and attention with effect sizes ranging from d = 0.3 to d = 0.7 (Abramovitch et al., 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2014). Although it should be noted that across these meta-analyses the magnitude of the effects varies (Snyder et al., 2014).

Different moderators have been investigated in order to explain the heterogeneity in these findings. However, depressive symptoms, age of onset, Selective Serotonin Reuptake Inhibitors (SSRIs), general motor-response slowing, and gender generally did not consistently moderate neuropsychological functioning in OCD (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014). Although it is impossible to rule out a deficit in general processing speed as a moderator (Snyder et al., 2014). Moreover, other authors have posed that OCD is associated with impaired confidence in memory, perception or attention rather than a clear deficit (e.g., Dek, van den Hout, Giele, & Engelhard, 2010; Hermans et al., 2008; Macdonald, Antony, Macleod, & Richter, 1997). For instance, Hermans et al. (2008) found that confidence in attention uniquely predicted checking behaviors and that repeated checking of individually selected compulsive actions resulted in increased distrust in attention in OCD patients.

More recently, studies started investigating the predictive effect of neuropsychological functions on treatment response. For instance, D'Alcante et al. (2012) found that OCD patients with better cognitive and executive abilities at baseline were more likely to respond to Cognitive Behavioral Therapy (CBT) or fluoxetine. Interestingly, they found that increased mental flexibility predicted a better response to CBT and a worse response to fluoxetine, suggesting that patients with different neuropsychological profiles may respond differently to certain types of treatment. Similarly, Braga et al. (2016) found a trend in which patients who responded to group

CBT performed better on tests assessing information processing speed, set shifting and working memory compared to non-responders. However, Bolton, Raven, Madronal-Luque, and Marks (2000) found that none of the neuropsychological deficits at baseline were able to predict response to behavioral treatment.

Inhibition is of specific interest in OCD given the repetitive nature of obsessions and compulsions (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). Nigg (2000) identified four types of inhibition of a motor or cognitive response: (1) Interference control, which prevents interference due to resource or stimulus competition. This type of inhibition is often assessed by a Stroop task in which participants inhibit the meaning of color words in order to name the color of its print (MacLeod, 1991). (2) Cognitive inhibition, which suppresses irrelevant information from working memory. (3) Behavioral inhibition or response inhibition, which refers to the ability to inhibit a prepotent response (Logan, 1994). This type of inhibition is often assessed by a go/no-go task or a stop-signal task (SST). (4) Oculomotor inhibition, which refers to effortful suppression of reflexive saccades. This type of inhibition is often assessed by the antisaccade task in which participants must resist reflexive eye movements towards a new peripheral target and move their eyes in the opposite direction. Although inhibition has been subdivided in these four types, Friedman and Miyake (2004) found that interference control, response inhibition and oculomotor inhibition are highly related and are part of a single latent variable.

Much research has focused on the link between response inhibition and OCD. For response inhibition there are both studies that find an impairment compared to controls (e.g., Abramovitch, Dar, Schweiger, & Hermesh, 2011; Menzies et al., 2007) and studies that find similar performance (e.g., Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Krishna et al., 2011). Abramovitch and Cooperman (2015) argue that the inconsistency in the results of response inhibition is partly due to its assessment. Studies assessing response inhibition with the SST generally show impairments compared to controls (e.g., de Wit et al., 2012; Menzies et al., 2007), while studies assessing response inhibition with the go/no-go task often report no difference in performance (e.g., Bohne et al., 2008; Krishna et al., 2011). Moreover, Snyder et al. (2014) showed that the effect size for OCD patients compared to controls was medium for stop-signal RTs and only small and nonsignificant for accuracy on the go/no-go task.

In a go/no-go task participants respond to series of go stimuli (for instance colored squares) but cannot respond to no-go stimuli (for instance red squares). From the start of the trial it is clear a participant should inhibit their response, which suggests that this task provides a measure of action suppression (Eagle, Bari, & Robbins, 2008). In a SST participants need to respond to a series of targets, however typically in 25% to 30% of the trials participants are presented with an auditory stop signal. The time between the presentation of the target and the stop signal is the stop signal delay and often starts at 250ms. When participants hear the signal they need to inhibit their response to the target. Response inhibition is then operationalized as Stop Signal Reaction Times (SSRTs). SSRTs are calculated based on the horse-race model (Logan, 1994). This model assumes that go and stopping processes compete. When the go process is faster than the stopping process this will result in failed response inhibition. When the stopping process is faster than the go process this will result in successful inhibition. Since the SST requires participants to inhibit an already prepared response, the SST is a measure of action cancellation (Eagle et al., 2008). The fact that an effect for inhibition is often found with the SST but not with the go/no-go task suggests that OCD has particular difficulties with action cancellation (as assessed by the SST) rather than action suppression (as assessed by the go/no-go task; Abramovitch & Cooperman, 2015; Eagle et al., 2008). Furthermore, the SST has a higher inhibitory load (Schachar et al., 2007) and has different underlying neural substrates than the go/no-go task (Eagle et al., 2008).

Chamberlain et al. (2005) have suggested that the repetitive obsessive thoughts and compulsions stem from a deficit in inhibition. They therefore considered inhibition to be an *endophenotype* of OCD. An endophenotype is considered a measurable component that connects a disease to the distal genotype (Gottesman & Gould, 2003). Endophenotypes are signs of genetic risk factors and are thus not influenced by current symptomatology or valence of stimuli. This trait view is supported by studies demonstrating that OCD patients and their healthy relatives perform similarly on inhibition (e.g., Menzies et al., 2007), OCD patients perform similarly in remission and pre- compared to post-treatment (e.g., Bannon, Gonsalvez, Croft, & Boyce, 2006; Braga et al., 2016).

In contrast, Abramovitch and Cooperman (2015) argue that studies generally find only small to moderate impairments in inhibition, meaning that a significant change in performance following treatment is unlikely. Nonetheless some studies did find improvement in neuropsychological performance after treatment (e.g., Andrés et al., 2008; Kuelz et al., 2006; Voderholzer et al., 2013). Furthermore, some studies show an association between the severity of OCD symptoms and neuropsychological functioning (e.g., Abramovitch et al., 2011; Trivedi et al., 2008). However, meta-analyses could not identify OCD symptoms severity as a consistent moderator for neuropsychological performance (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014). Abramovitch and Cooperman (2015) argue that methodological difficulties such as a variety of measures for OCD severity and restricted range might hinder finding an association between neuropsychological performance and symptom severity.

In line with the view that OCD symptoms may influence neuropsychological performance, Abramovitch, Dar, Hermesh, and Schweiger (2012) introduced the executive overload model in OCD (see Figure 1 for an overview). In this model the fronto-striatal hyperactivation and overflow of obsessive thoughts is due to continuous attempts to control automatic processes. Subsequently the overflow of obsessive thoughts consume cognitive resources leading to an overload of the executive system. This in turn leads to impairments on executive tasks such as response inhibition. When these neuropsychological impairments become evident for the patient (e.g., being late at appointments) this elicits fear of impulsivity and hence leads to further efforts to control automatic processes. Subsequently, this results in a vicious cycle in which these increased efforts evoke further obsessive thoughts, leading to increased overload to the executive system and more neuropsychological impairments.

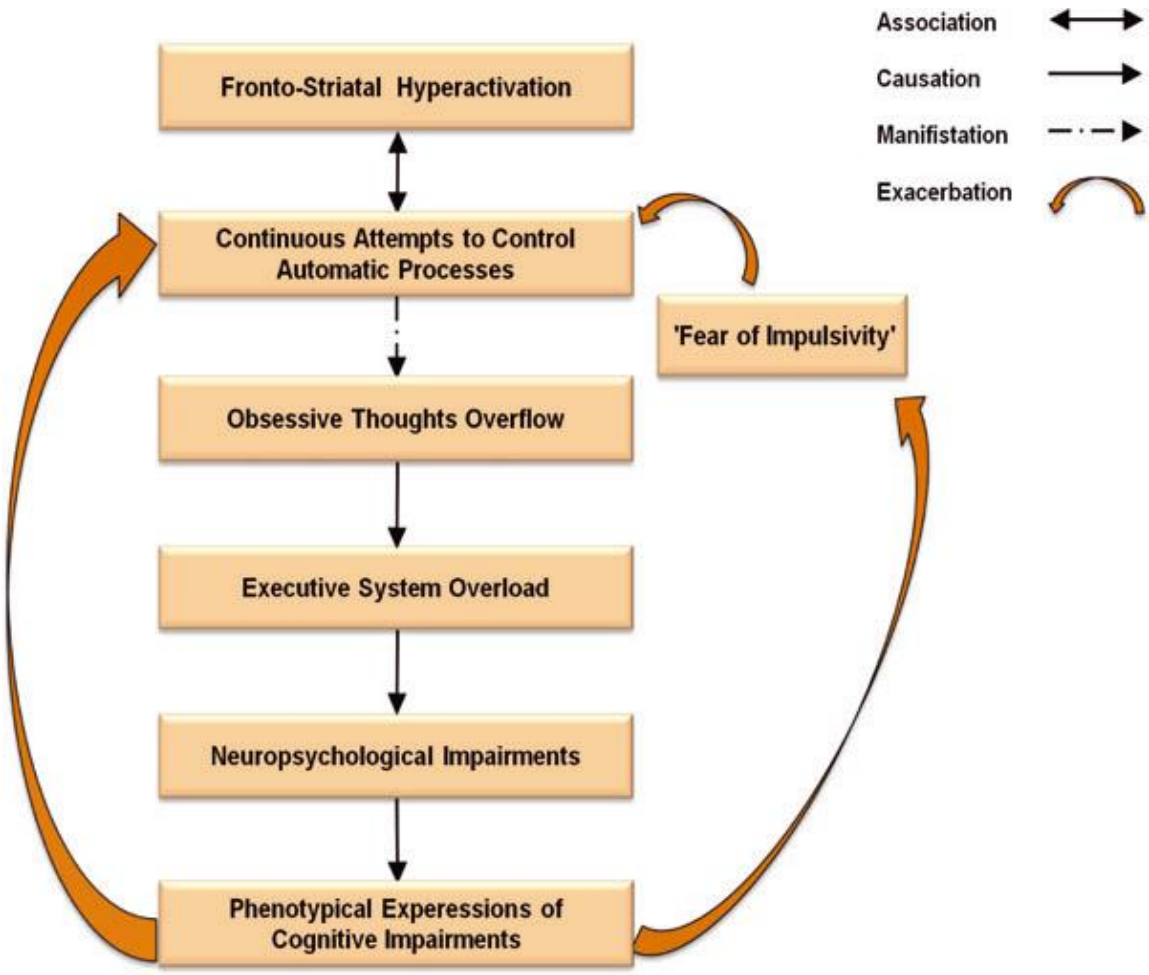

Figure 1. Executive overload model of Abramovitch et al. (2012). Reprinted from "Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD," by A. Abramovitch, R. Dar, H. Hermesh, & A. Schweiger, 2012, *Journal of Neuropsychology*, *6,* 161-191. Copyright © 2011 by John Wiley Sons, Inc. Reprinted by permission of John Wiley & Sons, Inc.

Another issue that has often been overlooked in research on the link between OCD and inhibition is the possibility that difficulties in inhibition could be exacerbated in the context of disorder-relevant stimuli. In line with this hypothesis, OCD patients experience difficulty to inhibit compulsive behavior (e.g., washing hands) in the context of specific stimuli (e.g., a family member), yet no difficulty inhibiting the same compulsive behavior in the context of other stimuli (e.g., a dog; Linkovski, Kalanthroff, Henik, & Anholt, 2016). Interestingly, Linkovski et al. (2016) set out to take the disorder-relevance of stimuli into account. In their first experiment they did not find an effect of

repeated checking and consequent decreased memory confidence on performance on a neutral SST. In their second experiment they added familiar stimuli as go signals in the SST and found that participants exhibited reduced accuracy in stopping trials to familiar stimuli compared to unfamiliar stimuli, regardless of whether they previously performed a repeated checking task or a simple action task. This suggests that at least familiarity, which often correlates with disorder-relevance, can have an effect on inhibition capacity. However, they found no effects on stop signal reaction times.

In summary, response inhibition is of specific interest in OCD. However, the literature concerning the role of inhibition in OCD is mixed. One of the issues in the literature is the state-trait debate. Some authors suggest response inhibition is an endophenotype and thus a trait of OCD (Chamberlain et al., 2005), which suggests that poor response inhibition would make someone more vulnerable to develop OCD and response inhibition is not influenced by current symptoms or type of stimuli. In contrast, the executive overload model (Abramovitch et al., 2012) suggests that current OCD symptoms can lead to an overload of the executive system, which subsequently leads to decreased response inhibition. Therefore, decreased inhibition could be a state rather than a trait marker in OCD. A second issue in the literature is the lack of studies taking valence-specificity into account. The few studies that take valence-specificity into account suggest that difficulties in inhibition could be larger in the context of disorder-relevant stimuli. In order to clarify these issues, experimental research to further elucidate the role of context-dependence and valence-specificity in inhibition in the context of OCD is necessary.

Selective Attention in OCD

Another factor that has been put forward as one of the mechanisms that contributes to the development and maintenance of OCD is selective attention (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007; Muller & Roberts, 2005). Selective attention refers to selectively attending to threatening stimuli over neutral stimuli. The theory of role of selective attention in OCD stems from theories on selective attention in anxiety. Although the DSM 5 (American Psychiatric Association, 2013) considers obsessive-compulsive and related disorders as a separate diagnostic class, OCD and anxiety disorders overlap in several substantial ways

(Abramowitz & Jacoby, 2015). OCD and anxiety disorders are both characterized by excessive irrational fear and avoidance behavior. In OCD compulsions are often performed in order to reduce obsessional anxiety. Thus they are functionally similar to avoidance behavior or safety-seeking strategies in anxiety disorders. Furthermore, similar to anxiety disorders, OCD is maintained by cognitive distortions and negative reinforcement. Moreover, for both anxiety disorders and OCD exposure-based therapy has proved to be one of the most effective treatment interventions. Based on these facts Abramowitz and Jacoby (2015) argue that OCD has mistakenly been identified as a separate diagnostic class. The merit of theoretical models of selective attention in anxiety for OCD is further corroborated by the meta-analysis of Bar-Haim et al. (2007) in which they found no significant difference in the results for attentional bias in OCD compared to anxiety disorders.

Based on their meta-analysis Bar-Haim et al. (2007) developed an integrative model on selective attention in anxiety. This model comprises four stages of threat processing. The first stage consists of pre-attentively evaluating stimuli in the environment. If stimuli are labeled as a threat, cognitive resources will be allocated to those stimuli in the next stage of processing, resulting in interruption of ongoing activity and a conscious anxious state. Subsequently, the context of the threat and available coping resources will be assessed and the threat will be compared with prior learning experiences and memory. Finally, if the stimulus is still labeled as a threat, current goals will be interrupted and attention will be oriented towards threat in the last stage of processing. Bar-Haim et al. (2007) suggest a role of selective attention in the etiology of disorders by arguing that disorders can stem from abnormalities in processing at different stages.

Van Bockstaele et al. (2014) investigated the empirical support for a causal relationship between selective attention and anxiety. In line with Bar-Haim et al. (2007), they concluded that selective attention is likely a cognitive vulnerability factor for anxiety based on studies that show that a change in selective attention can influence vulnerability to stress (e.g., Verhaak, Smeenk, van Minnen, & Kraaimaat, 2004). However, in contrast to Bar-Haim et al. (2007), Van Bockstaele et al. (2014) concluded that symptoms can also influence selective attention. For instance, Foa and McNally (1986) found that attentional bias towards threat was reduced after successful

exposure treatment in OCD patients. Since exposure therapy decreased OCD symptoms and this is in turn was followed by a decrease in selective attention, this study provides support for the idea that OCD symptoms can influence selective attention.

Studies investigating selective attention in the context of OCD have indeed found an attentional bias for OCD-related stimuli in subclinical or clinical OCD (e.g., Amir, Najmi, & Morrison, 2009; Lavy, Van Oppen, & Van Den Hout, 1994; Moritz, Von Muehlenen, Randjbar, Fricke, & Jelinek, 2009; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996). However, there have been multiple studies that failed to find an association between OCD and selective attention (e.g., Harkness, Harris, Jones, & Vaccaro, 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008). Due to the inconsistent literature on selective attention in OCD compared to the consistent findings in anxiety, Summerfeldt and Endler (1998) concluded that an attentional bias in OCD has only been reliably demonstrated in OCD with contamination concerns. Although the meta-analysis of Bar-Haim et al. (2007) found no significant difference between anxiety disorders and OCD in selective attention.

From the current literature on selective attention in OCD no conclusions can be drawn regarding the direction of causality. It is unclear whether selective attention influences OCD symptoms or whether OCD symptoms also influence selective attention. Research using prospective designs in order to examine the influence of selective attention on OCD symptoms is limited. A study that explicitly examined the link between selective attention and OCD in subclinical contamination fear participants found that an experimental reduction of attentional bias resulted in increased behavioral approach towards contamination stimuli (Najmi & Amir, 2010). This study suggests that attentional bias can have an effect on subsequent OCD symptoms.

Another issue in the literature on selective attention in OCD is content-specificity: is selective attention specific for disorder-congruent stimuli or for threatening stimuli in general? Pergamin-Hight, Naim, Bakermans-Kranenburg, van Ijzendoorn, and Bar-Haim (2015) aimed to address this issue in their meta-analysis. They found that attentional bias was specific for disorder-congruent stimuli in anxiety disorders. This suggests that selective attention could be affected by previous learning and memories. Type of anxiety disorder (i.e., post-traumatic stress disorder, panic disorder, social anxiety disorder and OCD) was not a significant moderator. However, it

is important to note that this meta-analysis only included four studies on OCD. Moreover, out of these four studies only one study found significant evidence for content-specificity. Therefore, more research on content-specificity in selective attention in the context of OCD is warranted.

Selective attention is often assessed in a dot probe task, in which two pictures are presented on a screen below and above a fixation cross. These pictures can consist of a pair of two neutral pictures or a pair of a threatening picture and a neural picture. After the pictures appeared a dot typically appears on one of the two locations where the pictures appeared previously. Selective attention is operationalized with several indices: The most often used index is the attentional bias score, the tendency to allocate attention to threatening stimuli over neutral stimuli. This index is usually calculated by subtracting the time it takes to respond to a dot when a threatening stimulus previously appeared on the same location (i.e., congruent trials) from the time it takes to respond to a dot when a neutral stimulus previously appeared on the same location (i.e., incongruent trials). Second, attentional interference is used, a measure that determines the extent that a threatening pictures interferes with attention allocation. This index is usually assessed by subtracting the time it takes to respond to dot when the previous pictures were both neutral from the time it takes to respond to incongruent trials. Traditionally, these measures were considered as relatively stable biases in time. However, the index of attentional bias and attentional interference have repeatedly shown unreliability. This suggests they are not stable traits, but rater dynamic processes (Rodebaugh et al., 2016). This led to the development of a third type of selective attention index: trial-level based (TL-BS) selective attention measures. For instance, attentional bias variability assesses attentional bias on trial level based on temporally contiguous trials and subsequently determines the variability of attentional bias. Similarly, Bradley et al. (2016) found no evidence of OCD symptoms predicting vigilance or delayed disengagement, but OCD symptoms did predict dynamic selective attention: the tendency to repeatedly re-orient and fixate upon OCD stimuli over time. To date, the research on attentional bias variability in the context of OCD is scarce. Therefore, this doctoral dissertation will consider attentional bias both with the traditional bias scores as a stable concept and with the new trial-level bias scores approach considering attentional bias as a dynamic process.

In summary, selective attention has been implicated in OCD. However, results regarding selective attention in OCD are mixed. Different views exist regarding the nature of the link between selective attention and OCD. Bar-Haim et al. (2007) suggest that selective attention is a vulnerability factor for OCD. This view is supported by Van Bockstaele et al. (2014), however Van Bockstaele et al. (2014) also discuss the possibility that symptoms can influence selective attention. The latter view has rarely been investigated. A second issue pertains to the content-specificity of selective attention. Pergamin-Hight et al. (2015) found that selective attention was specific for disorder-relevant stimuli. However, this meta-analysis included only four studies on OCD from which only one study actually reported evidence for content-specificity. Further research is necessary in order to elucidate the effect of content-specificity and OCD symptoms on selective attention.

Combined Cognitive Bias Hypothesis

To date, most research has investigated information processing factors in isolation. Muller and Roberts (2005) argued that information processing factors could interact in the etiology and maintenance of OCD symptoms. The notion of interacting combined cognitive biases has been further elaborated by Hirsch, Clark, and Mathews (2006). In their combined cognitive biases hypothesis they pose that cognitive biases can influence each another and/or can interact so that the effect of each bias separately on symptoms is influenced by other biases. Therefore combinations of biases should have a greater impact on disorders than information processing factors in isolation.

A theory that takes into account the interaction between information processing factors is the Attentional Control Theory (ACT; Eysenck, Derakshan, Santos, & Calvo, 2007). This theory poses that the effects of anxiety on attentional processes are pivotal in order to understand how anxiety affects cognitive performance. Internal (e.g., intrusive thoughts) or external (e.g., distressing pictures) threat stimuli direct attention towards the source of threat. This concept is related to the bottom-up attentional system, which is influenced by salient or threatening stimuli (internal or external; Corbetta & Shulman, 2002). Increased bottom-up capture (i.e., selective attention) subsequently decreases the control of the second attentional system: top-down

control. Top-down control is governed by current goals, expectations and knowledge (Corbetta & Shulman, 2002). Inhibition is one of the main functions underlying top-down control and is especially impaired when task demands on working memory are high. Furthermore, top-down control and bottom-up capture influence each other bidirectionally. For instance, decreased top-down control is subsequently more susceptible to influences of bottom-up capture. These bidirectional effects should be stronger under stressful conditions when anxiety levels are high. In contrast, individuals with high inhibition capacity may be less susceptible to influences of bottom-up capture.

Moreover, ACT distinguishes between effectiveness and efficiency of performance. The effectiveness of the performance refers to the quality of the task performance, for instance measured by accuracy. Efficiency refers to the effort necessary to establish the effectiveness of performance, for instance measured by reaction times. Effects of decreased top-down control are most evident on efficiency of performance.

However, in some cases increased bottom-up capture can be beneficial for cognitive performance. When threat-related stimuli are relevant to the cognitive task at hand and thus align with current goals, the interaction between bottom-up capture and top-down control should lead to enhanced cognitive performance.

In the context of OCD the bottom-up system is likely influenced by obsessive thoughts and selective attention to threat-related stimuli. This system could interact with inhibition capacity in the development and maintenance of OCD symptoms. For instance, the tendency to attend to obsessive thoughts and threat-related stimuli and a difficulty in response inhibition when confronted with such stimuli could exacerbate OCD symptoms. Furthermore, the ACT implies that the experience of OCD symptoms should increase selective attention to OCD-related stimuli and decrease inhibition capacity.

Research Objectives of the Dissertation

Based on the gaps in the literature described above, the current dissertation had three specific research aims that are crucial in the further understanding of the link between selective attention, inhibition and OCD symptoms.

(1) Examining whether information processing biases are stable or context-dependent. As described above, in the context of inhibition there is a state-trait debate. Some researchers have argued that inhibition should be considered as an endophenotype for OCD (Chamberlain et al., 2005), in which decreased inhibition capacity would make someone more vulnerable to develop OCD symptoms. However, Abramovitch et al. (2012) proposed a theoretical model in which current OCD symptoms can also influence inhibition capacity. Also in the context of selective attention, Bar-Haim et al. (2007) implicated selective attention in the etiology of anxiety disorders. However, Van Bockstaele et al. (2014) suggested that symptoms can also have an effect on selective attention. In order to investigate the context-dependence of inhibition and selective attention, this dissertation will compare inhibition and selective attention during neutral conditions to its assessment in the context of elicited OCD symptoms.

(2) Understanding the role of general vs. valence-specific biases. As described above, there has been debate on whether there is a general impairment in inhibition or selective attention towards generally negative stimuli or whether these biases are specific for disorder-relevant stimuli. According to ACT (Eysenck et al., 2007) disorder-relevant stimuli should have a stronger effect compared to neutral stimuli. In order to examine valence-specificity, the studies in this dissertation will compare selective attention and inhibition in the context of generally negative stimuli to performance in the context of disorder-relevant stimuli.

(3) Testing whether OCD symptoms are best predicted by single or multiple information processing biases. Muller and Roberts (2005) and Hirsch et al. (2006) have posed that information processing factors can interact in the development and maintenance of symptoms. An interaction between inhibition and selective attention could have a stronger effect on OCD symptoms than these factors in isolation. For instance, it is plausible that selective attention to threat-related stimuli is particularly harmful when individuals have difficulties in inhibition. In this dissertation the link between response inhibition, selective attention, their interaction and OCD symptoms will be clarified by examining the predictive value of these factors on OCD symptoms prospectively.

Overview of the Chapters

As OCD symptoms are prevalent in non-clinical populations, OCD can be meaningfully studied in analogue samples (Abramowitz et al., 2014). Moreover, Abramowitz et al. (2014) argued OCD symptoms are dimensional instead of categorical, are phenomenologically similar, and have similar etiological and maintenance factors in clinical and non-clinical populations. Therefore non-clinical and subclinical populations are well-suited for research on the mechanisms of OCD and will be used throughout this dissertation.

In order to be able to investigate context-dependence of inhibition and selective attention (research aim 1), there is a need for ethical and efficacious OCD symptom induction procedures. There are many procedures available in order to elicit temporary OCD symptoms, however a comprehensive review on these procedures is lacking. Therefore, **chapter 2** consists of a meta-analysis of different induction procedures of OCD symptoms. For this meta-analysis the efficacy of these procedures was investigated in clinical and nonclinical participants over different moderators. The moderators included different induction categories (i.e., threat-related material, disgust, mental contamination, perfectionism/certainty, responsibility, thought-action fusion, and performing compulsions), symptom dimensions of OCD (i.e., checking, contamination fear, symmetry, or general OCD regardless of symptoms dimension), modalities of presentation (i.e., verbal, visual, objects, behavior, or a combination of these), and level of individual tailoring. The meta-analysis included 4900 participants across 90 studies. Based on this meta-analysis one of the best procedures to elicit current OCD symptoms in nonclinical participants consisted of the mental contamination category.

In **chapter 3** we examined the link between OCD symptoms (research aim 1), OCD-related stimuli (research aim 2) and response inhibition. In order to investigate the effects of current OCD symptoms on response inhibition, the stop-signal task was administered before and after either an OCD symptom induction (n = 43) or a neutral mood induction (n = 40). In order to investigate whether underperformance on the stop-signal task would be specific for OCD-related stimuli, the stop-signal task included neutral, generally negative and OCD-related stimuli. Moreover, trait OCD symptoms were taken into account by comparing participants scoring high (n = 39) and low (n =

44) on contamination fear. Furthermore, we examined whether baseline inhibition capacity could predict the change in symptoms after an OCD symptom induction.

In **Chapter 4** context-dependence (research aim 1) and valence-specificity (research aim 2) was investigated in the context of selective attention. This chapter consists of two studies. The first study cross-sectionally investigated the effect of trait OCD symptoms on selective attention by comparing students scoring high (*n* = 32) and low (*n* = 32) on contamination fear on their performance on a dot probe task. The second study investigated the effect of current OCD symptoms by administering a dot probe task before and after either an induction of OCD symptoms (*n* = 35) or a neutral mood induction (*n* = 33). Furthermore, the second study investigated whether baseline selective attention for OCD-related stimuli could predict the change in symptoms after an OCD symptoms induction. In these studies selective attention was both considered as a dynamic process in time by looking at attentional bias at trial level and as a stable concept by looking at traditional attentional bias and interference scores.

Chapter 5 examined the predictive unique and interactive effects of selective attention and response inhibition on OCD symptoms (research aim 3). This was investigated by checking whether selective attention, response inhibition and their interaction can predict OCD symptoms over and above obsessive beliefs. Baseline OCD symptoms, selective attention, response inhibition and obsessive beliefs were assessed in students (*n* = 89) during a first session in the beginning of the semester. The influence on OCD symptoms was examined by an OCD symptom induction during the first session and questionnaires during the examination period (68 to 80 days after the first session). The examination period is a period of heightened stress, which is associated with OCD symptoms (Coles & Horng, 2006), and is therefore suitable as a more ecologically valid induction.

Finally, in **chapter 6** the main findings and implications from all chapters will be discussed, limitations of this dissertation and suggestions for future research are outlined.

References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163-1171. doi: 10.1016/j.cpr.2013.09.004

Abramovitch, A., & Cooperman, A. (2015). The cognitive neuropsychology of obsessive-compulsive disorder: A critical review. *Journal of Obsessive-Compulsive and Related Disorders, 5*, 24-36. doi: 10.1016/j.jocrd.2015.01.002

Abramovitch, A., Dar, R., Hermesh, H., & Schweiger, A. (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD. *Journal of Neuropsychology, 6*(2), 161-191. doi: 10.1111/j.1748-6653.2011.02021.x

Abramovitch, A., Dar, R., Schweiger, A., & Hermesh, H. (2011). Neuropsychological impairments and their association with obsessive-compulsive symptom severity in obsessive-compulsive disorder. *Archives of Clinical Neuropsychology*. doi: 10.1093/arclin/acr022

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

Abramowitz, J. S., & Jacoby, R. J. (2015). Obsessive-compulsive and related disorders: A critical review of the new diagnostic class. *Annual Review of Clinical Psychology, 11*(1), 165-186. doi: 10.1146/annurev-clinpsy-032813-153713

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive–compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157. doi: 10.1016/j.brat.2008.10.020

Andrés, S., Lázaro, L., Salamero, M., Boget, T., Penadés, R., & Castro-Fornieles, J. (2008). Changes in cognitive dysfunction in children and adolescents with obsessive-compulsive disorder after treatment. *Journal of Psychiatric Research, 42*(6), 507-514. doi: 10.1016/j.jpsychires.2007.04.004

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Ball, S. G., Baer, L., & Otto, M. W. (1996). Symptom subtypes of obsessive-compulsive disorder in behavioral treatment studies: A quantitative review. *Behaviour Research and Therapy, 34*(1), 47-51. doi: 10.1016/0005-7967(95)00047-2

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive–compulsive disorder: State or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11-12), 1031-1038. doi: 10.1080/j.1440-1614.2006.01928.x

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1-24. doi: 10.1037/0033-2909.133.1.1

Bloch, M. H., Landeros-Weisenberger, A., Rosario, M. C., Pittenger, C., & Leckman, J. F. (2008). Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry, 165*(12), 1532-1542. doi: 10.1176/appi.ajp.2008.08020320

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive–compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141-150. doi: 10.1016/j.jpsychires.2006.11.008

Bolton, D., Raven, P., Madronal-Luque, R., & Marks, I. M. (2000). Neurological and neuropsychological signs in obsessive compulsive disorder: Interaction with behavioural treatment. *Behaviour Research and Therapy, 38*(7), 695-708. doi: 10.1016/S0005-7967(99)00139-4

Bradley, M. C., Hanna, D., Wilson, P., Scott, G., Quinn, P., & Dyer, K. F. W. (2016). Obsessive–compulsive symptoms and attentional bias: An eye-tracking methodology. *Journal of Behavior Therapy and Experimental Psychiatry, 50*, 303-308. doi: 10.1016/j.jbtep.2015.10.007

Braga, D. T., Abramovitch, A., Fontenelle, L. F., Ferrão, Y. A., Gomes, J. B., Vivan, A. S., . . . Cordioli, A. V. (2016). Neuropsychological predictors of treatment response to

cognitive behavioral group therapy in obesssive-compulsive disorder. *Depression and Anxiety, 33*(9), 848-861. doi: 10.1002/da.22509

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience & Biobehavioral Reviews, 29*(3), 399-419. doi: 10.1016/j.neubiorev.2004.11.006

Coles, M. E., & Horng, B. (2006). A prospective test of cognitive vulnerability to obsessive-compulsive disorder. *Cognitive Therapy and Research, 30*(6), 723-734. doi: 10.1007/s10608-006-9033-x

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201-215.

D'Alcante, C. C., Diniz, J. B., Fossaluza, V., Batistuzzo, M. C., Lopes, A. C., Shavitt, R. G., . . . Hoexter, M. Q. (2012). Neuropsychological predictors of response to randomized treatment in obsessive–compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 39*(2), 310-317. doi: 10.1016/j.pnpbp.2012.07.002

de Wit, S. J., de Vries, F. E., van der Werf, Y. D., Cath, D. C., Heslenfeld, D. J., Veltman, E. M., . . . van den Heuvel, O. A. (2012). Presupplementary motor area hyperactivity during response inhibition: A candidate endophenotype of obsessive-compulsive disorder. *American Journal of Psychiatry, 169*(10), 1100-1108. doi: 10.1176/appi.ajp.2012.12010073

Dek, E. C. P., van den Hout, M. A., Giele, C. L., & Engelhard, I. M. (2010). Repeated checking causes distrust in memory but not in attention and perception. *Behaviour Research and Therapy, 48*(7), 580-587. doi: 10.1016/j.brat.2010.03.009

Eagle, D. M., Bari, A., & Robbins, T. W. (2008). The neuropsychopharmacology of action inhibition: Cross-species translation of the stop-signal and go/no-go tasks. *Psychopharmacology, 199*(3), 439-456. doi: 10.1007/s00213-008-1127-6

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353. doi: 10.1037/1528-3542.7.2.336

Farris, S. G., McLean, C. P., Van Meter, P. E., Simpson, H. B., & Foa, E. B. (2013). Treatment response, symptom remission and wellness in obsessive-compulsive disorder. *The Journal of clinical psychiatry, 74*(7), 685-690. doi: 10.4088/JCP.12m07789

Foa, E. B., & McNally, R. J. (1986). Sensitivity to feared stimuli in obsessive-compulsives: A dichotic listening analysis. *Cognitive Therapy and Research, 10*(4), 477-485. doi: 10.1007/bf01173299

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology: General, 133*(1), 101-135.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160*(4), 636-645. doi: 10.1176/appi.ajp.160.4.636

Harkness, E. L., Harris, L. M., Jones, M. K., & Vaccaro, L. (2009). No evidence of attentional bias in obsessive compulsive checking on the dot probe paradigm. *Behaviour Research and Therapy, 47*(5), 437-443. doi: 10.1016/j.brat.2009.02.004

Hermans, D., Engelen, U., Grouwels, L., Joos, E., Lemmens, J., & Pieters, G. (2008). Cognitive confidence in obsessive-compulsive disorder: Distrusting perception, attention and memory. *Behaviour Research and Therapy, 46*(1), 98-113. doi: 10.1016/j.brat.2007.11.001

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223-236. doi: 10.1016/j.beth.2006.02.001

Krishna, R., Udupa, S., George, C. M., Kumar, K. J., Viswanath, B., Kandavel, T., . . . Reddy, Y. C. J. (2011). Neuropsychological performance in OCD: A study in medication-naïve patients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 35*(8), 1969-1976. doi: 10.1016/j.pnpbp.2011.09.009

Kuelz, A. K., Riemann, D., Halsband, U., Vielhaber, K., Unterrainer, J., Kordon, A., & Voderholzer, U. (2006). Neuropsychological impairment in obsessive-compulsive disorder—improvement over the course of cognitive behavioral treatment.

Journal of Clinical and Experimental Neuropsychology, 28(8), 1273-1287. doi: 10.1080/13803390500507246

Langlois, F., Freeston, M. H., & Ladouceur, R. (2000). Differences and similarities between obsessive intrusive thoughts and worry in a non-clinical population: Study 1. *Behaviour Research and Therapy, 38*(2), 157-173. doi: 10.1016/S0005-7967(99)00027-3

Lavy, E., Van Oppen, P., & Van Den Hout, M. (1994). Selective processing of emotional information in obsessive compulsive disorder. *Behaviour Research and Therapy, 32*(2), 243-246. doi: 10.1016/0005-7967(94)90118-X

Leckman, J. F., Grice, D. E., Boardman, J., Zhang, H. P., Vitale, A., Bondi, C., . . . Pauls, D. L. (1997). Symptoms of obsessive-compulsive disorder. *American Journal of Psychiatry, 154*(7), 911-917.

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of Anxiety Disorders, 30*, 48-58. doi: 10.1016/j.janxdis.2014.12.016

Linkovski, O., Kalanthroff, E., Henik, A., & Anholt, G. E. (2016). Stop checking: Repeated checking and its effects on response inhibition and doubt. *Journal of Behavior Therapy and Experimental Psychiatry, 53*, 84-91. doi: 10.1016/j.jbtep.2014.12.007

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory and language*. San Diego, CA: Academic Press.

Macdonald, P. A., Antony, M. M., Macleod, C. M., & Richter, M. A. (1997). Memory and confidence in memory judgments among individuals with obsessive compulsive disorder and non-clinical controls. *Behaviour Research and Therapy, 35*(6), 497-505. doi: 10.1016/S0005-7967(97)00013-2

MacLeod, C. M. (1991). Half a century of research on the stroop effect: An integrative review. *Psychological Bulletin, 109*(2), 163-203. doi: 10.1037/0033-2909.109.2.163

Markarian, Y., Larson, M. J., Aldea, M. A., Baldwin, S. A., Good, D., Berkeljon, A., . . . McKay, D. (2010). Multiple pathways to functional impairment in obsessive–

compulsive disorder. *Clinical Psychology Review, 30*(1), 78-88. doi: 10.1016/j.cpr.2009.09.005

Mataix-Cols, D., do Rosario-Campos, M. C., & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *American Journal of Psychiatry, 162*(2), 228-238. doi: 10.1176/appi.ajp.162.2.228

McKay, D., Abramowitz, J. S., Calamari, J. E., Kyrios, M., Radomsky, A., Sookman, D., . . . Wilhelm, S. (2004). A critical evaluation of obsessive-compulsive disorder subtypes: Symptoms versus mechanisms. *Clinical Psychology Review, 24*(3), 283-313. doi: 10.1016/j.cpr.2004.04.003

Melloni, M., Urbistondo, C., Sedeño, L., Gelormini, C., Kichic, R., & Ibanez, A. (2012). The extended fronto-striatal model of obsessive compulsive disorder: Convergence from event-related potentials, neuropsychology and neuroimaging. *Frontiers in Human Neuroscience, 6*(259). doi: 10.3389/fnhum.2012.00259

Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C.-H., del Campo, N., . . . Bullmore, E. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*(12), 3223-3236. doi: 10.1093/brain/awm205

Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions. *Current Directions in Psychological Science, 21*(1), 8-14. doi: 10.1177/0963721411429458

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Moritz, S., Fischer, B. K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., & Jelinek, L. (2008). Words may not be enough! No increased emotional Stroop effect in obsessive–compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104. doi: 10.1016/j.brat.2008.05.005

Moritz, S., Von Muehlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive–compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371. doi: 10.1017/S1355617709090511

Moritz, S., & von Mühlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229. doi: 10.1002/da.20294

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive–Compulsive Disorder: A review. *Journal of Anxiety Disorders, 19*(1), 1-28. doi: 10.1016/j.janxdis.2003.12.001

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126*(2), 220-246. doi: 10.1037//0033-2909.126.2.220

Pergamin-Hight, L., Naim, R., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., & Bar-Haim, Y. (2015). Content specificity of attention bias to threat in anxiety disorders: A meta-analysis. *Clinical Psychology Review, 35*, 10-18. doi: 10.1016/j.cpr.2014.10.005

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy, 35*(9), 793-802. doi: 10.1016/S0005-7967(97)00040-5

Rachman, S. (1998). A cognitive theory of obsessions: Elaborations. *Behaviour Research and Therapy, 36*(4), 385-401. doi: 10.1016/S0005-7967(97)10041-9

Rachman, S., & de Silva, P. (1978). Abnormal and normal obsessions. *Behaviour Research and Therapy, 16*(4), 233-248. doi: 10.1016/0005-7967(78)90022-0

Rodebaugh, T. L., Scullin, R. B., Langer, J. K., Dixon, D. J., Huppert, J. D., Bernstein, A., . . . Lenze, E. J. (2016). Unreliability as a threat to understanding psychopathology: The cautionary tale of attentional bias. *Journal of Abnormal Psychology, 125*(6), 840-851. doi: 10.1037/abn0000184

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/mp.2008.94

Salkovskis, P. M. (1985). Obsessional-compulsive problems: A cognitive-behavioural analysis. *Behaviour Research and Therapy, 23*(5), 571-583. doi: 10.1016/0005-7967(85)90105-6

Schachar, R., Logan, G. D., Robaey, P., Chen, S., Ickowicz, A., & Barr, C. (2007). Restraint and cancellation: Multiple inhibition deficits in attention deficit hyperactivity

disorder. *Journal of Abnormal Child Psychology, 35*(2), 229-238. doi: 10.1007/s10802-006-9075-2

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: A meta-analysis. *Psychological Medicine, 44*(6), 1121-1130. doi: 10.1017/S0033291713001803

Skapinakis, P., Caldwell, D., Hollingworth, W., Bryden, P., Fineberg, N., Salkovskis, P., . . . Lewis, G. (2016). A systematic review of the clinical effectiveness and cost-effectiveness of pharmacological and psychological interventions for the management of obsessive-compulsive disorder in children/adolescents and adults. *Health Technology Assessment, 20*(43), 1-392. doi: 10.3310/hta20430

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2014). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science*. doi: 10.1177/2167702614534210

Summerfeldt, L. J., & Endler, N. S. (1998). Examining the evidence for anxiety-related cognitive biases in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 12*(6), 579-598. doi: 10.1016/S0887-6185(98)00035-8

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in obsessional compulsive disorder. *Behaviour Research and Therapy, 34*(1), 53-60. doi: 10.1016/0005-7967(95)00041-U

Trivedi, J. K., Dhyani, M., Goel, D., Sharma, S., Singh, A. P., Sinha, P. K., & Tandon, R. A. (2008). Neurocognitive dysfunction in patients with obsessive compulsive disorder. *African Journal of Psychiatry, 11*(3), 204-209.

Van Bockstaele, B., Verschuere, B., Tibboel, H., De Houwer, J., Crombez, G., & Koster, E. H. W. (2014). A review of current evidence for the causal impact of attentional bias on fear and anxiety. *Psychological Bulletin, 140*(3), 682-721. doi: 10.1037/a0034834

Verhaak, C. M., Smeenk, J. M. J., van Minnen, A., & Kraaimaat, F. W. (2004). Neuroticism, preattentive and attentional biases towards threat, and anxiety before and after a severe stressor: A prospective study. *Personality and Individual Differences, 36*(4), 767-778. doi: 10.1016/S0191-8869(03)00131-4

Voderholzer, U., Schwartz, C., Freyer, T., Zurowski, B., Thiel, N., Herbst, N., . . . Kuelz, A. K. (2013). Cognitive functioning in medication-free obsessive-compulsive

patients treated with cognitive-behavioural therapy. *Journal of Obsessive-Compulsive and Related Disorders, 2*(3), 241-248. doi: 10.1016/j.jocrd.2013.03.003

CHAPTER 2

OBSESSIONS AND COMPULSIONS IN THE LAB: A META-ANALYSIS OF PROCEDURES TO INDUCE SYMPTOMS OF OBSESSIVE-COMPULSIVE DISORDER[1]

ABSTRACT

Efficacious induction procedures of symptoms of obsessive-compulsive disorder (OCD) are necessary in order to test central tenets of theories on OCD. However, the efficacy of the current range of induction procedures remains unclear. Therefore, this meta-analysis set out to examine the efficacy of induction procedures in participants with and without OCD symptoms. Moreover, we explored whether the efficacy varied across different moderators (i.e., induction categories, symptom dimensions of OCD, modalities of presentation, and level of individual tailoring). In total we included 4900 participants across 90 studies. The analyses showed that there was no difference in studies using subclinical and clinical participants, confirming the utility of analogue samples. Induction procedures evoked more symptoms in (sub)clinical OCD than in healthy participants, which was most evident in the contamination symptom dimension of OCD. Analysis within (sub)clinical OCD showed a large effect size of induction procedures, especially for the threat and responsibility category and when stimuli were tailored to individuals. Analysis within healthy participants showed a medium effect size of induction procedures. The magnitude of the effect in healthy individuals was stronger for mental contamination, thought-action fusion and threat inductions.

[1] Based on De Putter, L. M. S., Van Yper, L., & Koster, E. H. W. (2017). Obsessions and compulsions in the lab: A meta-analysis of procedures to induce symptoms of obsessive-compulsive disorder. *Clinical Psychology Review*, *52*, 137-147. doi: 10.1016/j.cpr.2017.01.001

Introduction

Obsessive-compulsive disorder (OCD) is an impairing and persistent disorder characterized by obsessions and/or compulsions (American Psychiatric Association; APA, 2013). Its lifetime prevalence is 2-3.5%, making it the fourth most common mental disorder with high economic and societal costs (Angst et al., 2004; Rasmussen & Eisen, 1992; Ruscio, Stein, Chiu, & Kessler, 2010). Obsessions consist of images or thoughts that are experienced as intrusive and often evoke anxiety and distress (APA, 2013). Compulsions are defined as repetitive actions that occur either internally (e.g., repetitive counting) or externally (e.g., excessive hand washing). In order to reduce anxiety, patients with OCD use a variety of compulsions. However compulsions can also be performed independently from obsessions (APA, 2013). Although there are many efficacious psychological and pharmacological treatments for OCD, many patients suffer from symptoms even after undergoing treatment (Fisher & Wells, 2005).

In order to advance treatments, improved understanding of OCD is required. A key prerequisite for developing and testing theories of OCD is the ability to induce symptoms of OCD in laboratory settings. This is paramount in order to study OCD symptom elicitation, regulation, and their psychological as well as neurological correlates in a controlled environment (Abramovitch & Cooperman, 2015). For instance, there has been a long standing state-trait debate in neuropsychological dysfunctions in OCD, in which it is unclear whether a neuropsychological deficit precedes the development of OCD or whether OCD symptoms cause neuropsychological deficits (Abramovitch & Cooperman, 2015). Such debates can only be resolved by research using carefully considered symptom provocation paradigms. Although a wide variety of symptom provocation procedures have been used across studies, there is no systematic review examining and comparing the efficacy of these different procedures in inducing OCD symptoms. Therefore, there is currently no systematic evaluation of how successful these induction procedures are relative to each other in inducing symptoms in different populations. This is problematic since there is substantial heterogeneity in induction procedures that are used and their efficacy. For research purposes, it would be interesting to have a clear overview on which procedures currently exist and how they compare to other procedures in terms of efficacy in order to allow optimal induction of symptoms in the lab. Furthermore, the issue of efficacious provocation

procedures is not merely relevant for studies on OCD patients. Abramowitz et al. (2014) highlighted the importance of analogue studies using samples of subclinical participants or even healthy samples to advance our knowledge of clinical OCD. Moreover, some OCD symptom inductions have been designed to provoke OCD symptoms even in healthy participants (e.g., Mataix-Cols, Lawrence, Wooderson, Speckens, & Phillips, 2009). Therefore the current meta-analysis sought to determine which procedures are most efficacious in inducing OCD-related symptoms in samples with and without OCD symptoms. Below, we start by identifying different categories of inductions that are being used, which will serve as a key moderator. Finally, we will describe the approach of the current meta-analysis.

Categories of Provocation Procedures

Based on the current literature we identified seven categories of provocation procedures. The first category is presenting threat-related material. This method has been used by the first studies investigating OCD during the experience of symptoms (e.g., Breiter et al., 1996; McGuire et al., 1994; Rauch et al., 1994; Zohar et al., 1989). In this category OCD symptoms are elicited by exposing individuals to stimuli that are directly related to concerns typical for OCD. Stimuli can be tailored to individuals to match their OCD-related concerns (e.g., OCD patients can take pictures of their triggers; Schienle, Schäfer, Stark, Walter, & Vaitl, 2005). However, standardized procedures are also commonly used, for instance a standardized picture set for every symptom dimension of OCD was designed by Mataix-Cols et al. (2009).

The second category is disgust induction. Disgust is characterized by a typical, universal physiological response, facial expression, and withdrawal/avoidance pattern (Rozin & Fallon, 1987). The triggers of disgust are largely universal and easily identifiable. They often include representations related to animals, bodily products and/or decay (Rachman, 1994). One could argue that disgust is related to the threat category for contamination fear. Contamination fear is frequently present in OCD (e.g., Olatunji, Cisler, McKay, & Phillips, 2010). For instance, feces are both disgusting and hold the potential to jeopardize one's health by contamination. However, in contrast to contamination fears, in disgust over and above any possible harm, the item itself is offensive (Rozin & Fallon, 1987). For instance a cockroach fully sterilized which could

not carry any possible diseases would still evoke disgust in most people (Rozin & Fallon, 1987).

The third category is mental contamination. More recently and related to general disgust, OCD symptoms are evoked by mental contamination induction procedures. Mental contamination is a sense of internal dirtiness which has only an indirect connection with soiled material and often emerges in the absence of psychical contact (for an overview Rachman, 2004b). It is often characterized by a moral element (Rachman, 2004b). In this category we will focus on studies with a moral element that evoke OCD symptoms. Typical inductions for the mental contamination category include guilt scenarios (i.e., recalling a memory in which you felt very guilty; e.g., Shin et al., 2000) and the non-consensual kiss paradigm (i.e., imagining that someone tries to kiss you without your consent; e.g., Fairbrother, Newth, & Rachman, 2005).

The fourth category is perfectionism/certainty, a factor empirically derived by the Obsessive-Compulsive Cognitions Working Group (OCCWG, 2005). This category consists of two related phenomena: (1) the feeling of incompleteness and (2) the feeling of uncertainty. The feeling of incompleteness has also been described as "not just right experiences": the uncomfortable sensation that something (i.e., actions, intentions or perception) is not just right or fundamentally imperfect/incomplete (Coles, Frost, Heimberg, & Rheaume, 2003; Summerfeldt, Kloosterman, Antony, & Swinson, 2014). Related to not just right experiences is intolerance of uncertainty, which has often been associated with OCD (Gentes & Ruscio, 2011). The OCCWG (2001) defines intolerance of uncertainty as "beliefs about the necessity for being certain, that one has poor capacity to cope with unpredictable change, and that it is difficult to function adequately in ambiguous situations" (p. 1004). Examples of inductions in this category include false feedback on memory trials (e.g., Alcolado & Radomsky, 2011), viewing a cluttered table (e.g., Cougle, Fitch, Jacobson, & Lee, 2013) and a visual search task with target absent trials (e.g., Toffolo, van den Hout, Hooge, Engelhard, & Cath, 2013).

The fifth category is responsibility. The OCCWG (2001) describes this cognitive factor as "the belief that one has power that is pivotal to bring about or prevent subjectively crucial negative outcomes. These outcomes are perceived as essential to prevent and may have consequences in the real world and/or moral level" (p. 1002-

1003). According to the cognitive theory of Salkovskis (1999) the interpretation of obsessions as an indication that one might be responsible for harm if threat is not prevented leads to adverse mood and urge to engage in compulsions. Subsequently, adverse mood and compulsions increase the likelihood of further obsessions, perceived threat and interpretation of responsibility. Thus responsibility is thought to play a pivotal role in the maintenance of OCD. Examples of inductions for the responsibility category include signing a contract that the patient is fully responsible for any consequences (e.g., Radomsky, Rachman, & Hammond, 2001) and classifying capsules in different colors in order to develop a system that makes the distribution of medication safer (e.g., Arntz, Voncken, & Goosen, 2007).

The sixth category is thought-action fusion (TAF), which is closely related to another empirically derived cognitive factor by the OCCWG (2005): "control of thoughts". The underlying premise of TAF is that having a certain thought is equivalent to doing the act itself (moral TAF) or increases the likelihood of its occurrence (likelihood TAF; OCCWG, 1997). Note that many provocations by means of TAF could also be considered as mental contamination provocations. For instance, the TAF induction "I hope I have sex with my brother" used in a study by Berman, Abramowitz, Pardue, and Wheaton (2010) is morally repellent and could induce feelings of contamination. However in the current meta-analysis we considered TAF separately from mental contamination since all TAF inductions strongly rely on the premise that having a certain thought is equivalent to doing the act itself, which is not a necessary condition for mental contamination. The most typical example of a TAF induction is to instruct participants to write "I hope (name of a loved one) is in a car accident" (e.g., Rassin, 2001).

The seventh category is performing compulsions. Deacon and Maack (2008) pose that performing compulsions (such as hand washing) can exacerbate symptoms by increasing selective attention toward potential threat, since performing compulsions requires attentional allocation to a potential threat. Increased perception of threat could subsequently lead to threat overestimation and increased OCD symptoms (Deacon & Maack, 2008). Other studies found that repeated checking causes cognitive distrust (Boschen & Vuksanovic, 2007; Hermans et al., 2008; Hermans, Martens, De Cort, Pieters, & Eelen, 2003), which could similarly lead to an exacerbation of OCD

symptoms. Examples of inductions by performing compulsions are a task in which participants have to check a stove (e.g., Cougle et al., 2013) or instructing participants to perform safety behaviors for one week (e.g., Olatunji, Etzel, Tomarken, Ciesielski, & Deacon, 2011).

The Present Meta-analysis

In the current meta-analysis we examine the efficacy of induction procedures in eliciting OCD-related symptoms in healthy individuals and individuals with elevated OCD symptoms (subclinical and clinical). Since it is plausible that depending on diagnostic status participants respond differently to induction procedures, we will first test whether the between-group comparison of OCD patients and subclinical participants directly compared to a healthy control group are significant. If these effects are significant, this would indicate that depending on diagnostic status there is indeed a differential response. We will also test whether this effect is different depending on subclinical or clinical status. If this effect is different, OCD, subclinical and healthy participants will all be analyzed separately in the within-group analyses. If this effect is not significant, this would indicate that OCD and subclinical participants respond similarly to inductions and can be grouped together in the within-subjects analyses.

Category of induction will be included as one of four moderators that could be important for the effect of induction procedures. The second moderator is level of individual tailoring, where we will test if inductions that are tailored to participants are more efficacious than standardized inductions. To date, studies have suggested that this moderator can have a significant effect on induction procedures (Baioui et al., 2013; Morgiève et al., 2014; Schienle et al., 2005). Symptom dimension of OCD is another factor that might influence the effect of induction procedures and has therefore been included as the third moderator. OCD is a phenotypic heterogeneous disorder and the content of obsessions and/or compulsions can vary substantially. Within symptom dimensions the content of obsessions and/or compulsions is more consistent (Mataix-Cols, do Rosario-Campos, & Leckman, 2005). In line with the symptom dimensions identified by Mataix-Cols et al. (2005) in this meta-analysis we will distinguish between the contamination dimension, checking dimension, symmetry dimension and, if no specific symptom dimension was targeted, general OCD. Finally,

provided that in the literature on threat processing several studies have found differences in verbal versus pictorial information (Lees, Mogg, & Bradley, 2005; Reinholdt-Dunne, Mogg, & Bradley, 2009; Stormark & Torkildsen, 2004), we will include modality of presentation as a moderator.

Method

Literature Search

Electronic databases (Web of Science and Pubmed) were searched on July 31, 2015 to identify studies. The following key words were used: *OCD*, *obsessi** or **compuls**, combined with *symptom** and *provo**, *induc**, *elicit**, *thought-action fusion*, *TAF, mental contam** or *disgust**. Furthermore, relevant reviews that were found through the search (Atmaca, 2013; Chapman & Anderson, 2013; Haynes & Mallet, 2010; Kwon, Jang, Choi, & Kang, 2009; Linden, 2006; Rachman, 2010; Rotge et al., 2009; Shafran & Rachman, 2004) and reference lists of selected articles were screened for additional relevant studies.

Inclusion Criteria

Studies were selected if they met the following inclusion criteria: (a) the study was a published or in press journal article (peer-reviewed) written in English; (b) studies included an induction of OCD symptoms in adults; (c) studies assessed OCD symptoms (e.g., general OCD symptoms, anxiety/distress, obsessions, urge to indulge in compulsions and performing compulsions) after the induction in a within-subjects or between-subjects design; (d) data allowing the computation of effect sizes was available or available upon request.

Studies with general negative mood inductions or hoarding were excluded. General negative mood inductions were not included because the purpose of the meta-analysis was to examine inductions of specific relevance to OCD. Hoarding inductions were excluded since according to APA (2013) this is no longer considered a symptom dimension of OCD. Finally, studies investigating an increase in OCD symptoms after medication were excluded. Healthy samples, subclinical samples, and clinical samples were allowed as long as an OCD induction took place. Furthermore, comorbidity was allowed as long as the primary focus of the article was on the induction of OCD

symptoms. When symptom provocation was measured before and after treatment, only the measures before treatment were used to avoid effects of habituation. When both within-subjects and between-subjects comparisons were available for participants of the same diagnostic group within a study, only the data of the within-subjects comparison was selected since this usually provided the most conservative estimate.

Thought suppression can be considered as an elicitor of OCD symptoms such as intrusive thoughts as well. However, in the current meta-analysis these studies will not be included, since extensive meta-analyses already exist on this subject (e.g., Abramowitz, Tolin, & Street, 2001; Magee, Harden, & Teachman, 2012). When studies explored combined effects of thought suppression and another OCD symptom elicitor, only the data concerning the latter were considered.

Study Selection

The search resulted in a total of 1807 articles after duplicates removed. The meta-analysis was conducted in two steps: (1) First, all abstracts from the electronic databases search were screened for the potential for inclusion. Subsequently reviews (Atmaca, 2013; Chapman & Anderson, 2013; Haynes & Mallet, 2010; Kwon et al., 2009; Linden, 2006; Rachman, 2010; Rotge et al., 2009; Shafran & Rachman, 2004) and selected articles were screened for additional relevant studies. This resulted in the inclusion of a total of 190 articles. (2) Full copies of articles were read of the 190 articles. When an article met the selection criteria but did not report sufficient data to calculate the effect size, authors were requested to provide the additional data necessary for inclusion (k = 89, response rate = 43 percent). If these data were not retrieved and it was not possible to make an estimate of the missing data, the article was excluded (k = 36). This resulted in the final inclusion of 80 articles reporting 90 independent studies. See Figure 1 for an overview of the study selection.

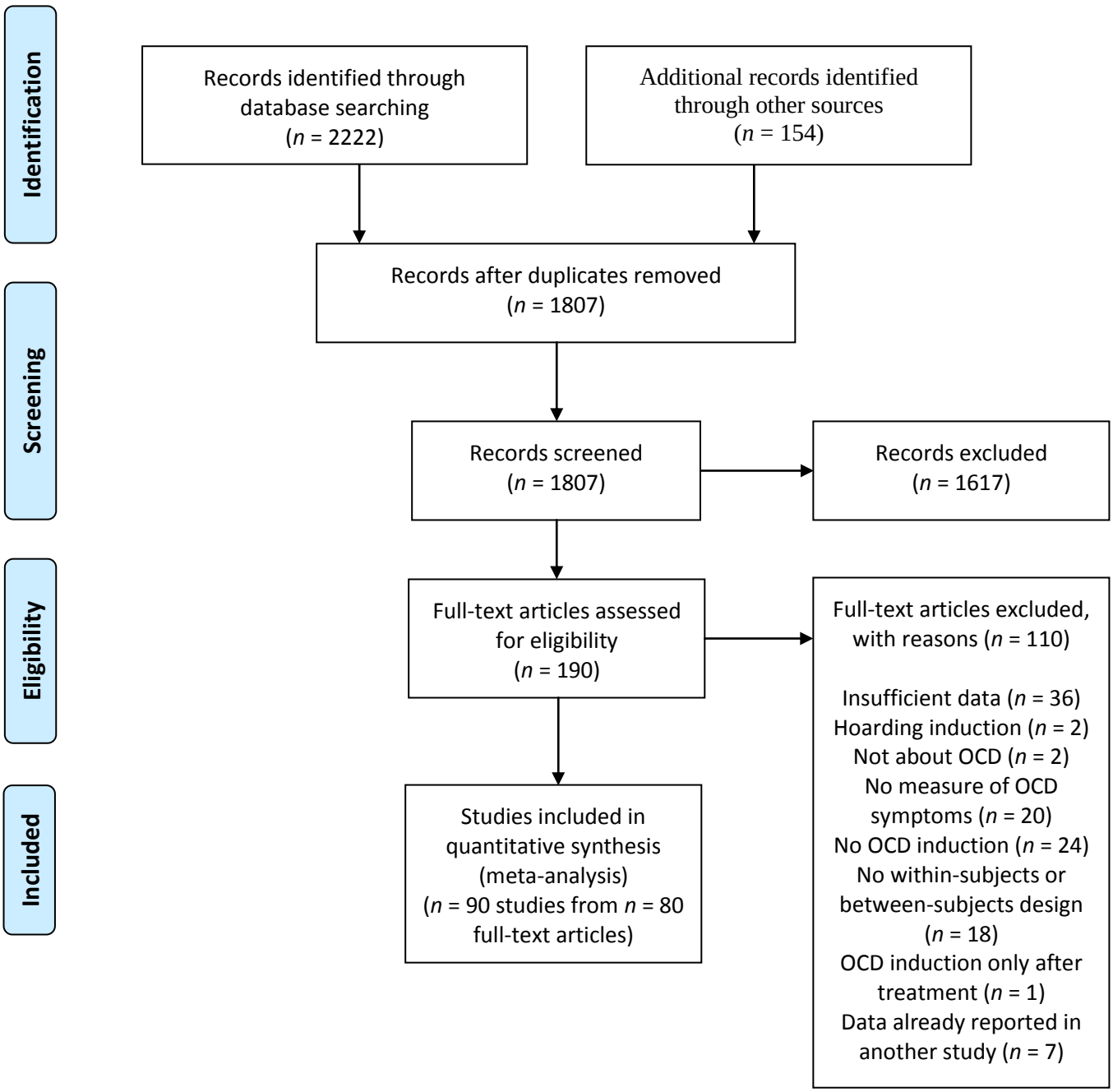

Figure 1. Prisma flow diagram of study selection.

Coding Procedure

Relevant information from each included study was coded by two independent coders using a predefined coding strategy. General information, characteristics of the induction, characteristics of the participants, characteristics of the comparison, and outcome were coded. General information that was coded consisted of the year of publication and the country in which the study was conducted. The characteristics of

the induction that were coded consisted of: (a) the category of induction (threat-related stimuli, disgust, mental contamination, perfectionism/certainty, responsibility, TAF, or performing compulsions), (b) which symptom dimension of OCD the induction targeted (in line with the symptom dimensions identified by Mataix-Cols et al. (2005) we used checking, contamination, symmetry[2], or general OCD regardless of symptom dimension), (c) whether the induction was idiosyncratic or standardized, and (d) the modality of the presentation of the induction. An induction was only considered idiosyncratic when stimuli were tailored to individuals in all groups in studies comparing diagnostic groups. The modalities considered are: (1) verbal: this includes inductions based on verbal instructions and stimuli presented auditory such as music; (2) visual: this includes inductions with pictures or movies; (3) objects: this includes induction through real-life exposure with objects; (4) behavior: this modality includes performing safety behaviors by means of an induction such as checking stoves or washing hands; (5) combination: this includes inductions using a combination of the aforementioned modalities.

Characteristics of the participants that were coded consisted of clinical status (healthy, subclinical or clinical) and sample size per group. The type of the comparison was also coded (within-subjects design and between-subjects design). Finally, outcome measurements were classified in five categories: (a) general OCD symptoms (e.g. the Padua Inventory), (b) compulsions, (c) compulsion urge, (d) obsessive thoughts, and (e) anxiety. Anxiety was operationalized as a broad category including anxiety, discomfort, and general distress. Inter rater agreement of the coding scheme was fair to excellent (mean κ = .71, range = .46-.87). An overview of studies included in the meta-analysis with their corresponding coding is presented in Appendix A.

Data Analysis

All analyses were performed with comprehensive meta-analysis software version 2.2.064 (CMA; Borenstein, Hedges, Higgins, & Rothstein, 2005) using a random

[2] The only two studies that focused on the symmetry symptom dimension used healthy participants. As healthy participants do not have symptoms corresponding to a symptom dimension, no symptom dimension analysis was carried out within healthy participants. Therefore symmetry could not be included in the symptom dimension analyses within (sub)clinical participants and comparison between (sub)clinical and healthy participants.

effects model. The random effects model is the most appropriate model for this meta-analysis since we can assume that there are actual differences between studies in effect size of different types of induction. This heterogeneity was tested by the *Q*-statistic with a *p*-value of .05 and the I^2-statistic. The *Q*–statistic is based on the ratio of observed variation to the within-study error and is dependent on the magnitude of the excess dispersion and the number of studies. This statistic can be used to test whether the heterogeneity is statistically significant. The I^2-statistic is independent of the number of studies and refers to the ratio of true variation between studies to total observed variation between studies (including random error) and reflects the magnitude of the heterogeneity (Borenstein, Hedges, Higgins, & Rothstein, 2009).

Hedges's *g* was chosen as an effect size estimate since it controls for variations in sample size between studies (Borenstein et al., 2009). Hedges's *g* is interpreted similarly to Cohen's *d* with *g* = 0.2-0.5 defined as a small effect size, *g* = 0.5-0.8 defined as a medium effect size, and *g* = 0.8 or greater defined as a large effect size (Cohen, 1988). Whenever possible, effect sizes for between-subjects designs were calculated with the sample sizes, means and standard deviations. For within-subjects designs and between-subjects designs with carefully matched groups effect sizes were calculated with the sample size, means, standard deviations and the exact correlation between time points or groups. If these measures were not available or not available upon request, Hedges's *g* was calculated with *t*-values and sample sizes, *p*-values of *t*-tests and sample size, or when applicable χ^2-value and sample size (as recommended by Borenstein et al., 2009).

Provided the potentially differential response to symptom provocation as a function of diagnostic status of the participants (healthy, subclinical or clinical status), we started by testing whether we could combine studies directly comparing clinical to healthy participants and studies directly comparing subclinical to healthy participants by adding comparison (subclinical versus healthy or clinical versus healthy) as a moderator. If this moderator would be significant, this would suggest that studies with subclinical participants instead of clinical participants differ substantially and should be analyzed separately. If this difference would not be significant, this would suggest that these studies can be grouped together in the same analyses. Second, we tested whether there was a significant difference in (sub)clinical participants versus healthy

participants by using studies that directly compare (sub)clinical participants to healthy participants. If these analyses would show that this effect is significant, this suggests that (sub)clinical participants react differently to inductions than healthy participants. This would mean that in the within-group analyses (sub)clinical participants would need to be analyzed separately from healthy participants. If this comparison is not significant, this would mean that healthy participants and (sub)clinical participants can be grouped together in the same within-group analyses. Finally, we continued with an examination of the key moderators (i.e., category of induction, symptom dimension of OCD, modality of presentation, and level of tailoring to individuals) between- and within-groups. We did not do a moderator analysis on symptom dimension of OCD within healthy participants, as these participants were healthy and did not have clinical symptoms corresponding to any symptom dimension.

In the general analyses, moderators were grouped in order to yield one effect size per independent study. Furthermore, all dependent measures were grouped in order to form a broad outcome of OCD symptoms. In order to be conservative, whenever the correlation between dependent measures that needed to be combined into a composite effect size for a study was unknown, we used the default settings for dependent measures in CMA which assumes the correlation between dependent measures is 1. When the correlation between dependent measures is 1, the standard error for the point estimate computed across outcomes will likely be overestimated. Therefore this composite effect size will be conservative with regard to Type I error rates (Borenstein et al., 2009). However since this correlation is very unlikely, we repeated the analyses with the more realistic correlation of .5 as a means of a sensitivity analysis. Furthermore, whenever possible we used sensitivity analyses to test whether the conclusions were robust for all separate outcome measures (anxiety, compulsion urge, compulsions, obsessions and symptoms measured in general). Finally, since the design of a study can have a significant impact on its effect size, we also checked whether conclusions were robust for the different designs of the studies (i.e., between-subjects or within-subjects comparison) by means of a sensitivity analysis.

Within diagnostic status, subgroup analyses were planned for category of induction, studies with idiosyncratic inductions versus studies with standardized inductions, modality of presentation and the symptom dimension of OCD targeted in

the induction. Since in some cases there were less than five studies available per subgroup, a random effects model was used to combine subgroups to yield an overall effect. If there are less than five studies within a subgroup, the estimation of τ^2 is likely to be imprecise. In this case the increased accuracy of pooling the estimate of τ^2 over more studies is likely to exceed any real differences between groups in the true value of τ^2 (Borenstein et al., 2009). With a random effects model τ^2 is computed within subgroups and subsequently pooled across subgroups. In order to be conservative, whenever a study provided information on more than one subgroup for a comparison of the differences between subgroups, the correlation between these dependent measures was set to 0. This results in a larger standard error of the difference and thus *p*-values for the difference between subgroups are likely to be conservative with regard to type I errors (Borenstein et al., 2009).

Finally, the presence and impact of publication bias was investigated by generating funnel plots and computing Duval and Tweedie's (2000) trim-and-fill procedure using a random effects model. The theory behind a funnel plot is that studies with smaller sample sizes are more prone to error and are at the greatest risk for being lost since small and moderate effects are unlikely to be published. In the presence of publication bias the funnel plot will be asymmetrical with studies unevenly presented above or below the mean. The trim-and-fill procedure estimates the number of missing studies that would correct publication bias and computes an effect size without publication bias. Publication bias was assessed for the main analyses.

Results

Analyses between Diagnostic Groups

In order to test whether the comparisons of subclinical versus healthy participants and clinical versus healthy participants need to be analyzed separately an analysis was conducted with comparison (subclinical versus healthy or clinical versus healthy) as a moderator. The difference between these comparisons was not significant ($Q(1) = 0.19$, $p = .666$), therefore studies directly comparing OCD participants to healthy control participants and studies directly comparing subclinical OCD participants to healthy control participants will be included in the same analyses. Note that the effect sizes that we report on the between diagnostic group comparisons do not represent the

magnitude of the effect of the induction for (sub)clinical OCD in itself, but the incremental effect of the induction for (sub)clinical OCD beyond a healthy control group.

General effect. In total there were 28 studies directly comparing (sub)clinical OCD to healthy control participants including 670 (sub)clinical and 697 healthy participants in total. These studies showed a large effect size for induction methods of OCD symptoms in (sub)clinical OCD beyond a healthy control group (g = 0.81, p < .001, 95% CI = [0.64; 0.97]). There was significant evidence of heterogeneity (Q(27) = 53.72, p = .002, I^2 = 49.74), however there were no outliers. Thus, as expected, (sub)clinical OCD participants show more OCD symptoms after an induction than healthy control participants.

Categories. There was no significant difference (Q(5) = 6.68, p = .246) between the disgust category (g = 0.92, p < .001, 95% CI = [0.63; 1.22]), perfectionism/certainty category (g = 0.43, p = .026, 95% CI = [0.05; 0.81]), the repeated compulsions category (g = 0.69, p = .029, 95% CI = [0.07; 1.3]), the responsibility category (g = 0.65, p = .064, 95% CI = [-0.04; 1.34]), the TAF category (g = 0.56, p = .064, 95% CI = [-0.03; 1.15]), and the threat category (g = 0.94, p < .001, 95% CI = [0.70; 1.18]) in the incremental effect of induction procedures for (sub)clinical OCD beyond a healthy control group. There was evidence for heterogeneity in the disgust category (Q(7) = 14.81, p = .038, I^2 = 52.74), repeated compulsions category (Q(1) = 6.16, p = .013, I^2 = 83.76), and the threat category (Q(11) = 19.77, p = .049, I^2 = 44.36), but not for the perfectionism/certainty category (Q(3) = 0.68, p = .877, I^2 = 0.00), the responsibility category (Q(1) = 1.17, p = .279, I^2 = 14.65), or the TAF category (Q(1) = 1.15, p = .284, I^2 = 12.87).

Idiosyncratic. There was no significant difference (Q(1) = 0.13, p = .716) between studies using idiosyncratic material (g = 0.68, p = .059, 95% CI = [-0.03; 1.39]) and studies using standardized material (g = 0.82, p < .001, 95% CI = [0.65; 0.99]) in the incremental effect of induction procedures for (sub)clinical OCD beyond a healthy control group. There was evidence of heterogeneity for studies using standardized material (Q(25) = 53.42, p = .001, I^2 = 53.20), but not for studies using idiosyncratic inductions (Q(1) = 0.17, p = .677, I^2 = 0.00).

Symptom dimension of OCD. There was a significant difference (Q(2) = 8.00, p = .018) in studies comparing (sub)clinical OCD to healthy controls in the inductions

targeting the contamination dimension (g = 1.03, p < .001, 95% CI = [0.81; 1.24]), the checking dimension (g = 0.58, p < .001, 95% CI = [0.34; 0.82]), and general OCD (g = 0.70, p < .001, 95% CI = [0.37; 1.04]) in the incremental effect of induction procedures for (sub)clinical OCD beyond a healthy control group. Furthermore, there was evidence of heterogeneity for the contamination dimension (Q(13) = 28.19, p = .009, I^2 = 53.89), but not for general OCD (Q(4) = 5.07, p = .280, I^2 = 21.15) or the checking dimension (Q(9) = 7.84, p = .551, I^2 = 0.00).

Modality of presentation. There was no significant difference (Q(3) = 1.97, p = .578) in the efficacy of inductions using objects (g = 0.94, p < .001, 95% CI = [0.66; 1.23]), inductions using behavior (g = 0.67, p = .040, 95% CI = [0.03; 1.32]), verbal inductions (g = 0.62, p = .002, 95% CI = [0.23; 1.01]), and visual inductions (g = 0.77, p < .001, 95% CI = [0.55; 0.99]) in the incremental effect of induction procedures for (sub)clinical OCD beyond a healthy control group. Furthermore, there was evidence of heterogeneity for inductions using objects (Q(8) = 21.48, p = .006, I^2 = 62.75), inductions using behavior (Q(1) = 6.16, p = .013, I^2 = 83.76), and visual inductions (Q(14) = 25.76, p = .028, I^2 = 45.66), but not for verbal inductions (Q(5) = 2.59, p = .764, I^2 = 0.00).

Analyses within Diagnostic Groups

Since analyses between diagnostic groups indicated a significant large effect size when comparing healthy participants to (sub)clinical participants, there is evidence that (sub)clinical OCD participants respond differentially to induction procedures than healthy participants. However, analyses showed no significant difference between the comparison clinical OCD vs. healthy controls and the comparison subclinical OCD vs. healthy controls. Furthermore, there were only four studies available for subclinical participants, which would render analysis of the key moderators within subclinical participants underpowered. Therefore studies with subclinical and clinical OCD participants are combined and analyzed separately from healthy participants.

Induction effects within (sub)clinical OCD. In total there were 24 studies using (sub)clinical OCD participants with a total of 485 participants. Within (sub)clinical OCD only two studies exceeded g = 3 (Baioui et al., 2013; Chen, Xie, Han, Cui, & Zhang, 2004) and were therefore identified as outliers. These studies were excluded from further analyses. Overall induction methods for (sub)clinical OCD had a large effect size (g =

0.95, $p < .001$, 95% CI = [0.71; 1.20]). This effect was characterized by evidence for heterogeneity ($Q(21) = 119.81$, $p < .001$, $I^2 = 82.47$).

***Categories*.** Studies examining the (sub)clinical OCD subgroup only provided sufficient data for a comparison between the disgust, repeated compulsions, responsibility, and threat category. The difference between the estimated effect size of the threat category ($g = 1.24$, $p < .001$, 95% CI = [0.90; 1.59]), the responsibility category ($g = 0.81$, $p = .003$, 95% CI = [0.28; 1.34]), the disgust category ($g = 0.47$, $p = .174$, 95% CI = [-0.21; 1.15]), and the repeated compulsions category ($g = 0.21$, $p = .585$, 95% CI = [-0.55; 0.97]) was significant ($Q(3) = 8{,}56$, $p = .036$). In contrast to the threat category ($Q(12) = 93.04$, $p < .001$, $I^2 = 87.10$) and the responsibility category ($Q(4) = 14.55$, $p = .006$, $I^2 = 72.50$), there was no significant evidence for heterogeneity for the disgust category ($Q(2) = 1.36$, $p = .507$, $I^2 = 0.00$) or the repeated compulsions category ($Q(1) = 1.36$, $p = .243$, $I^2 = 26.61$).

Idiosyncratic. The difference between the estimated effect size of studies using idiosyncratic material and studies using standardized material was significant ($Q(1) = 5.11$, $p = .024$), with a higher effect size for studies using idiosyncratic material ($g = 1.39$, $p < .001$, 95% CI = [0.96; 1.82]) than studies using standardized material ($g = 0.81$, $p < .001$, 95% CI = [0.53; 1.08]). There was evidence for heterogeneity for both studies using idiosyncratic inductions ($Q(7) = 29.46$, $p < .001$, $I^2 = 76.24$) and studies using standardized material ($Q(15) = 84.01$, $p < .001$, $I^2 = 82.14$).

Symptom dimension *of OCD*. Inductions targeting the contamination dimension ($g = 0.71$, $p < .001$, 95% CI = [0.35; 1.07]), the checking dimension ($g = 1.11$, $p < .001$, 95% CI = [0.66; 1.55]), and inductions designed for general OCD ($g = 1.04$, $p < .001$, 95% CI = [0.69; 1.38]) were equally efficacious ($Q(2) = 2.35$, $p = .309$). Furthermore, heterogeneity was evident in all symptom dimensions of OCD (contamination: $Q(7) = 33.13$, $p < .001$, $I^2 = 78.87$; checking: $Q(4) = 26.39$, $p < .001$, $I^2 = 84.84$; general OCD: $Q(8) = 20.62$, $p = .008$, $I^2 = 61.20$).

***Modality of presentation*.** There was a significant difference ($Q(3) = 10.15$, $p = .017$) in the efficacy of visual inductions ($g = 1.41$, $p < .001$, 95% CI = [0.95; 1.86]), verbal inductions ($g = 0.99$, $p < .001$, 95% CI = [0.63; 1.35]), inductions using objects ($g = 0.85$, $p < .001$, 95% CI = [0.49; 1.21]), and inductions using behavior ($g = 0.21$, $p = .482$, 95% CI = [-0.38; 0.80]). Furthermore, heterogeneity was evident in visual inductions ($Q(4) =$

16.06, p = .003, I^2 = 75.09) and inductions with objects (Q(7) = 38.72, p < .001, I^2 = 81.92), but not in verbal inductions (Q(7) = 12.08, p = .098, I^2 = 42.04) or inductions using behavior (Q(1) = 1.36, p = .243, I^2 = 26.61).

Induction effects within healthy participants. In total there were 58 studies including 3449 healthy participants. Only two studies exceeded g = 2 (Baioui et al., 2013; Mataix-Cols et al., 2008) and were subsequently removed as outliers. Overall OCD induction methods for healthy participants are characterized by a medium effect size (g = 0.58, p < .001, 95% CI = [0.47; 0.69]). This effect was characterized by evidence for heterogeneity (Q(55) = 287.12, p < .001, I^2 = 80.84).

***Categories*.** Three studies were outliers based on their effect size within their corresponding categories and were excluded from this subgroup analysis (Bocci & Gordon, 2007; Dorfan & Woody, 2011; Suda et al., 2014). There was a significant difference (Q(6) = 32.99, p < .001) between the mental contamination category (g = 0.80, p < .001, 95% CI = [0.60; 1.00]), the TAF category (g = 0.78, p < .001, 95% CI = [0.57; 0.99]), the threat category (g = 0.50, p < .001, 95% CI = [0.25; 0.74), the perfectionism/certainty category (g = 0.48, p < .001, 95% CI = [0.26; 0.70]), the repeated compulsions category (g = 0.42, p < .001, 95% CI = [0.22; 0.62]), the responsibility category (g = 0.41, p = .019, 95% CI = [0.07; 0.75]), and the disgust category (g = 0.15, p = .094, 95% CI = [-0.03; 0.32]). Furthermore, heterogeneity was evident in the threat category (Q(4) = 29.95, p < .001, I^2 = 86.64), and the repeated compulsions category (Q(5) = 18.33, p = .003, I^2 = 72.72), but not in the mental contamination category (Q(15) = 21.46, p = .123, I^2 = 30.11), the disgust category (Q(8) = 13.12, p = .108, I^2 = 39.04), the perfectionism/certainty category (Q(5) = 7.51, p = .185, I^2 = 33.45), the TAF category (Q(7) = 8.32, p = .306, I^2 = 15.83), or the responsibility category (Q(4) = 1.12, p = .890, I^2 = 0.00).

Idiosyncratic. Although the estimated effect size of studies using idiosyncratic material is higher (g = 0.87, p < .001, 95% CI = [0.43; 1.31]) than studies using standardized material (g = 0.56, p < .001, 95% CI = [0.45; 0.68]), this difference did not reach significance (Q(1) = 1.77, p = .183). Contrary to studies using standardized material (Q(50) = 276.62, p < .001, I^2 = 81.92), there was no significant evidence for heterogeneity for studies using idiosyncratic inductions (Q(4) = 0.30, p = .99, I^2 = 0.00).

***Modality of presentation*.** Studies on healthy participants used behavior, objects, verbal, visual and a combination of modalities for inductions. There was no significant difference ($Q(4) = 5.68$, $p = .225$) in the efficacy of inductions using behavior ($g = 0.41$, $p = .002$, 95% CI = [0.15; 0.68]), verbal inductions ($g = 0.67$, $p < .001$, 95% CI = [0.52; 0.82]), visual inductions ($g = 0.66$, $p < .001$, 95% CI = [0.40; 0.92]), inductions using objects ($g = 0.42$, $p = .006$, 95% CI = [0.12; 0.72]), and studies using a combination of modalities ($g = 0.38$, $p = .020$, 95% CI = [0.06; 0.70]). Furthermore, heterogeneity was evident in all modalities (behavior: $Q(6) = 18.43$, $p = .005$, $I^2 = 67.44$; objects: $Q(4) = 87.90$, $p < .001$, $I^2 = 95.45$; verbal: $Q(32) = 119.48$, $p < .001$, $I^2 = 73.22$; visual: $Q(8) = 35.44$, $p < .001$, $I^2 = 77.42$; combination of modalities: $Q(5) = 11.39$, $p = .044$, $I^2 = 56.11$).

Publication Bias

Analyses between diagnostic groups. For studies directly comparing (sub)clinical OCD to healthy controls the trim-and-fill procedure estimated one study with an effect size higher than the mean, $g = 0.84$, 95% CI = [0.67; 1.00], $Q = 60.27$, but exclusion of this study did not significantly change the results. In line with this result, the funnel plot showed some asymmetry suggesting the presence of missing studies with effect sizes above the mean and the possibility of obtaining a slightly under-inflated estimate of the true differences between (sub)clinical OCD and healthy controls.

Analysis within diagnostic groups. For studies using induction procedures within (sub)clinical OCD the trim-and-fill procedure estimated 7 studies with an effect size lower than the mean, $g = 0.68$, 95% CI = [0.43; 0.92], $Q = 184.17$. Moreover, exclusion of these studies resulted in a significantly larger effect size. In line with this result, the funnel plot showed asymmetry suggesting the presence of missing studies with effect sizes under the mean and the possibility of obtaining an over-inflated estimate of the true effect size.

For studies using induction procedures within healthy participants the trim-and-fill procedure estimated 12 studies with an effect size lower than the mean, $g = 0.45$, 95% CI = [0.34; 0.57], $Q = 374.26$. Exclusion of these studies resulted in a significantly larger effect size. In line with this result, the funnel plot showed asymmetry suggesting the presence of missing studies with effect sizes under the mean and the possibility of

obtaining an over-inflated estimate of the true effect size. Funnel plots are presented in Appendix B.

Sensitivity Analysis

Since the default correlation of CMA between dependent measures of 1 is very unlikely, we repeated the analyses with the more realistic correlation of .5 as a means of a sensitivity analysis. The specific effect sizes and their corresponding heterogeneity were only slightly different and the only conclusion that changed was the difference between categories within (sub)clinical participants, which changed from significant to marginally significant ($Q(3) = 7.09$, $p = .069$).

Furthermore, whenever possible we tested whether the conclusions were robust for all separate outcome measures (anxiety, compulsion urge, compulsions, obsessions and symptoms measured in general). Most conclusions were robust. In studies comparing (sub)clinical OCD to healthy control participants the difference between inductions targeting the checking dimension, the contamination dimension, and general OCD was no longer significant for the anxiety outcome ($Q(2) = 1.86$, $p = .395$). Likewise, this effect was no longer significant for the combined other outcomes (i.e., excluding anxiety) ($Q(2) = 5.18$, $p = .075$). Therefore, this non-significant effect is likely due to a lack of power. Moreover, within (sub)clinical participants the difference between categories was no longer significant for every outcome separately. This effect is due to the absence of the repeated compulsions category. Studies from the repeated compulsions category use compulsions (Boschen & Vuksanovic, 2007) or symptoms (Deacon & Maack, 2008) as outcome measures. Without the repeated compulsions category the difference between these categories was no longer significant ($Q(2) = 4.27$, $p = .118$). Similarly, within (sub)clinical participants the difference between modalities was no longer significant for every outcome separately. This non-significant effect is also driven by the absence of the behavior modality, which consisted of the same studies as the repeated compulsions category. Without the behavior modality the difference between modalities was no longer significant ($Q(2) = 2.98$, $p = .225$). Also within (sub)clinical participants the difference between idiosyncratic and standardized inductions was no longer significant for the symptom outcome ($Q(1) = 3.12$, $p = .078$) and the anxiety outcome ($Q(1) = 3.42$, $p = .065$). However, this non-significant effect is

likely due to lack of power since the effect was significant when combining the symptom outcome and the anxiety outcome ($Q(1) = 5.45$, $p = .020$).

Furthermore, within healthy participants for the compulsion urge outcome the comparison between the following categories was no longer significant ($Q(2) = 4.67$, $p = .097$): the mental contamination category, the perfectionism/certainty category, and the responsibility category. These effects could due to lack of power since these analyses are based on fewer studies ($k = 21$ instead of $k = 27$ for these categories). Finally, we checked whether conclusions were robust for the different designs of the studies (between-subjects or within-subjects). All conclusions proved to be robust.

Discussion

This meta-analysis set out to examine the efficacy of induction procedures in healthy and (sub)clinical participants both within and across diagnostic status. Efficacious inductions of obsessive-compulsive symptoms are a cornerstone of experimental studies investigating the nature of and processes involved in OCD, which are considered crucial in the development of theoretical models (Abramovitch & Cooperman, 2015). Based on the current available research literature, we examined whether the efficacy of induction procedures varied across the different induction categories, symptom dimensions of OCD, modalities of presentation, and tailoring to individual fears. Here, we present the main results, the quality of the current obtained evidence, and their implications.

Main Findings

First, we discuss the main results at the level of the comparisons across diagnostic status. The difference between studies directly comparing clinical OCD vs. healthy participants and studies directly comparing subclinical OCD vs. healthy participants was not significant. Therefore these studies were included in the same analyses. Analysis of 28 studies directly comparing (sub)clinical OCD to healthy control participants showed a large incremental effect of induction procedures in OCD relative to healthy controls. Yet, it is noteworthy that substantial heterogeneity was observed across studies. This effect did not vary significantly over categories, modalities of presentation, or level of idiosyncratic stimulus selection. However, there was a

significant difference in the magnitude of the effect between symptom dimensions of OCD targeted. That is, induction procedures presented to individuals of the contamination dimension showed a large effect size whereas inductions presented to general OCD (regardless of symptom dimension) and the checking dimension showed medium effect sizes. This result indicates that, based on the current literature, the difference in the magnitude of the effect of inductions in (sub)clinical OCD relative to healthy participants is most evident in the contamination dimension and smaller in general OCD and the checking dimension.

Second, several interesting findings emerged from the within-group analyses. Since analyses between diagnostic groups indicated a significant large effect size when comparing healthy participants to (sub)clinical participants, there is evidence that (sub)clinical participants respond differently to induction procedures than healthy participants. Therefore, within-group analyses were conducted for healthy participants and (sub)clinical participants separately. Based on 22 studies with (sub)clinical OCD, our meta-analysis showed a large effect size of induction procedures. However, publication bias analysis showed that this might be an over-inflated estimate and there was substantial heterogeneity across studies. There were no significant moderation effects by symptom dimension of OCD. Importantly though, the magnitude of induction varied across categories with large effects for the threat category and the responsibility category and small effects for the disgust and repeated compulsions category. Sensitivity analysis showed that the significant difference between categories was driven by the small effect of the repeated compulsions category. A second significant moderator was modality of presentation, with large effects for visual inductions, verbal inductions and inductions using objects and a small effect for inductions solely based on behavior. A third significant moderator was the level of individual tailoring: the effect was significantly stronger for studies that tailored the induction procedure to participants than for studies using standardized material.

Within healthy participants an analysis on 56 studies showed a medium effect size of inducing OCD symptoms. However, publication bias analysis showed that this might be an over-inflated estimate and there was substantial heterogeneity across studies. This effect did not vary across level of individual tailoring or modality of presentation. Importantly though, this effect varied across categories with the

strongest effect for mental contamination, followed by TAF inductions, threat inductions, small effect sizes for perfectionism/certainty, repeated compulsions, and responsibility inductions, and a very small and not significant effect size for disgust inductions.

Practical Recommendations for Inducing OCD Symptoms in the Lab

These results have some interesting implications for research. First, the finding that results of induction procedures in subclinical participants did not significantly differ from clinical participants is convenient for research, since subclinical participants are more easily recruited. The finding that subclinical participants did not significantly differ from clinical participants confirms the utility of analogue samples and is in line with the review of Abramowitz et al. (2014).

Studies investigating the effect of OCD symptoms within (sub)clinical OCD will benefit most from inductions from the threat and responsibility category and least from the disgust and repeated compulsions category. The strongest effects were obtained when stimuli were tailored to individuals. Examples of efficacious procedures within the threat category that are idiosyncratic include asking patients to take pictures of their triggers themselves (e.g., Morgiève et al., 2014) and exposing participants to their triggers (e.g., to gauze with a personal feared substance; Simpson, Tenke, Towey, Liebowitz, & Bruder, 2000). An example of a potent OCD symptom induction in the responsibility category is asking the participant to sign a contract that he/she is fully responsible for any consequences (e.g., Lopatka & Rachman, 1995; Radomsky et al., 2001).

Studies that need to provoke symptoms within healthy participants will benefit most from induction procedures within the mental contamination, TAF, or threat category, although other categories can also be efficacious. This finding is convenient for research on healthy participants, since TAF and mental contamination procedures often have standardized protocols and are easily implemented. The only category that was not significantly efficacious within healthy participants was disgust. A typical example of an efficacious OCD symptom induction procedure within the mental contamination category is the non-consensual kiss task (e.g., Elliott & Radomsky, 2009, 2012; Fairbrother et al., 2005; Herba & Rachman, 2007). A possible OCD symptom

induction within the threat category is reading a number of intrusive thoughts common for OCD and imagining having these thoughts (e.g., Davey, Meeten, Barnes, & Dash, 2013).

Another interesting finding is that both in the analysis within (sub)clinical participants and healthy participants the effects of threat inductions were large, while the effect of repeated compulsions and disgust were small. This finding suggests that although repeated compulsions (Deacon & Maack, 2008) and disgust can have an influence on OCD (Olatunji et al., 2010), they may not be sufficient in order to induce OCD symptoms.

Quality and Quantity of the Available Research

Based on the current meta-analysis, several important observations can be made with regard to this field of research. First, our meta-analysis set out to explore the efficacy of different induction procedures. As stated before, ethical concerns are highly important here and the aim of a powerful induction of OCD symptoms needs to be balanced against ethical considerations of tolerability and harm. To date, few studies include an explicit measure of tolerability. For instance, it is possible to use a VAS scale on which participants can assess how tolerable the induction is for them. Future studies should include such a measure so that the balance between tolerability and efficacy for different categories can be investigated in future meta-analyses.

Second, the large number of studies (k = 56) using induction procedures in healthy participants relative to the number of studies (k = 22) using induction procedures in (sub)clinical OCD is remarkable and qualifies the current conclusions, since we were only able to investigate a limited amount of moderators in (sub)clinical OCD. To date there were no studies available that used inductions within (sub)clinical OCD for the mental contamination category, TAF category, and perfectionism/certainty category. Thus, the categories that were most efficacious within healthy participants (mental contamination and TAF) have not been investigated as a means of an induction within (sub)clinical OCD. Based on the current data it is still unclear whether these categories generate a similar strong effect in (sub)clinical OCD or whether this effect is specific for healthy participants. Although induction procedures need to be administered in an ethically responsible and careful manner in OCD populations, it

would be interesting to see whether these categories also prove to be most efficacious within (sub)clinical OCD. Thus, more studies investigating induction procedures in (sub)clinical OCD are necessary in order to make a comparison between all categories of induction.

A final key limitation of the current meta-analysis is that in the main analyses we were not able to conduct more fine-grained analyses on different relevant outcomes, because a lack of standardization across studies. For instance, distress, anxiety, discomfort, obsessions, compulsion urge, compulsions and general symptoms can be closely related, but it has been argued that they are distinct phenomena (e.g., Rachman, 2004a). Moreover there were differences in how these dependent variables in the studies were measured. In order to allow a more fine-grained comparison between studies it would be recommended that novel studies using induction procedures use a more standardized set of outcome measures to capture changes in obsessive and compulsive symptoms. Relatedly, administering measures of baseline levels of anxiety and OCD symptoms before induction procedures is crucial in order to be able to gauge the magnitude of induced symptoms. This is important for any conclusions within studies as well as across studies.

Limitations of the Current Conclusions

This meta-analysis has several limitations. First, although categories of induction procedures were based upon theoretically and empirically distinct categories of OCD factors, induction procedures within categories could still vary substantially. For instance, within the responsibility category, induction procedures ranged from signing a contract that the patient was fully responsible for any consequences (e.g., Radomsky et al., 2001) to classifying capsules in different colors in order to develop a system that makes the distribution of medication safer (e.g., Arntz et al., 2007). This may have limited the validity of conclusions based upon these categories and introduced heterogeneity. More research with standardized induction procedures in these categories is necessary to further decrease heterogeneity and increase the validity of the current results. For instance inductions within the TAF and the mental contamination category have standardized protocols and proved to be highly efficacious. Standardization of induction methods does not exclude the possibility of

tailoring the stimuli to individuals. For instance the procedure of tailoring stimuli to individuals (e.g., take pictures of their triggers; Schienle et al., 2005) could be standardized which will already decrease heterogeneity.

Second, another source of heterogeneity was the difference in designs between studies. Although it is still possible to perform a meta-analysis on studies with different designs, differences between study designs can influence the validity of conclusions (Borenstein et al., 2009). However, our sensitivity analyses showed no differences in the conclusions of the meta-analysis when only within-subjects or only between-subjects designs were selected and it is thus unlikely that this variety affected our conclusions.

Finally, one could comment on the fact that the dependent variables included in the meta-analysis were quite varied which could have added noise in our analyses. However, a review on anxiety measures has shown that many anxiety measures based on self-report correlate substantially (Rossi & Pourtois, 2012). Furthermore, sensitivity analyses were performed in order to check whether the conclusions were robust. Hence it is unlikely that this variety in outcome measures had a significant impact on our conclusions. Unfortunately, there were only limited studies using psychophysiological measures, which prevented including these anxiety measures in our meta-analysis. Here it would be interesting for future research to include more psychophysiological measures.

Conclusion

In this meta-analysis we examined the efficacy of different inductions in the elicitation of OCD symptoms within and across diagnostic groups. In general within every diagnostic group effect sizes of induction procedures were significant, confirming the general capacity of induction procedures to induce OCD symptoms. Furthermore, there was no difference in studies using subclinical and clinical participants, confirming the utility of analogue samples. However, the effect size of induction procedures between (sub)clinical and healthy groups was significant, suggesting that induction procedures are more efficacious in (sub)clinical OCD than in healthy participants. This difference was most evident in the contamination dimension. Within studies of (sub)clinical participants inductions for the threat and responsibility category were most efficacious, especially when stimuli were tailored to the participants. Within studies of

healthy participants inductions were most efficacious for the mental contamination, TAF, and threat category and least efficacious for the disgust category.

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References

Abramovitch, A., & Cooperman, A. (2015). The cognitive neuropsychology of obsessive-compulsive disorder: A critical review. *Journal of Obsessive-Compulsive and Related Disorders, 5*, 24-36. doi: 10.1016/j.jocrd.2015.01.002

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

Abramowitz, J. S., Tolin, D. F., & Street, G. P. (2001). Paradoxical effects of thought suppression: A meta-analysis of controlled studies. *Clinical Psychology Review, 21*(5), 683-703. doi: 10.1016/S0272-7358(00)00057-X

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Alcolado, G. M., & Radomsky, A. S. (2011). Believe in yourself: Manipulating beliefs about memory causes checking. *Behaviour Research and Therapy, 49*(1), 42-49. doi: 10.1016/j.brat.2010.10.001

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Arntz, A., Voncken, M., & Goosen, A. C. A. (2007). Responsibility and obsessive–compulsive disorder: An experimental test. *Behaviour Research and Therapy, 45*(3), 425-435. doi: 10.1016/j.brat.2006.03.016

Atmaca, M. (2013). The effects of psychopharmacologic and therapeutic approaches on neuro-imaging in obsessive-compulsive disorder. *Current Neuropharmacology, 11*(1), 109-113. doi: 10.2174/157015913804999414

Baioui, A., Pilgramm, J., Merz, C. J., Walter, B., Vaitl, D., & Stark, R. (2013). Neural response in obsessive-compulsive washers depends on individual fit of triggers. *Frontiers in Human Neuroscience, 7*(143), 1-13. doi: 10.3389/fnhum.2013.00143

Berman, N. C., Abramowitz, J. S., Pardue, C. M., & Wheaton, M. G. (2010). The relationship between religion and thought action fusion: Use of an in vivo

paradigm. *Behaviour Research and Therapy, 48*(7), 670-674. doi: 10.1016/j.brat.2010.03.021

Bocci, L., & Gordon, P. K. (2007). Does magical thinking produce neutralising behaviour? An experimental investigation. *Behaviour Research and Therapy, 45*(8), 1823-1833. doi: 10.1016/j.brat.2007.02.003

Borenstein, M., Hedges, L., Higgins, J., & Rothstein, H. (2005). *Comprehensive meta-analysis version 2*. Englewood, NJ: Biostat, 104.

Borenstein, M., Hedges, L. V., Higgins, J. P. T., & Rothstein, H. R. (2009). *Introduction to meta-analysis*. Chichester, England: John Wiley & Sons. doi: 10.1002/9780470743386

Boschen, M. J., & Vuksanovic, D. (2007). Deteriorating memory confidence, responsibility perceptions and repeated checking: Comparisons in OCD and control samples. *Behaviour Research and Therapy, 45*(9), 2098-2109. doi: 10.1016/j.brat.2007.03.009

Breiter, H. C., Rauch, S. L., Kwong, K. K., Baker, J. R., Weisskoff, R. M., Kennedy, D. N., . . . Rosen, B. R. (1996). Functional magnetic resonance imaging of symptom provocation in obsessive-compulsive disorder. *Archives of General Psychiatry, 53*(7), 595-606. doi: 10.1001/archpsyc.1996.01830070041008

Chapman, H. A., & Anderson, A. K. (2013). Things rank and gross in nature: A review and synthesis of moral disgust. *Psychological Bulletin, 139*(2), 300-327. doi: 10.1037/a0030964

Chen, X. L., Xie, J. X., Han, H. B., Cui, Y. H., & Zhang, B. Q. (2004). MR perfusion-weighted imaging and quantitative analysis of cerebral hemodynamics with symptom provocation in unmedicated patients with obsessive-compulsive disorder. *Neuroscience Letters, 370*, 206-211. doi: 10.1016/j.neulet.2004.08.019

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2 ed.). Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Coles, M. E., Frost, R. O., Heimberg, R. G., & Rheaume, J. (2003). "Not just right experiences": Perfectionism, obsessive-compulsive features and general psychopathology. *Behaviour Research and Therapy, 41*(6), 681-700. doi: 10.1016/S0005-7967(02)00044-X

Cougle, J. R., Fitch, K. E., Jacobson, S., & Lee, H. J. (2013). A multi-method examination of the role of incompleteness in compulsive checking. *Journal of Anxiety Disorders, 27*(2), 231-239. doi: 10.1016/j.janxdis.2013.02.003

Davey, G. C. L., Meeten, F., Barnes, G., & Dash, S. R. (2013). Aversive intrusive thoughts as contributors to inflated responsibility, intolerance of uncertainty, and thought-action fusion. *Clinical Neuropsychiatry, 10*(3), 30-44.

Deacon, B., & Maack, D. J. (2008). The effects of safety behaviors on the fear of contamination: An experimental investigation. *Behaviour Research and Therapy, 46*(4), 537-547. doi: 10.1016/j.brat.2008.01.010

Dorfan, N. M., & Woody, S. R. (2011). Danger appraisals as prospective predictors of disgust and avoidance of contaminants. *Journal of Social and Clinical Psychology, 30*(2), 105-132.

Duval, S., & Tweedie, R. (2000). Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56*(2), 455-463. doi: 10.1111/j.0006-341X.2000.00455.x

Elliott, C. M., & Radomsky, A. S. (2009). Analyses of mental contamination: Part I, experimental manipulations of morality. *Behaviour Research and Therapy, 47*(12), 995-1003. doi: 10.1016/j.brat.2009.03.004

Elliott, C. M., & Radomsky, A. S. (2012). Mental contamination: The effects of imagined physical dirt and immoral behaviour. *Behaviour Research and Therapy, 50*(6), 422-427. doi: 10.1016/j.brat.2012.03.007

Fairbrother, N., Newth, S. J., & Rachman, S. (2005). Mental pollution: Feelings of dirtiness without physical contact. *Behaviour Research and Therapy, 43*(1), 121-130. doi: 10.1016/j.brat.2003.12.005

Fisher, P. L., & Wells, A. (2005). How effective are cognitive and behavioral treatments for obsessive-compulsive disorder? A clinical significance analysis. *Behaviour Research and Therapy, 43*(12), 1543-1558. doi: 10.1016/j.brat.2004.11.007

Gentes, E. L., & Ruscio, A. M. (2011). A meta-analysis of the relation of intolerance of uncertainty to symptoms of generalized anxiety disorder, major depressive disorder, and obsessive-compulsive disorder. *Clinical Psychology Review, 31*(6), 923-933. doi: 10.1016/j.cpr.2011.05.001

Haynes, W. I. A., & Mallet, L. (2010). High-frequency stimulation of deep brain structures in obsessive-compulsive disorder: The search for a valid circuit. *European Journal of Neuroscience, 32*(7), 1118-1127. doi: 10.1111/j.1460-9568.2010.07418.x

Herba, J. K., & Rachman, S. (2007). Vulnerability to mental contamination. *Behaviour Research and Therapy, 45*(11), 2804-2812. doi: 10.1016/j.brat.2007.07.010

Hermans, D., Engelen, U., Grouwels, L., Joos, E., Lemmens, J., & Pieters, G. (2008). Cognitive confidence in obsessive-compulsive disorder: Distrusting perception, attention and memory. *Behaviour Research and Therapy, 46*(1), 98-113. doi: 10.1016/j.brat.2007.11.001

Hermans, D., Martens, K., De Cort, K., Pieters, G., & Eelen, P. (2003). Reality monitoring and metacognitive beliefs related to cognitive confidence in obsessive-compulsive disorder. *Behaviour Research and Therapy, 41*(4), 383-401. doi: 10.1016/S0005-7967(02)00015-3

Kwon, J. S., Jang, J. H., Choi, J. S., & Kang, D. H. (2009). Neuroimaging in obsessive-compulsive disorder. *Expert Review of Neurotherapeutics, 9*(2), 255-269. doi: 10.1586/14737175.9.2.255

Lees, A., Mogg, K., & Bradley, B. P. (2005). Health anxiety, anxiety sensitivity, and attentional biases for pictorial and linguistic health-threat cues. *Cognition and Emotion, 19*(3), 453-462. doi: 10.1080/02699930441000184

Linden, D. E. J. (2006). How psychotherapy changes the brain - the contribution of functional neuroimaging. *Molecular Psychiatry, 11*(6), 528-538. doi: 10.1038/sj.mp.4001816

Lopatka, C., & Rachman, S. (1995). Perceived responsibility and compulsive checking: An experimental analysis. *Behaviour Research and Therapy, 33*(6), 673-684. doi: 10.1016/0005-7967(94)00089-3

Magee, J. C., Harden, K. P., & Teachman, B. A. (2012). Psychopathology and thought suppression: A quantitative review. *Clinical Psychology Review, 32*(3), 189-201. doi: 10.1016/j.cpr.2012.01.001

Mataix-Cols, D., An, S. K., Lawrence, N. S., Caseras, X., Speckens, A., Giampietro, V., . . . Phillips, M. L. (2008). Individual differences in disgust sensitivity modulate neural

responses to aversive/disgusting stimuli. *European Journal of Neuroscience, 27*(11), 3050-3058. doi: 10.1111/j.1460-9568.2008.06311.x

Mataix-Cols, D., do Rosario-Campos, M. C., & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *American Journal of Psychiatry, 162*(2), 228-238. doi: 10.1176/appi.ajp.162.2.228

Mataix-Cols, D., Lawrence, N. S., Wooderson, S., Speckens, A., & Phillips, M. L. (2009). The Maudsley obsessive-compulsive stimuli set: Validation of a standardized paradigm for symptom-specific provocation in obsessive-compulsive disorder. *Psychiatry Research, 168*(3), 238-241. doi: 10.1016/j.psychres.2008.05.007

McGuire, P. K., Bench, C. J., Frith, C. D., Marks, I. M., Frackowiak, R. S. J., & Dolan, R. J. (1994). Functional anatomy of obsessive-compulsive phenomena. *British Journal of Psychiatry, 164*(4), 459-468. doi: 10.1192/bjp.164.4.459

McKay, D., Sookman, D., Neziroglu, F., Wilhelm, S., Stein, D. J., Kyrios, M., . . . Veale, D. (2015). Efficacy of cognitive-behavioral therapy for obsessive-compulsive disorder. *Psychiatry Research, 227*(1), 104-113. doi: 10.1016/j.psychres.2015.02.004

Morgiève, M., N'Diaye, K., Haynes, W. I., Granger, B., Clair, A. H., Pelissolo, A., & Mallet, L. (2014). Dynamics of psychotherapy-related cerebral haemodynamic changes in obsessive compulsive disorder using a personalized exposure task in functional magnetic resonance imaging. *Psychological Medicine, 44*(7), 1461-1473. doi: 10.1017/s0033291713002237

Obsessive Compulsive Cognitions Working Group. (1997). Cognitive assessment of obsessive-compulsive disorder. *Behaviour Research and Therapy, 35*(7), 667-681. doi: 10.1016/S0005-7967(97)00017-X

Obsessive Compulsive Cognitions Working Group. (2001). Development and initial validation of the obsessive beliefs questionnaire and the interpretation of intrusions inventory. *Behaviour Research and Therapy, 39*(8), 987-1006. doi: 10.1016/S0005-7967(00)00085-1

Obsessive Compulsive Cognitions Working Group. (2005). Psychometric validation of the obsessive belief questionnaire and interpretation of intrusions inventory-part 2: Factor analyses and testing of a brief version. *Behaviour Research and Therapy, 43*(11), 1527-1542. doi: 10.1016/j.brat.2004.07.010

Olatunji, B. O., Cisler, J., McKay, D., & Phillips, M. L. (2010). Is disgust associated with psychopathology? Emerging research in the anxiety disorders. *Psychiatry Research, 175*(1-2), 1-10. doi: 10.1016/j.psychres.2009.04.007

Olatunji, B. O., Etzel, E. N., Tomarken, A. J., Ciesielski, B. G., & Deacon, B. (2011). The effects of safety behaviors on health anxiety: An experimental investigation. *Behaviour Research and Therapy, 49*(11), 719-728. doi: 10.1016/j.brat.2011.07.008

Rachman, S. (1994). Pollution of the mind. *Behaviour Research and Therapy, 32*(3), 311-314. doi: 10.1016/0005-7967(94)90127-9

Rachman, S. (2004a). *Anxiety* (2nd ed.). East Sussex, England: Psychology Press.

Rachman, S. (2004b). Fear of contamination. *Behaviour Research and Therapy, 42*(11), 1227-1255. doi: 10.1016/j.brat.2003.10.009

Rachman, S. (2010). Betrayal: A psychological analysis. *Behaviour Research and Therapy, 48*(4), 304-311. doi: 10.1016/j.brat.2009.12.002

Radomsky, A. S., Rachman, S., & Hammond, D. (2001). Memory bias, confidence and responsibility in compulsive checking. *Behaviour Research and Therapy, 39*(7), 813-822. doi: 10.1016/S0005-7967(00)00079-6

Rasmussen, S. A., & Eisen, J. L. (1992). The epidemiology and clinical-features of obsessive-compulsive disorder. *Psychiatric Clinics of North America, 15*(4), 743-758.

Rauch, S. L., Jenike, M. A., Alpert, N. M., Baer, L., Breiter, H. C. R., Savage, C. R., & Fischman, A. J. (1994). Regional cerebral blood flow measured during symptom provocation in obsessive-compulsive disorder using oxygen 15-labeled carbon dioxide and positron emission tomography. *Archives of General Psychiatry, 51*(1), 62-70. doi: 10.1001/archpsyc.1994.03950010062008

Rassin, E. (2001). The contribution of thought–action fusion and thought suppression in the development of obsession-like intrusions in normal participants. *Behaviour Research and Therapy, 39*(9), 1023-1032. doi: 10.1016/S0005-7967(00)00075-9

Reinholdt-Dunne, M., Mogg, K., & Bradley, B. P. (2009). Effects of anxiety and attention control on processing pictorial and linguistic emotional information. *Behaviour Research and Therapy, 47*(5), 410-417. doi: 10.1016/j.brat.2009.01.012

Rossi, V., & Pourtois, G. (2012). Transient state-dependent fluctuations in anxiety measured using STAI, POMS, PANAS or VAS: A comparative review. *Anxiety Stress and Coping, 25*(6), 603-645. doi: 10.1080/10615806.2011.582948

Rotge, J. Y., Guehl, D., Dilharreguy, B., Tignol, J., Bioulac, B., Allard, M., . . . Aouizerate, B. (2009). Meta-analysis of brain volume changes in obsessive-compulsive disorder. *Biological Psychiatry, 65*(1), 75-83. doi: 10.1016/j.biopsych.2008.06.019

Rozin, P., & Fallon, A. E. (1987). A perspective on disgust. *Psychological Review, 94*(1), 23-41. doi: 10.1037//0033-295x.94.1.23

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the national comorbidity survey replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/Mp.2008.94

Salkovskis, P. M. (1999). Understanding and treating obsessive-compulsive disorder. *Behaviour Research and Therapy, 37*, S29-S52. doi:10.1016/S0005-7967(99)00049-2

Schienle, A., Schäfer, A., Stark, R., Walter, B., & Vaitl, D. (2005). Neural responses of OCD patients towards disorder-relevant, generally disgust-inducing and fear-inducing pictures. *International Journal of Psychophysiology, 57*(1), 69-77. doi: 10.1016/j.ijpsycho.2004.12.013

Shafran, R., & Rachman, S. (2004). Thought-action fusion: A review. *Journal of Behavioral Therapy and Experimental Psychiatry, 35*(2), 87-107. doi: 10.1016/j.jbtep.2004.04.002

Shin, L. M., Dougherty, D. D., Orr, S. P., Pitman, R. K., Lasko, M., Macklin, M. L., . . . Rauch, S. L. (2000). Activation of anterior paralimbic structures during guilt-related script-driven imagery. *Biological Psychiatry, 48*(1), 43-50. doi: 10.1016/s0006-3223(00)00251-1

Simpson, H. B., Tenke, C. E., Towey, J. B., Liebowitz, M. R., & Bruder, G. E. (2000). Symptom provocation alters behavioral ratings and brain electrical activity in obsessive-compulsive disorder: A preliminary study. *Psychiatry Research, 95*(2), 149-155. doi: 10.1016/S0165-1781(00)00177-3

Stormark, K. M., & Torkildsen, Ø. (2004). Selective processing of linguistic and pictorial food stimuli in females with anorexia and bulimia nervosa. *Eating Behaviors, 5*(1), 27-33. doi: 10.1016/j.eatbeh.2003.07.002

Suda, M., Brooks, S. J., Giampietro, V., Uher, R., Mataix-Cols, D., Brammer, M. J., . . . Campbell, I. C. (2014). Provocation of symmetry/ordering symptoms in anorexia nervosa: A functional neuroimaging study. *PLoS ONE, 9*(5), e97998. doi: 10.1371/journal.pone.0097998

Summerfeldt, L. J., Kloosterman, P. H., Antony, M. M., & Swinson, R. P. (2014). Examining an obsessive-compulsive core dimensions model: Structural validity of harm avoidance and incompleteness. *Journal of Obsessive-Compulsive and Related Disorders, 3*(2), 83-94. doi: 10.1016/j.jocrd.2014.01.003

Toffolo, M. B. J., van den Hout, M. A., Hooge, I. T. C., Engelhard, I. M., & Cath, D. C. (2013). Mild uncertainty promotes checking behavior in subclinical obsessive-compulsive disorder. *Clinical Psychological Science, 1*(2), 103-109. doi: 10.1177/2167702612472487

Zohar, J., Insel, T. R., Berman, K. F., Foa, E. B., Hill, J. L., & Weinberger, D. R. (1989). Anxiety and cerebral blood flow during behavioral challenge: Dissociation of central from peripheral and subjective measures. *Archives of General Psychiatry, 46*(6), 505-510. doi: 10.1001/archpsyc.1989.0181006002

Appendix A

Table A.1

Summary of studies included in the review

Authors	**Comparison**	**Number of participants**	**Intervention category**	**Idiosyncratic?**	**Symptom dimension focus**	**Modality**	**Design**	**Outcome**	**Hedges's *g***	**Standard error**
Adams and Lohr (2012)	healthy	33	disgust	no	contamination	objects	within	anxiety	0.16	0.13
Adler et al. (2000)	OCD	7	threat	yes	all	objects	within	anxiety	1.33	0.51
Agarwal et al. (2013)	OCD vs. healthy	9 vs. 9	threat	no	contamination	visual	between	anxiety	0.51	0.40
Alcolado and Radomsky (2011)	healthy	43 vs. 48	perfectionism/ certainty	no	checking	verbal	between	compulsion urge	0.48	0.21
Armstrong, Sarawgi, and Olatunji (2012)	subclinical vs. healthy	19 vs. 20	disgust	no	contamination	objects	between	anxiety	1.78	0.37
Arntz, Voncken, and Goosen (2007)	healthy	14 vs. 14	responsibility	no	checking	verbal	between	anxiety, compulsion urge, compulsions, and obsessions	0.30	0.37

	OCD	13 vs. 14	responsibility	no	checking	verbal	between	anxiety, compulsion urge, compulsions, and obsessions	0.49	0.38
	OCD vs. healthy	13 vs. 14	responsibility	no	checking	verbal	between	anxiety, compulsion urge, compulsions, and obsessions	0.95	0.40
Baioui et al. (2013)	healthy	19	threat	no	contamination	visual	within	compulsion urge	3.34	0.72
	OCD	19	threat	both	contamination	visual	within	compulsion urge	8.10	1.51
	OCD vs. healthy	19 vs. 19	threat	no	contamination	visual	between	compulsion urge	1.45	0.29
Berman, Abramowitz, Pardue, and Wheaton (2010)	healthy	73	TAF	no	all	verbal	within	anxiety	1.04	0.18
Berman, Wheaton, and Abramowitz	healthy	62	TAF	no	all	verbal	within	anxiety	0.91	0.29

(2012)										
Berman, Wheaton, Fabricant, Jacobson, and Abramowitz (2011)	healthy	107	TAF	no	all	verbal	within	anxiety	0.51	0.28
Berney, Sookman, Leyton, Young, and Benkelfat (2006)	OCD	16	threat	yes	all	objects	within	anxiety, compulsion urge, compulsions, and obsessions	0.49	0.20
Bocci and Gordon (2007)	healthy	49	TAF	no	all	visual	within	anxiety	1.71	0.24
Boschen and Vuksanovic (2007)	healthy	20 vs. 19	repeated compulsions and responsibility	no	checking	verbal and behavior	both	compulsion urge	0.27	0.24
	OCD	7 vs. 7	repeated compulsions and responsibility	no	checking	verbal and behavior	both	compulsion urge	0.50	0.31
	OCD vs. healthy	14 vs. 39	repeated compulsions	no	checking	verbal and behavior	between	compulsion urge	0.11	0.30

			and responsibility							
Brady and Lohr (2014)	subclinical vs. healthy	20 vs. 20	threat	no	contamination	objects	between	anxiety	1.89	0.38
Broderick, Grisham, and Weidemann (2013)	subclinical vs. healthy	26 vs. 26	disgust	no	contamination	visual	between	anxiety	1.48	0.31
Chen, Xie, Han, Cui, and Zhang (2004)	OCD	13	threat	yes	contamination	objects	within	anxiety and symptoms	4.46	1.03
Coles, Heimberg, Frost, and Steketee (2005)	subclinical vs. healthy	25 vs. 25	perfectionism/ certainty	no	all	objects	between	anxiety and compulsion urge	0.53	0.28
Cottraux et al. (1996)	OCD vs. healthy	10 vs. 10	threat	yes	checking	verbal	between	anxiety, compulsion urge, compulsions, and symptoms	0.82	0.45
Coughtrey, Shafran, and Rachman (2014) experiment 1	healthy	40	mental contamination	yes	contamination	verbal	within	anxiety and compulsion urge	0.92	0.19
Coughtrey, Shafran, and Rachman (2014)	subclinical	60	mental contamination	no	contamination	verbal	within	anxiety and compulsion	0.76	0.15

experiment 2			and threat					urge		
Cougle, Fitch, Jacobson, and Lee (2013) experiment 2	healthy	38	repeated compulsions	no	checking	behavior	within	anxiety	0.74	0.14
Cougle, Fitch, Jacobson, and Lee (2013) experiment 3	healthy	69 vs. 68	repeated compulsions and perfectionism/ certainty	no	checking	behavior and objects	both	anxiety	0.30	0.12
Cougle, Goetz, Hawkins, and Fitch (2012) experiment 2	healthy	28 vs. 29	mental contamination	yes	contamination	verbal	between	compulsions	0.85	0.27
Cougle, Purdon, Fitch, and Hawkins (2013) experiment 2	healthy	88	TAF	no	all	verbal	within	anxiety	0.72	0.10
Cuttler, Sirois-Delisle, Alcolado, Radomsky, and Taylor (2013)	healthy	42 vs. 63	perfectionism/ certainty	no	checking	verbal	between	compulsion urge	0.67	0.20
Davey, Bickerstaffe, and MacDonald (2006)	healthy	25	disgust	no	contamination	verbal	within	anxiety	-0.14	0.10
Davey, MacDonald, and Brierley (2008)	healthy	30	disgust	no	contamination	verbal	within	anxiety	0.32	0.18

Davey, Meeten, Barnes, and Dash (2013) experiment 1	healthy	30 vs. 30	threat	no	all	combination	between	anxiety	0.69	0.26
Davey, Meeten, Barnes, and Dash (2013) experiment 2	healthy	14 vs. 15	threat	no	all	combination	between	anxiety	1.03	0.39
de Vries et al. (2013)	healthy	10	perfectionism/ certainty	no	symmetry	visual	within	anxiety and compulsion urge	0.18	0.23
de Wit et al. (2015)	healthy	39	threat	no	all	visual	within	anxiety	0.38	0.17
	OCD	43	threat	no	all	visual	within	anxiety	1.21	0.19
	OCD vs. healthy	43 vs. 39	threat	no	all	visual	between	anxiety	1.12	0.24
Deacon and Maack (2008)	healthy	30	repeated compulsions	no	contamination	behavior	within	symptoms	0.66	0.13
	subclinical vs. healthy	26 vs. 30	threat and repeated compulsions	no	contamination	objects and behavior	between	anxiety, obsessions, and symptoms	0.80	0.28
	subclinical	26	repeated compulsions	no	contamination	behavior	within	symptoms	0.33	0.13
Dorfan and Woody (2011)	healthy	103	disgust	no	contamination	objects	within	anxiety	0.93	0.09

Doron, Derby, Szepsenwol, and Talmor (2012) experiment 1	healthy	14 vs. 15	mental contamination	no	contamination	visual	between	compulsion urge	0.90	0.38
Doron, Derby, Szepsenwol, and Talmor (2012) experiment 2	healthy	39 vs. 38	mental contamination	no	contamination	visual	between	compulsion urge	0.50	0.23
Doron, Derby, Szepsenwol, and Talmor (2012) experiment 3	healthy	41 vs. 45	mental contamination	no	contamination	visual	between	compulsion urge	0.49	0.22
Elliott and Radomsky (2009)	healthy	35 vs. 35	mental contamination	no	contamination	verbal	between	anxiety, compulsion urge, and compulsions	1.27	0.52
Elliott and Radomsky (2012)	healthy	35 vs. 33	mental contamination	no	contamination	verbal	between	anxiety, compulsion urge, and compulsions	1.73	0.55
Fairbrother, Newth, and Rachman (2005)	healthy	91 vs. 30	mental contamination	no	contamination	verbal	between	anxiety, compulsion urge, and	1.70	0.40

								compulsions		
Fitch and Cougle (2013) study 2	healthy	130	repeated compulsions	no	checking	behavior	within	anxiety	0.26	0.08
Hendler et al. (2003)	OCD	13	threat	yes	all	objects	within	anxiety	1.14	0.25
Herba and Rachman (2007)	healthy	100 vs. 20	mental contamination	no	contamination	verbal	between	compulsion urge, and compulsions	1.81	0.59
Jones and Bhattacharya (2014)	OCD vs. healthy	23 vs. 24	TAF	no	all	verbal	between	anxiety and compulsion urge	0.78	0.30
Kim et al. (2008)	OCD vs. healthy	33 vs. 30	threat	no	checking	visual	between	anxiety and compulsions	0.71	0.26
Kim et al. (2010)	OCD vs. healthy	30 vs. 27	threat	no	checking	visual	between	compulsions	0.77	0.27
Kim et al. (2012)	OCD vs. healthy	22 vs. 31	threat	no	checking	visual	between	compulsions	0.67	0.28
Ladouceur et al. (1995) experiment 1	healthy	30 vs. 30	responsibility	no	checking	verbal	between	anxiety, compulsion urge, and compulsions	0.23	0.26
Ladouceur et al. (1995) experiment 2	healthy	20 vs. 20	responsibility	no	checking	verbal	between	anxiety, compulsion	0.43	0.32

								urge, and compulsions		
Lopatka and Rachman (1995) experiment 1	OCD	30	responsibility	no	checking	verbal	within	anxiety and compulsion urge	1.42	0.19
Mancini, D'Olimpio, and Cieri (2004)	healthy	9 vs. 13	responsibility	no	checking	verbal	between	anxiety and compulsions	0.49	0.43
Mancini, Gangemi, Perdighe, and Marini (2008) experiment 1	healthy	55 vs. 49	mental contamination	yes	contamination	verbal	between	anxiety and compulsions	0.96	0.82
Mancini, Gangemi, Perdighe, and Marini (2008) experiment 2	healthy	75 vs. 35	mental contamination	yes	contamination	verbal	between	anxiety and compulsions	0.67	0.46
Marcks and Woods (2007)	healthy	117	TAF	no	all	verbal	within	anxiety	0.81	0.11
Marks et al. (2000)	OCD	13	threat	yes	all	verbal	within	anxiety	1.52	0.40
Marzillier and Davey (2005)	healthy	20	disgust	no	contamination	combination	within	anxiety	0.04	0.13
Mataix-Cols et al. (2004)	OCD vs. healthy	16 vs. 17	threat	no	checking and contamination	visual	between	anxiety	0.94	0.36
Mataix-Cols et al. (2008)	healthy	37	disgust	no	contamination	visual	within	anxiety	2.38	0.32

Mayer, Muris, Busser, and Bergamin (2009)	healthy	31	disgust	no	contamination	combination	within	anxiety	0.20	0.12
Meeten, Dash, Scarlet, and Davey (2012)	healthy	25 vs. 21	perfectionism/ certainty	no	all	combination	between	anxiety	0.64	0.30
Morgiève et al. (2014)	OCD	34	threat	both	checking	visual	within	anxiety	2.21	0.28
Myers and Wells (2013)	healthy	16 vs. 16	TAF	no	all	verbal	between	anxiety and obsessions	0.15	0.35
	subclinical vs. healthy	16 vs. 16	TAF	no	all	verbal	between	anxiety and obsessions	0.29	0.35
	subclinical	16 vs. 16	TAF	no	all	verbal	between	anxiety and obsessions	1.21	0.38
Najmi, Tobin, and Amir (2012)	subclinical vs. healthy	62 vs. 39	disgust	no	contamination	objects	between	anxiety	0.77	0.21
Olafsson et al. (2013)	subclinical vs. healthy	film: 30 vs. 30 other inductions: 15 vs. 15	disgust	no	contamination	objects and visual	between	anxiety, compulsion urge, and obsessions	0.54	0.34
Olatunji et al. (2014)	OCD	12	threat	no	contamination	visual	within	anxiety	2.01	0.49
Olatunji, Lohr, Sawchuk, and Tolin (2007)	subclinical vs. healthy	30 vs. 30	disgust and threat	no	contamination	objects and visual	between	anxiety	0.50	0.26
Olatunji and Armstrong (2009)	healthy	21	disgust	no	contamination	combination	within	anxiety	0.17	0.23
	subclinical vs.	36 vs. 47	disgust	no	contamination	objects and	between	anxiety	0.56	0.32

	healthy subclinical	21	disgust	no	contamination	combination combination	within	anxiety	0.29	0.21
Olatunji, Etzel, Tomarken, Ciesielski, and Deacon (2011)	healthy	30 vs. 30	repeated compulsions	no	contamination	behavior	between	compulsions	0.49	0.26
Rachman, Radomsky, Elliott, and Zysk (2012) experiment 1	healthy	19 vs. 20	mental contamination	no	contamination	verbal	between	compulsion urge and compulsions	0.17	0.32
Rachman, Radomsky, Elliott, and Zysk (2012) experiment 2	healthy	20 vs. 20	mental contamination	no	contamination	verbal	between	compulsion urge and compulsions	0.37	0.32
Rachman, Radomsky, Elliott, and Zysk (2012) experiment 3	healthy	20 vs. 20	mental contamination	no	contamination	verbal	between	compulsion urge and compulsions	0.79	0.33
Rachman, Radomsky, Elliott, and Zysk (2012) experiment 4	healthy	20 vs. 20	mental contamination	no	contamination	verbal	between	compulsion urge and compulsions	0.77	0.51
Radomsky and Rachman (1999)	healthy OCD OCD vs. healthy	20 10 10 vs. 20	threat threat threat	no no no	contamination contamination contamination	objects objects objects	within within between	anxiety anxiety anxiety	0.09 0.14 1.65	0.05 0.11 0.43
Radomsky, Rachman, and Hammond (2001)	OCD	11	responsibility	no	checking	verbal	within	anxiety	0.74	0.24

Radomsky, Senn, Lahoud, and Gelfand (2014)	healthy	49 vs. 47	threat	no	contamination	verbal	between	anxiety and compulsion urge	1.10	0.22
Rassin (2001)	healthy	19	TAF	no	all	verbal	within	anxiety	0.69	0.32
Rassin, Merckelbach, Muris, and Spaan (1999)	healthy	19 vs. 26	TAF	no	all	verbal	between	anxiety and obsessions	1.17	0.32
Rauch et al. (2002)	OCD	9	threat	yes	contamination	objects	within	anxiety and symptoms	1.30	0.43
Reuven, Liberman, and Dar (2014)	OCD vs. healthy	15 vs. 15	mental contamination	yes	all	verbal	between	anxiety	0.58	0.36
Rotge et al. (2012)	OCD vs. healthy	14 vs. 14	perfectionism/ certainty	no	checking	visual	between	anxiety and compulsions	0.49	0.37
Schienle, Schafer, Stark, Walter, and Vaitl (2005)	healthy	10	disgust	no	all	visual	within	anxiety	0.51	0.20
	OCD	10	threat and disgust	threat: yes disgust: no	all	visual	within	anxiety and symptoms	0.95	0.44
	OCD vs. healthy	10 vs. 10	disgust	no	contamination	visual	between	anxiety	0.97	0.46
Senn and Radomsky (2012)	healthy	33 vs. 48	disgust	no	contamination	verbal	between	anxiety and compulsion urge	-0.01	0.22
Shafran (1997)	OCD	36	responsibility	no	all	objects	within	anxiety and compulsion	0.47	0.17

								urge		
Y. W. Shin et al. (2006)	OCD	12	threat	yes	all	objects	within	anxiety	1.57	0.41
L. M. Shin et al. (2000)	healthy	8	mental contamination	yes	all	verbal	within	anxiety	0.94	0.39
Simpson, Tenke, Towey, Liebowitz, and Bruder (2000)	OCD	6	threat	no	contamination	objects and verbal	within	symptoms	2.13	0.79
Suda et al. (2014)	healthy	24	perfectionism/ certainty	no	symmetry	visual	within	anxiety	1.06	0.17
Summers, Fitch, and Cougle (2014)	healthy	284	perfectionism/ certainty	no	all	behavior, objects and verbal	within	anxiety	0.63	0.07
Toffolo, van den Hout, Hooge, Engelhard, and Cath (2013)	subclinical vs. healthy	34 vs. 31	perfectionism/ certainty	no	checking	visual	between	compulsions	0.47	0.25
Toffolo, van den Hout, Engelhard, Hooge, and Cath (2014)	subclinical vs. healthy	54 vs. 55	perfectionism/ certainty	no	checking	visual	between	compulsions	0.29	0.19
van den Heuvel et al. (2004)	healthy	10	disgust	no	contamination	visual	within	anxiety and obsessions	0.26	0.28
	OCD	11	disgust	no	contamination	visual	within	anxiety and obsessions	0.74	0.32

	OCD vs. healthy	11 vs. 10	disgust	no	contamination	visual	between	anxiety and obsessions	1.21	0.47

Note. The number of participants in between-group comparisons is written as experimental group vs. control group. Whenever design is coded with "both", the last group of the between-group comparison was used for the within-group comparison. TAF = thought action fusion.

References Articles used in Meta-Analysis

Adams, T. G., & Lohr, J. M. (2012). Disgust mediates the relation between attentional shifting and contamination aversion. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(4), 975-980. doi: 10.1016/j.jbtep.2012.03.002

Adler, C. M., McDonough-Ryan, P., Sax, K. W., Holland, S. K., Arndt, S., & Strakowski, S. M. (2000). fMRI of neuronal activation with symptom provocation in unmedicated patients with obsessive compulsive disorder. *Journal of Psychiatric Research, 34*(4-5), 317-324. doi: 10.1016/s0022-3956(00)00022-4

Agarwal, S. M., Jose, D., Baruah, U., Shivakumar, V., Kalmady, S. V., Venkatasubramanian, G., . . . Reddy, Y. C. J. (2013). Neurohemodynamic correlates of washing symptoms in obsessive-compulsive disorder: A pilot fMRI study using symptom provocation paradigm. *Indian Journal of Psychological Medicine, 35*(1), 67-74. doi: 10.4103/0253-7176.112208

Alcolado, G. M., & Radomsky, A. S. (2011). Believe in yourself: Manipulating beliefs about memory causes checking. *Behaviour Research and Therapy, 49*(1), 42-49. doi: 10.1016/j.brat.2010.10.001

Armstrong, T., Sarawgi, S., & Olatunji, B. O. (2012). Attentional bias toward threat in contamination fear: Overt components and behavioral correlates. *Journal of Abnormal Psychology, 121*(1), 232-237. doi: 10.1037/a0024453

Arntz, A., Voncken, M., & Goosen, A. C. A. (2007). Responsibility and obsessive-compulsive disorder: An experimental test. *Behaviour Research and Therapy, 45*(3), 425-435. doi: 10.1016/j.brat.2006.03.016

Baioui, A., Pilgramm, J., Merz, C. J., Walter, B., Vaitl, D., & Stark, R. (2013). Neural response in obsessive-compulsive washers depends on individual fit of triggers. *Frontiers in Human Neuroscience, 7*. 1-13. doi: 10.3389/fnhum.2013.00143

Berman, N. C., Abramowitz, J. S., Pardue, C. M., & Wheaton, M. G. (2010). The relationship between religion and thought action fusion: Use of an in vivo paradigm. *Behaviour Research and Therapy, 48*(7), 670-674. doi: 10.1016/j.brat.2010.03.021

Berman, N. C., Wheaton, M. G., & Abramowitz, J. S. (2012). The "Arnold Schwarzenegger effect": Is strength of the "victim" related to misinterpretations

of harm intrusions? *Behaviour Research and Therapy, 50*(12), 761-766. doi: 10.1016/j.brat.2012.09.002

Berman, N. C., Wheaton, M. G., Fabricant, L. E., Jacobson, S. R., & Abramowitz, J. S. (2011). The effects of familiarity on thought-action fusion. *Behaviour Research and Therapy, 49*(10), 695-699. doi: 10.1016/j.brat.2011.07.010

Berney, A., Sookman, D., Leyton, M., Young, S. N., & Benkelfat, C. (2006). Lack of effects on core obsessive-compulsive symptoms of tryptophan depletion during symptom provocation in remitted obsessive-compulsive disorder patients. *Biological Psychiatry, 59*(9), 853-857. doi: 10.1016/j.biopsych.2005.08.023

Bocci, L., & Gordon, P. K. (2007). Does magical thinking produce neutralising behaviour? An experimental investigation. *Behaviour Research and Therapy, 45*(8), 1823-1833. doi: 10.1016/j.brat.2007.02.003

Boschen, M. J., & Vuksanovic, D. (2007). Deteriorating memory confidence, responsibility perceptions and repeated checking: Comparisons in OCD and control samples. *Behaviour Research and Therapy, 45*(9), 2098-2109. doi: 10.1016/j.brat.2007.03.009

Brady, R. E., & Lohr, J. M. (2014). A behavioral test of contamination fear in excessive health anxiety. *Journal of Behavior Therapy and Experimental Psychiatry, 45*(1), 122-127. doi: 10.1016/j.jbtep.2013.09.011

Broderick, J., Grisham, J. R., & Weidemann, G. (2013). Disgust and fear responding in contamination-based obsessive-compulsive disorder during pictorial exposure. *Behavior Therapy, 44*(1), 27-38. doi: 10.1016/j.beth.2012.05.005

Chen, X. L., Xie, J. X., Han, H. B., Cui, Y. H., & Zhang, B. Q. (2004). MR perfusion-weighted imaging and quantitative analysis of cerebral hemodynamics with symptom provocation in unmedicated patients with obsessive-compulsive disorder. *Neuroscience Letters, 370*(2-3), 206-211. doi: 10.1016/j.neulet.2004.08.019

Coles, M. E., Heimberg, R. G., Frost, R. O., & Steketee, G. (2005). Not just right experiences and obsessive-compulsive features: Experimental and self-monitoring perspectives. *Behaviour Research and Therapy, 43*(2), 153-167. doi: 10.1016/j.brat.2004.01.002

Cottraux, J., Gerard, D., Cinotti, L., Froment, J. C., Deiber, M. P., Le Bars, D., . . . Mauguiere, F. (1996). A controlled positron emission tomography study of

obsessive and neutral auditory stimulation in obsessive-compulsive disorder with checking rituals. *Psychiatry Research, 60*(2-3), 101-112. doi: 10.1016/0165-1781(96)02697-2

Coughtrey, A. E., Shafran, R., & Rachman, S. J. (2014). The spontaneous decay and persistence of mental contamination: An experimental analysis. *Journal of Behavior Therapy and Experimental Psychiatry, 45*(1), 90-96. doi: 10.1016/j.jbtep.2013.09.001

Cougle, J. R., Fitch, K. E., Jacobson, S., & Lee, H. J. (2013). A multi-method examination of the role of incompleteness in compulsive checking. *Journal of Anxiety Disorders, 27*(2), 231-239. doi: 10.1016/j.janxdis.2013.02.003

Cougle, J. R., Goetz, A. R., Hawkins, K. A., & Fitch, K. E. (2012). Guilt and compulsive washing: Experimental tests of interrelationships. *Cognitive Therapy and Research, 36*(4), 358-366. doi: 10.1007/s10608-011-9359-x

Cougle, J. R., Purdon, C., Fitch, K. E., & Hawkins, K. A. (2013). Clarifying relations between thought-action fusion, religiosity, and obsessive-compulsive symptoms through consideration of intent. *Cognitive Therapy and Research, 37*(2), 221-231. doi: 10.1007/s10608-012-9461-8

Cuttler, C., Sirois-Delisle, V., Alcolado, G. M., Radomsky, A. S., & Taylor, S. (2013). Diminished confidence in prospective memory causes doubts and urges to check. *Journal of Behavior Therapy and Experimental Psychiatry, 44*(3), 329-334. doi: 10.1016/j.jbtep.2013.01.001

Davey, G. C. L., Bickerstaffe, S., & MacDonald, B. A. (2006). Experienced disgust causes a negative interpretation bias: A causal role for disgust in anxious psychopathology. *Behaviour Research and Therapy, 44*(10), 1375-1384. doi: 10.1016/j.brat.2005.10.006

Davey, G. C. L., MacDonald, B. A., & Brierley, L. (2008). The effect of disgust on anxiety ratings to fear-relevant, disgust-relevant and fear-irrelevant stimuli. *Journal of Anxiety Disorders, 22*(8), 1347-1354. doi: 10.1016/j.janxdis.2008.01.015

Davey, G. C. L., Meeten, F., Barnes, G., & Dash, S. R. (2013). Aversive intrusive thoughts as contributors to inflated responsibility, intolerance of uncertainty, and thought-action fusion. *Clinical Neuropsychiatry, 10*(3), 30-44.

de Vries, F. E., van den Heuvel, O. A., Cath, D. C., Groenewegen, H. J., van Balkom, A. J., Boellaard, R., . . . Veltman, D. J. (2013). Limbic and motor circuits involved in symmetry behavior in Tourette's syndrome. *CNS Spectrums, 18*(1), 34-42. doi: 10.1017/s1092852912000703

de Wit, S. J., van der Werf, Y. D., Mataix-Cols, D., Trujillo, J. P., van Oppen, P., Veltman, D. J., & van den Heuvel, O. A. (2015). Emotion regulation before and after transcranial magnetic stimulation in obsessive compulsive disorder. *Psychological Medicine*, 1-15. doi: 10.1017/s0033291715001026

Deacon, B., & Maack, D. J. (2008). The effects of safety behaviors on the fear of contamination: An experimental investigation. *Behaviour Research and Therapy, 46*(4), 537-547. doi: 10.1016/j.brat.2008.01.010

Dorfan, N. M., & Woody, S. R. (2011). Danger appraisals as prospective predictors of disgust and avoidance of contaminants. *Journal of Social and Clinical Psychology, 30*(2), 105-132.

Doron, G., Derby, D. S., Szepsenwol, O., & Talmor, D. (2012). Tainted love: Exploring relationship-centered obsessive compulsive symptoms in two non-clinical cohorts. *Journal of Obsessive-Compulsive and Related Disorders, 1*(1), 16-24. doi: 10.1016/j.jocrd.2011.11.002

Elliott, C. M., & Radomsky, A. S. (2009). Analyses of mental contamination: Part I, experimental manipulations of morality. *Behaviour Research and Therapy, 47*(12), 995-1003. doi: 10.1016/j.brat.2009.03.004

Elliott, C. M., & Radomsky, A. S. (2012). Mental contamination: The effects of imagined physical dirt and immoral behaviour. *Behaviour Research and Therapy, 50*(6), 422-427. doi: 10.1016/j.brat.2012.03.007

Fairbrother, N., Newth, S. J., & Rachman, S. (2005). Mental pollution: Feelings of dirtiness without physical contact. *Behaviour Research and Therapy, 43*(1), 121-130. doi: 10.1016/j.brat.2003.12.005

Fitch, K. E., & Cougle, J. R. (2013). An evaluation of obsessive beliefs as predictors of performance on in vivo assessments of obsessive-compulsive symptoms. *Cognitive Therapy and Research, 37*(2), 207-220. doi: 10.1007/s10608-012-9457-4

Hendler, T., Goshen, E., Zwas, S. T., Sasson, Y., Gal, G., & Zohar, J. (2003). Brain reactivity to specific symptom provocation indicates prospective therapeutic outcome in OCD. *Psychiatry Research-Neuroimaging, 124*(2), 87-103. doi: 10.1016/s0925-4927(03)00091-x

Herba, J. K., & Rachman, S. (2007). Vulnerability to mental contamination. *Behaviour Research and Therapy, 45*(11), 2804-2812. doi: 10.1016/j.brat.2007.07.010

Jones, R., & Bhattacharya, J. (2014). A role for the precuneus in thought-action fusion: Evidence from participants with significant obsessive-compulsive symptoms. *Neuroimage: Clinical, 4*, 112-121. doi: 10.1016/j.nicl.2013.11.008

Kim, K., Kim, C.-H., Cha, K. R., Park, J., Han, K., Kim, Y. K., . . . Kim, S. I. (2008). Anxiety provocation and measurement using virtual reality in patients with obsessive-compulsive disorder. *Cyberpsychology & Behavior, 11*(6), 637-641. doi: 10.1089/cpb.2008.0003

Kim, K., Kim, S. I., Cha, K. R., Park, J., Rosenthal, M. Z., Kim, J. J., . . . Kim, C. H. (2010). Development of a computer-based behavioral assessment of checking behavior in obsessive-compulsive disorder. *Comprehensive Psychiatry, 51*(1), 86-93. doi: 10.1016/j.comppsych.2008.12.001

Kim, K., Roh, D., Kim, C.-H., Cha, K. R., Rosenthal, M. Z., & Kim, S. I. (2012). Comparison of checking behavior in adults with or without checking symptom of obsessive-compulsive disorder using a novel computer-based measure. *Computer Methods and Programs in Biomedicine, 108*(1), 434-441. doi: 10.1016/j.cmpb.2012.03.014

Ladouceur, R., Rhéaume, J., Freeston, M. H., Aublet, F., Jean, K., Lachance, S., . . . de Pokomandy-morin, K. (1995). Experimental manipulations of responsibility: An analogue test for models of obsessive-compulsive disorder. *Behaviour Research and Therapy, 33*(8), 937-946. doi: 10.1016/0005-7967(95)00024-R

Lopatka, C., & Rachman, S. (1995). Perceived responsibility and compulsive checking: An experimental analysis. *Behaviour Research and Therapy, 33*(6), 673-684. doi: 10.1016/0005-7967(94)00089-3

Mancini, F., D'Olimpio, F., & Cieri, L. (2004). Manipulation of responsibility in non-clinical subjects: Does expectation of failure exacerbate obsessive-compulsive behaviors? *Behaviour Research and Therapy, 42*(4), 449-457. doi: 10.1016/s0005-7967(03)00153-0

Mancini, F., Gangemi, A., Perdighe, C., & Marini, C. (2008). Not just right experience: Is it influenced by feelings of guilt? *Journal of Behavior Therapy and Experimental Psychiatry, 39*(2), 162-176. doi: 10.1016/j.jbtep.2007.02.002

Marcks, B. A., & Woods, D. W. (2007). Role of thought-related beliefs and coping strategies in the escalation of intrusive thoughts: An analog to obsessive-compulsive disorder. *Behaviour Research and Therapy, 45*(11), 2640-2651. doi: 10.1016/j.brat.2007.06.012

Marks, I. M., O'Dwyer, A. M., Meehan, O., Greist, J., Baer, L., & McGuire, P. (2000). Subjective imagery in obsessive-compulsive disorder before and after exposure therapy - Pilot randomised controlled trial. *British Journal of Psychiatry, 176*, 387-391. doi: 10.1192/bjp.176.4.387

Marzillier, S., & Davey, G. (2005). Anxiety and disgust: Evidence for a unidirectional relationship. *Cognition and Emotion, 19*(5), 729-750. doi: 10.1080/02699930441000436

Mataix-Cols, D., An, S. K., Lawrence, N. S., Caseras, X., Speckens, A., Giampietro, V., . . . Phillips, M. L. (2008). Individual differences in disgust sensitivity modulate neural responses to aversive/disgusting stimuli. *European Journal of Neuroscience, 27*(11), 3050-3058. doi: 10.1111/j.1460-9568.2008.06311.x

Mataix-Cols, D., Wooderson, S., Lawrence, N., Brammer, M. J., Speckens, A., & Phillips, M. L. (2004). Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. *Archives of General Psychiatry, 61*(6), 564-576. doi: 10.1001/archpsyc.61.6.564

Mayer, B., Muris, P., Busser, K., & Bergamin, J. (2009). A disgust mood state causes a negative interpretation bias, but not in the specific domain of body-related concerns. *Behaviour Research and Therapy, 47*(10), 876-881. doi: 10.1016/j.brat.2009.07.001

Meeten, F., Dash, S. R., Scarlet, A. L. S., & Davey, G. C. L. (2012). Investigating the effect of intolerance of uncertainty on catastrophic worrying and mood. *Behaviour Research and Therapy, 50*(11), 690-698. doi: 10.1016/j.brat.2012.08.003

Morgieve, M., N'Diaye, K., Haynes, W. I., Granger, B., Clair, A. H., Pelissolo, A., & Mallet, L. (2014). Dynamics of psychotherapy-related cerebral haemodynamic changes in obsessive compulsive disorder using a personalized exposure task in

functional magnetic resonance imaging. *Psychological Medicine, 44*(7), 1461-1473. doi: 10.1017/s0033291713002237

Myers, S. G., & Wells, A. (2013). An experimental manipulation of metacognition: A test of the metacognitive model of obsessive-compulsive symptoms. *Behaviour Research and Therapy, 51*(4-5), 177-184. doi: 10.1016/j.brat.2013.01.007

Najmi, S., Tobin, A., & Amir, N. (2012). Psychometric properties of a behavioral test of contamination-related obsessive-compulsive symptoms. *Cognitive Therapy and Research, 36*(3), 228-233. doi: 10.1007/s10608-010-9332-0

Olafsson, R. P., Emmelkamp, P. M. G., Gunnarsdottir, E. R., Snaebjornsson, T. I., Olason, D. T., & Kristjansson, A. (2013). Suppressing disgust related thoughts and performance on a subsequent behavioural avoidance task: Implications for OCD. *Behaviour Research and Therapy, 51*(3), 152-160. doi: 10.1016/j.brat.2012.11.008

Olatunji, B. O., & Armstrong, T. (2009). Contamination fear and effects of disgust on distress in a public restroom. *Emotion, 9*(4), 592-597. doi: 10.1037/a0016109

Olatunji, B. O., Etzel, E. N., Tomarken, A. J., Ciesielski, B. G., & Deacon, B. (2011). The effects of safety behaviors on health anxiety: An experimental investigation. *Behaviour Research and Therapy, 49*(11), 719-728. doi: 10.1016/j.brat.2011.07.008

Olatunji, B. O., Ferreira-Garcia, R., Caseras, X., Fullana, M. A., Wooderson, S., Speckens, A., . . . Mataix-Cols, D. (2014). Predicting response to cognitive behavioral therapy in contamination-based obsessive-compulsive disorder from functional magnetic resonance imaging. *Psychological Medicine, 44*(10), 2125-2137. doi: 10.1017/s0033291713002766

Olatunji, B. O., Lohr, J. M., Sawchuk, C. N., & Tolin, D. F. (2007). Multimodal assessment of disgust in contamination-related obsessive-compulsive disorder. *Behaviour Research and Therapy, 45*(2), 263-276. doi: 10.1016/j.brat.2006.03.004

Rachman, S., Radomsky, A. S., Elliott, C. M., & Zysk, E. (2012). Mental contamination: The perpetrator effect. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(1), 587-593. doi: 10.1016/j.jbtep.2011.08.002

Radomsky, A. S., & Rachman, S. (1999). Memory bias in obsessive-compulsive disorder (OCD). *Behaviour Research and Therapy, 37*(7), 605-618. doi: 10.1016/S0005-7967(98)00151-X

Radomsky, A. S., Rachman, S., & Hammond, D. (2001). Memory bias, confidence and responsibility in compulsive checking. *Behaviour Research and Therapy, 39*(7), 813-822. doi: 10.1016/S0005-7967(00)00079-6

Radomsky, A. S., Senn, J. M., Lahoud, M., & Gelfand, L. A. (2014). An informational pathway to the development of a contamination-related memory bias. *Journal of Behavior Therapy and Experimental Psychiatry, 45*(3), 339-342. doi: 10.1016/j.jbtep.2014.02.006

Rassin, E. (2001). The contribution of thought-action fusion and thought suppression in the development of obsession-like intrusions in normal participants. *Behaviour Research and Therapy, 39*(9), 1023-1032. doi: 10.1016/S0005-7967(00)00075-9

Rassin, E., Merckelbach, H., Muris, P., & Spaan, V. (1999). Thought-action fusion as a causal factor in the development of intrusions. *Behaviour Research and Therapy, 37*(3), 231-237. doi: 10.1016/S0005-7967(98)00140-5

Rauch, S. L., Shin, L. M., Dougherty, D. D., Alpert, N. M., Fischman, A. J., & Jenike, M. A. (2002). Predictors of fluvoxamine response in contamination-related obsessive compulsive disorder: A PET symptom provocation study. *Neuropsychopharmacology, 27*(5), 782-791. doi: 10.1016/s0893-133x(02)00351-2

Reuven, O., Liberman, N., & Dar, R. (2014). The effect of physical cleaning on threatened morality in individuals with obsessive-compulsive disorder. *Clinical Psychological Science, 2*(2), 224-229. doi: 10.1177/2167702613485565

Rotge, J. Y., Langbour, N., Dilharreguy, B., Bordessoulles, M., Guehl, D., Bioulac, B., . . . Burbaud, P. (2012). Contextual and behavioral influences on uncertainty in obsessive-compulsive disorder. *Cortex, 62*, 1-10. doi: 10.1016/j.cortex.2012.12.010

Schienle, A., Schafer, A., Stark, R., Walter, B., & Vaitl, D. (2005). Neural responses of OCD patients towards disorder-relevant, generally disgust-inducing and fear-inducing pictures. *International Journal of Psychophysiology, 57*(1), 69-77. doi: 10.1016/j.ijpsycho.2004.12.013

Senn, J. M., & Radomsky, A. S. (2012). Well that changes everything! The genesis of memory bias for threat with implications for delayed onset in anxiety disorders. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(4), 1019-1025. doi: 10.1016/j.jbtep.2012.04.003

Shafran, R. (1997). The manipulation of responsibility in obsessive-compulsive disorder. *British Journal of Clinical Psychology, 36*(3), 397-407. doi: 10.1111/j.2044-8260.1997.tb01247.x

Shin, L. M., Dougherty, D. D., Orr, S. P., Pitman, R. K., Lasko, M., Macklin, M. L., . . . Rauch, S. L. (2000). Activation of anterior paralimbic structures during guilt-related script-driven imagery. *Biological Psychiatry, 48*(1), 43-50. doi: 10.1016/s0006-3223(00)00251-1

Shin, Y. W., Kwon, J. S., Kim, J. J., Kang, D. H., Youn, T., Kang, K. W., . . . Lee, M. C. (2006). Altered neural circuit for working memory before and after symptom provocation in patients with obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica, 113*(5), 420-429. doi: 10.1111/j.1600-0447.2005.00709.x

Simpson, H. B., Tenke, C. E., Towey, J. B., Liebowitz, M. R., & Bruder, G. E. (2000). Symptom provocation alters behavioral ratings and brain electrical activity in obsessive-compulsive disorder: A preliminary study. *Psychiatry Research, 95*(2), 149-155. doi: 10.1016/S0165-1781(00)00177-3

Suda, M., Brooks, S. J., Giampietro, V., Uher, R., Mataix-Cols, D., Brammer, M. J., . . . Campbell, I. C. (2014). Provocation of symmetry/ordering symptoms in anorexia nervosa: A functional neuroimaging study. *PLoS ONE, 9*(5), e97998. doi: 10.1371/journal.pone.0097998

Summers, B. J., Fitch, K. E., & Cougle, J. R. (2014). Visual, tactile, and auditory "Not Just Right" experiences: Associations with obsessive-compulsive symptoms and perfectionism. *Behavior Therapy, 45*(5), 678-689. doi: 10.1016/j.beth.2014.03.008

Toffolo, M. B. J., van den Hout, M. A., Engelhard, I. M., Hooge, I. T. C., & Cath, D. C. (2014). Uncertainty, checking, and intolerance of uncertainty in subclinical obsessive compulsive disorder: An extended replication. *Journal of Obsessive-*

Compulsive and Related Disorders, 3(4), 338-344. doi: 10.1016/j.jocrd.2014.08.004

Toffolo, M. B. J., van den Hout, M. A., Hooge, I. T. C., Engelhard, I. M., & Cath, D. C. (2013). Mild uncertainty promotes checking behavior in subclinical obsessive-compulsive disorder. *Clinical Psychological Science, 1*(2), 103-109. doi: 10.1177/2167702612472487

van den Heuvel, O. A., Veltman, D. J., Groenewegen, H. J., Dolan, R. J., Cath, D. C., Boellaard, R., . . . van Dyck, R. (2004). Amygdala activity in obsessive-compulsive disorder with contamination fear: A study with oxygen-15 water positron emission tomography. *Psychiatry Research: Neuroimaging, 132*(3), 225-237. doi: 10.1016/j.pscychresns.2004.06.007

Appendix B: Funnel Plots

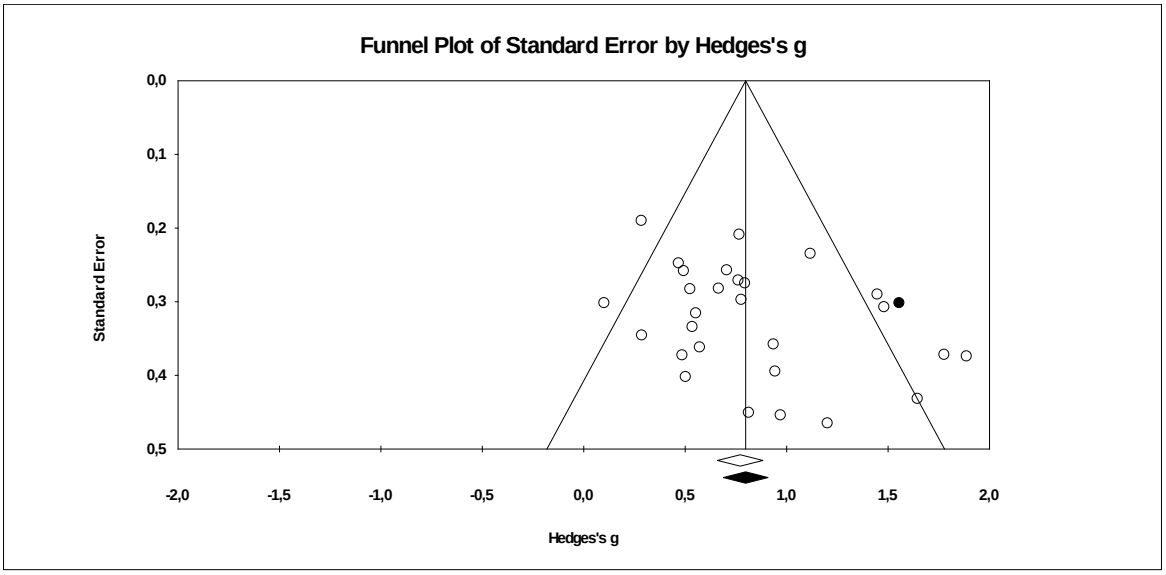

Figure B.1. Funnel plot of standard error by Hedges's *g* of publication bias for the direct comparison of (sub)clinical OCD versus healthy controls. The white dots represent the included studies and the black dots represent imputed studies based on Duval and Tweedie's (2000) trim-and-fill procedure.

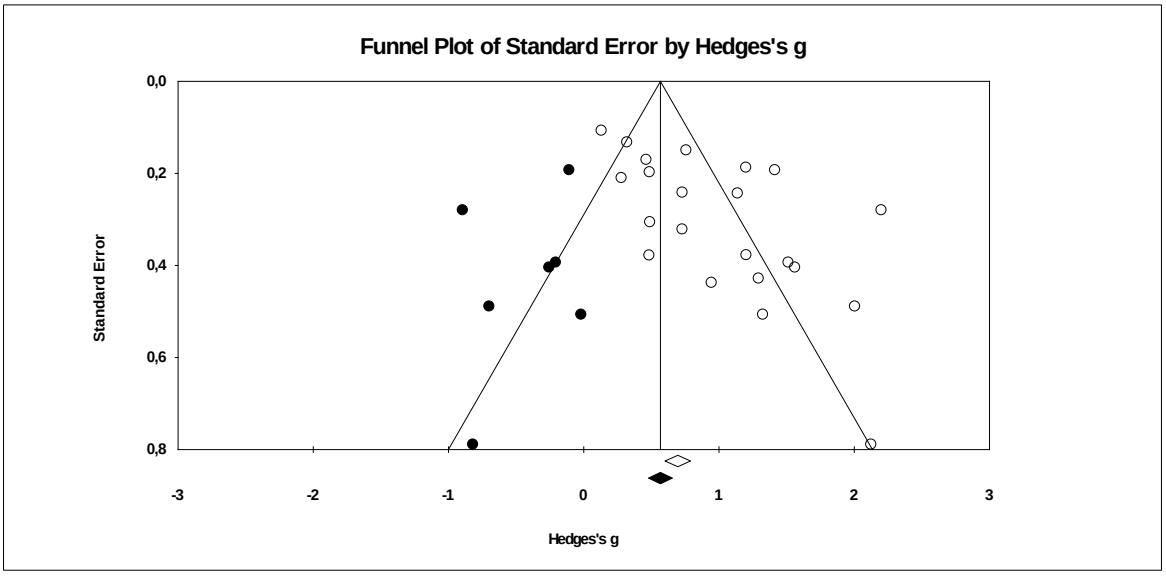

Figure B.2. Funnel plot of standard error by Hedges's *g* of publication bias within studies with (sub)clinical participants. Funnel plot of publication bias for the direct comparison of (sub)clinical OCD versus healthy controls. The white dots represent the included studies and the black dots represent imputed studies based on Duval and Tweedie's (2000) trim-and-fill procedure.

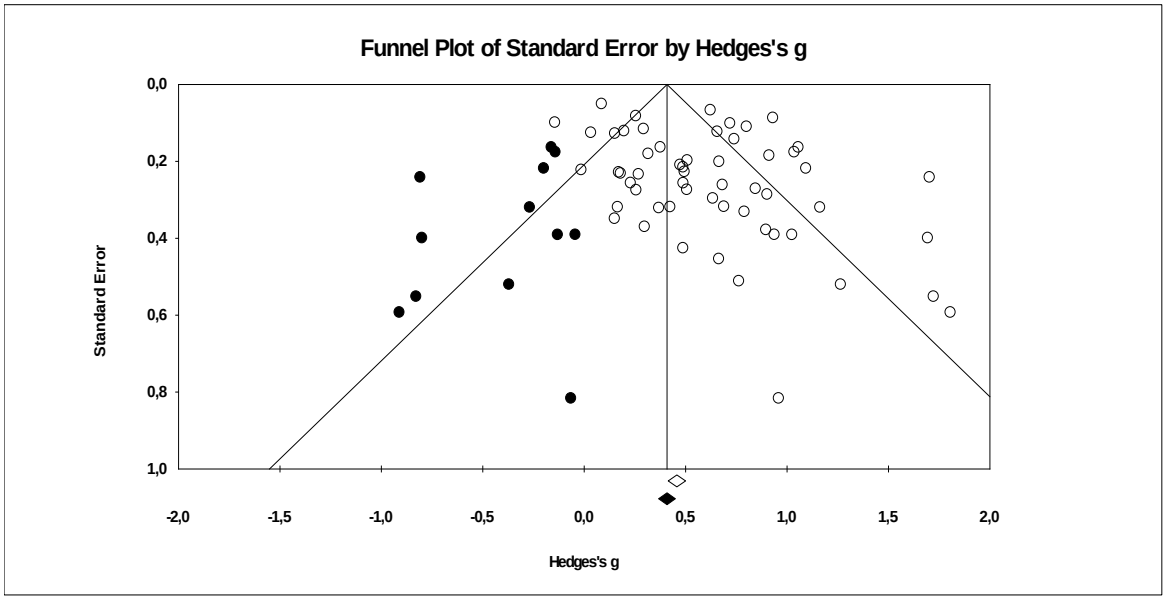

Figure B.3. Funnel plot of standard error by Hedges's *g* of publication bias within studies with healthy participants. Funnel plot of publication bias for the direct comparison of (sub)clinical OCD versus healthy controls. The white dots represent the included studies and the black dots represent imputed studies based on Duval and Tweedie's (2000) trim-and-fill procedure.

CHAPTER 3

DO OBSESSIVE-COMPULSIVE SYMPTOMS AND DISORDER-RELEVANT STIMULI AFFECT INHIBITION CAPACITY?[1]

ABSTRACT

The current study set out to investigate trait versus state views regarding inhibitory deficits in obsessive-compulsive disorder (OCD). Furthermore, it was investigated whether inhibitory deficits could be specific for OCD-relevant stimuli. Participants were selected on high (*n* = 40) vs. low (*n* = 44) contamination fear and subsequently randomly assigned to receive either a neutral induction or an OCD symptom induction. Participants performed a stop-signal task including contamination-specific, general negative, and neutral pictures before and after the induction. In contrast to state views, no change in inhibitory performance after the OCD symptom induction and no differential effect of disorder-specific picture valence was found. Although the absence of a change in inhibitory performance supports the endophenotype view, other predictions of this model were not confirmed. More specifically, baseline inhibition capacity did not predict an increase in symptoms after an OCD symptom induction. Moreover, contrary to expectations, participants high in contamination fear marginally outperformed low contamination fear controls. Therefore, the results of the current study are inconclusive regarding the state-trait debate, but are clearly in contrast with the idea of trait inhibitory deficits in contamination fear.

[1] Based on De Putter, L. M. S., Cromheeke, S., Anholt, G. E., Mueller, S. C., & Koster, E. H. W. (2017). *Do obsessive-compulsive symptoms and disorder-relevant stimuli affect inhibition capacity?* Manuscript submitted for publication.

Introduction

Obsessive-compulsive disorder (OCD) is a persistent and highly invalidating psychiatric disorder characterized by intrusive thoughts and/or compulsions (American Psychiatric Association, 2013). It is the fourth most common psychiatric disorder, with a lifetime prevalence of 2-3.5% and is characterized by high levels of individual suffering and also substantial economic and societal costs (Angst et al., 2004; Ruscio, Stein, Chiu, & Kessler, 2010). Despite the availability of many efficacious psychological and pharmacological treatments for OCD, many patients suffer from symptoms even after undergoing treatment (Fisher & Wells, 2005). In order to improve treatment, a better understanding of OCD is required.

There is a wealth of research on the etiological and maintaining factors of this disorder. Abnormal functioning of the frontostriatal circuit in OCD has been established as one of the main neural models for OCD. This neural circuit underlies executive functioning (Pauls, Abramovitch, Rauch, & Geller, 2014). Therefore, much of the research on the mechanisms of OCD has focused on the relation between executive functioning and OCD (for meta-analyses see Abramovitch, Abramowitz, & Mittelman, 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2014). Given the repetitive nature of obsessions and compulsions, response inhibition is of specific interest in OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). Response inhibition refers to the ability to inhibit a prepotent motor response (Logan, 1994).

There are distinct views on the nature of these deficits. Chamberlain et al. (2005) suggested response inhibition to be an endophenotype of OCD, which thus would be a sign of increased genetic risk for developing OCD. This implies that a deficit in inhibition is state independent (Gottesman & Gould, 2003). Thus, factors such as the valence of stimuli and current OCD symptoms should not affect inhibition capacity. Studies that support the endophenotype (trait) view show underperformance in inhibition both in OCD patients and their healthy relatives (Menzies et al., 2007), similar underperformance in OCD patients in remission, and similar underperformance in OCD patients pre- compared to post-treatment (Bannon, Gonsalvez, Croft, & Boyce, 2006). In contrast, Abramovitch and Cooperman (2015) argue that the current empirical evidence challenges this assumption. For instance, although some studies do not find differences

in neuropsychological performance after treatment, other research has shown improvement in neuropsychological performance following successful treatment (e.g., Andrés et al., 2008; Kuelz et al., 2006; Voderholzer et al., 2013). Moreover, some studies find an association between neuropsychological functioning and OCD symptom severity (e.g., Abramovitch, Dar, Schweiger, & Hermesh, 2011; Trivedi et al., 2008), although the results are mixed (see Kuelz, Hohagen, & Voderholzer, 2004). However, the lack of a clear association between neuropsychological functioning and OCD severity could be due to methodological shortcomings (Abramovitch & Cooperman, 2015).

As an alternative to the endophenotype (trait) view, Abramovitch, Dar, Hermesh, and Schweiger (2012) introduced the executive overload model of OCD. In this state model the overflow of symptoms in OCD, which is associated with hyperactivity of the frontostriatal system, is caused by continuous attempts of OCD patients to control automatic processes. This subsequently leads to an overload on the executive system that causes neuropsychological impairments. The manifestations of these cognitive impairments can subsequently activate "fear of impulsivity" or the feeling that one is not in control. In order to compensate, patients exert increased control over automatic processes, which results in a vicious cycle. This state model implies that an OCD symptom induction in the lab could overload the executive system, which should subsequently lead to an underperformance in inhibition tasks.

To date, few studies took such context dependent effects of current OCD symptoms and valence-specific stimuli into account. Indeed, some research that has taken into account the valence-specificity of stimuli has found that disorder-relevant stimuli influence inhibition capacity (Harkin & Kessler, 2012; Linkovski, Kalanthroff, Henik, & Anholt, 2016). Furthermore, currently most research contributing to the state-trait debate has been of correlational nature. Therefore it is not possible to establish the direction of the influence of inhibition on OCD (Abramovitch & Cooperman, 2015).

The current study tested the differential hypothesis of trait versus state models of inhibitory control in OCD in the context of disorder-relevant stimuli. We investigated whether a deficit in inhibition would be specific for a symptomatic state by assessing inhibition before and after an OCD symptom induction. According to the trait view this manipulation should have little effect on inhibitory control whereas state-related views

predict changes in line with state manipulations. Furthermore, we examined whether a deficit inhibition is specific for disorder-relevant stimuli. This was investigated by using negative, OCD-relevant, and neutral pictures in a SST. Finally, if inhibition capacity is indeed an endophenotype, we expected that baseline capacity to inhibit disorder-relevant stimuli would predict the magnitude of the increase of symptoms after an OCD symptom induction.

In the current study we focused on the contamination subtype of OCD, as contamination fear is relatively easy to induce in the laboratory (Rachman, 2004). Contamination fear is one of the most common subtypes of OCD (Ball, Baer, & Otto, 1996) and consists of fears of being contaminated or spreading contamination (Markarian et al., 2010). One of the methods that is used to elicit contamination fear symptoms in the lab is mental contamination (De Putter, Van Yper, & Koster, 2017). Mental contamination consists of a sense of internal dirtiness and is often characterized by a moral element (Rachman, 2004). Mental contamination is often evoked by the non-consensual kiss paradigm, in which participants imagine that someone tries to kiss them without their consent (e.g., Elliott & Radomsky, 2012). In order to test the effect of a contamination fear induction on inhibition, we chose to select at-risk participants scoring high on contamination fear (HCF) and participants scoring low on contamination fear (LCF) for two reasons. First, since response inhibition has been suggested as an endophenotype of OCD (Chamberlain et al., 2005) we would expect to observe decreased inhibition capacity in at-risk participants. Second, the utility of analogue samples in the research on the mechanisms underlying OCD has already been demonstrated elegantly by Gibbs (1996) and Abramowitz et al. (2014).

Method

Participants

According to an a priori power analysis based on a medium effect size (f = 0.25), with α = 0.05 and a power of 0.9, we needed a minimum of 64 participants in total. In total 91 healthy females ranging in age from 17 to 34 years (M = 19.29, SD = 2.07) participated. Undergraduate students of Ghent University interested in participating in experiments could subscribe to the website http://www.screeningpsychologie.be/, where they filled out the contamination subscale of the Padua Inventory revised online

(PI-R; Van Oppen, Hoekstra, & Emmelkamp, 1995). Participants were invited to the laboratory when they scored 2 or lower for the LCF group and 13 or higher for the HCF group. Thirteen is the average score of an OCD patient on the PI-R washing subscale and thus is a representative score for an analogue sample (Van Oppen et al., 1995). Furthermore, this is in line with the cut-off for HCF used in previous research (e.g., Deacon & Maack, 2008). Since symptoms can fluctuate over time and we were interested in those participants that had stable OCD symptoms, these criteria were checked again with the PI-R washing subscale at the beginning of the experiment as the pre-selection could have taken place two months before the actual experiment. Whenever the score of a participant in the HFC group was lower than 9 (mean plus 1SD of the score in a healthy control population) the participant was excluded. Similarly, participants of the LCF group were excluded if they scored higher than 4, the mean for the PI-washing subscale for the healthy control population (Van Oppen et al., 1995). This resulted in 44 participants in the LCF group and 40 participants in the HCF fear group. The study was approved by the ethical committee at Ghent University. Informed consent was obtained from all individual participants included in the study. Participants were either paid 20 euro or received course credit for their contribution.

Measures

Impulsiveness–Venturesomeness–Empathy questionnaire (I7). Since impulsivity can have an effect on inhibition, group differences in impulsivity were checked with the Impulsiveness subscale of the I7 (Eysenck, Pearson, Easting, & Allsopp, 1985; Lijffijt, Caci, & Kenemans, 2005). The impulsiveness subscale of the I7 consists of 19 dichotomous (yes/no) items.

Mood and Anxiety Symptoms Questionnaire (MASQ-D30). Since depression levels can have an effect on cognitive functioning (McDermott & Ebmeier, 2009), the anhedonic depression scale of the short adaptation of the MASQ (Wardenaar et al., 2010; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) was used to check for group differences in levels of depression. The anhedonic depression scale of the MASQ-D30 consists of 10 items on a scale rated from 1 (not at all) to 5 (very much).

Padua Inventory-revised (PI-R). The PI-R (Van Oppen et al., 1995) was used in order to assess OCD symptoms. The PI-R consists of five subscales: impulses, washing,

checking, rumination and precision. The 41 items are rated on a scale from 0 (never/not at all) to 4 (very often).

Mental Contamination Report (MCR). The MCR as designed by Radomsky, Elliott, Rachman, Fairbrother, and Newth (2008) was administered after the induction as a manipulation check of the OCD symptom induction. This version is a modification of the mental contamination report as used by previous studies (Fairbrother, Newth, & Rachman, 2005; Herba & Rachman, 2007). It consists of 21 items assessing internal negative emotions (i.e., how participants feel about themselves), external negative emotions (i.e., how participants feel about themselves and/or the man in the scenario), feelings of dirtiness, urge to wash, ease to imagine the scenario, desirability of the kiss, the man's morality before and after the kiss, and whether participants experienced a previous non-consensual sexual encounter. All ratings use a scale from 0 (not at all) to 100 (completely).

Visual Analogue Scales (VAS). As another manipulation check seven VAS were adopted from the Profile of Mood States (McNair, Lorr, & Dropplemann, 1992) in line with Rossi and Pourtois (2012). Positive mood was estimated using the mean of the scales "energetic", "satisfied", and "happy". Negative mood was estimated using the mean of the scales "angry", "tense", "depressed", and "disgusted", a scale added because of the relevance of disgust for contamination OCD (Broderick, Grisham, & Weidemann, 2013).

Dimensional Obsessive-Compulsive Scale (DOCS). Three items of the contamination subscale of the DOCS (Abramowitz et al., 2010) were adapted in order to measure momentary symptoms after the induction. The adapted questions were: "How much time have you spent during the experiment on thinking about contamination?", "How much time have you spent during the experiment on washing or cleaning behaviors because of contamination?", and "How difficult was it for you during the experiment to disregard thoughts about contamination and refrain from behaviors such as washing, showering, cleaning and other decontamination routines when you tried to do so?". These items were rated on a scale from 0 (none at all/not at all difficult) to 4 (most of the time/extremely difficult).

Hand washing. As a manipulation check of the induction we included washing behavior as an analogue of compulsive behavior for the contamination subtype of OCD.

We asked all participants at the end of the experiment to wash their hands using hand sanitizer. The time spent on washing hands was measured with a stopwatch in seconds.

Materials

Stop-Signal Task (SST). In order to assess inhibition capacity in the context of viewing contamination-related stimuli, the adapted SST (Logan, 1994) of Verbruggen and De Houwer (2007) was used. This task ran using Presentation® software (version 17.2, Neurobehavioral Systems). In this task participants were presented with a fixation cross for 500ms (70 x 100 pixels) followed by a picture for 500ms (384 x 288 pixels) and subsequently the target ("#" or "@", 100 x 100 pixels). Participants were instructed to respond as quickly as possible to the target with key "D" to the "#" and key "K" to the "@" on an AZERTY keyboard. This mapping rule was reversed for half of the participants. A response was required within 1250ms. The intertrial interval was set at 1500ms. A clearly audible stop-signal (75ms) was presented on 30% of the trials through headphones. In this case participants were required to inhibit their response. The stop-signal delay (SSD) was initially set at 250ms and continuously adjusted using a separate staircase tracking procedure (Levitt, 1971) to attain a probability of stopping of 50%. More specifically, whenever participants successfully inhibited their response, the SSD was increased by 25ms and whenever participants responded after a stop-signal, the SSD was decreased with 25ms. Note that the longer the SSD, the more difficult it is to inhibit a response.

The task started with a practice phase of 30 trials in which participants received immediate feedback on their performance. The experimental phase consisted of eight blocks of 60 trials in which participants received feedback on their performance on the end of every block (accuracy, mean reaction time, and mean probability of stopping).

For this study the pictures were neutral, negative or contamination-related. We presented 160 trials per picture type and 48 stop trials per picture type. Every picture was presented four times during the SST. In total 40 neutral (e.g., a leaf) and 40 negative (e.g., a gun) pictures were selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997). The 40 contamination-related pictures (e.g., a dirty toilet) were selected from the IAPS, the Maudsley Obsessive-Compulsive Stimuli Set (Mataix-Cols, Lawrence, Wooderson, Speckens, & Phillips, 2009), the picture

set of Morein-Zamir et al. (2013) and publically available online sources. In order to match negative and contamination-related pictures on arousal, these pictures were rated by an independent sample (*n* = 28) on arousal, and how much fear and disgust the pictures elicited on a Likert scale ranging from 1 (none) to 9 (very much). Furthermore, they rated the valence of the pictures on a Likert scale ranging from 1 (negative) to 9 (positive)[2].

The Stop-Signal Reaction Times (SSRTs) were estimated using the integration method[3]. The integration method assumes that the point at which the stop process finishes is equal to the n^{th} reaction time of the distribution of the trials in which there was no stop-signal. The n^{th} reaction time is equal to the point in the distribution at which the integral equals the probability of responding after a stop-signal. The SSRT can then be calculated by subtracting the SSD from the finishing time (Verbruggen, Chambers, & Logan, 2013). In this study the split-half reliability of the SST was satisfactory (first SST r_{sb} = .85; second SST r_{sb} = .91).

OCD symptom induction. The Non-Consensual Kiss (NCK) task, as used by Elliott and Radomsky (2012), was selected for an OCD symptom induction. This induction was selected since a meta-analysis on induction procedures of OCD symptoms (De Putter et al., 2017) revealed that mental contamination, and specifically the NCK task, was one of the strongest inductions that also elicited symptoms in healthy participants. The audio script of the NCK task was the same as the script of the non-consensual physically dirty condition of Elliott and Radomsky (2012). In this induction participants listen to a scenario that describes a party and at the end of the party they are kissed non-consensual by a physically dirty man. The audio script for the neutral induction was based on the consensual physically clean condition of Elliott and Radomsky (2012). In order to make the script more neutral, the consensual kiss on the mouth was substituted with a kiss on the cheek as a means of saying goodbye, which is a common

[2] *M* arousal OCD pictures = 4.17, *SD* arousal OCD pictures = 0.94, *M* arousal negative pictures = 4.90, *SD* arousal negative pictures = 0.73; *M* fear OCD pictures = 2.56, *SD* fear OCD pictures = 0.91, *M* fear negative pictures = 4.29, *SD* fear negative pictures = 1.38; *M* disgust OCD pictures = 4.51, *SD* disgust OCD pictures = 1.44, *M* disgust negative pictures = 3.01, *SD* disgust negative pictures = 1.06; *M* valence OCD pictures = 3.63, *SD* valence OCD pictures = 0.60, *M* valence negative pictures = 3.01, *SD* valence negative pictures = 0.63

[3] For every participant the assumption of the horse race model was examined by checking if the signal respond RT was faster than the no-signal RT. Sensitivity analyses showed that all results were still robust if participants violating this assumption were excluded.

informal way of saying goodbye in Belgium. The audio recordings were administered through headphones and participants were instructed to imagine being the woman described in the scenario and that the events were happening at that moment in time.

Reminder Induction. During the second SST there was a short break between every two blocks (three breaks in total) in which participants rated their current disgust level, right before and after being asked to focus on the scenario again on the moment they received a kiss. This was done in order to ensure that the induction would remain active throughout the second SST.

Procedure

See Figure 1 for an overview of the procedure. After reading and signing the informed consent, participants filled out the I_7, MASQ, and PI-R. Subsequently participants performed the first SST. After the SST participants filled out the VAS scales. Subsequently, subclinical and healthy participants were randomly allocated to either the neutral mood induction or the OCD symptom induction. Following the induction participants filled out the VAS scales again, the MCR, and the DOCS. Afterwards, participants performed the second SST, during which they were reminded of the induction every two blocks and rated their disgust levels. Finally, participants were asked to wash their hands using hand sanitizer and the time they spent on washing their hands was recorded in seconds using a stopwatch. At the end of the study the participants were fully debriefed.

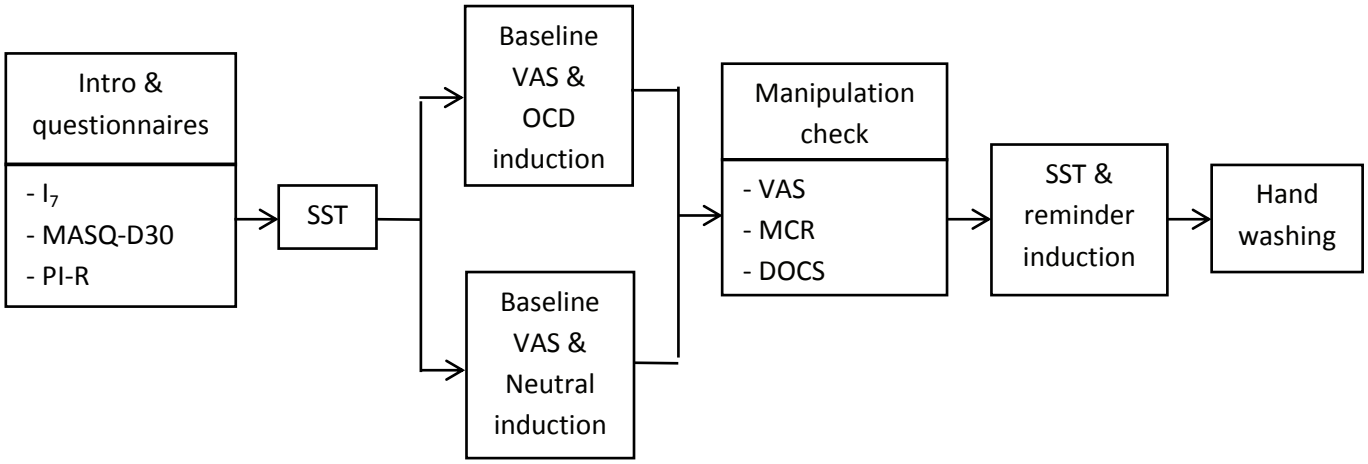

Figure 1. Overview of the procedure. I_7 = Impulsiveness–Venturesomeness–Empathy questionnaire, MASQ-D30 = Mood and Anxiety Symptoms Questionnaire, PI-R = Padua Inventory Revised, SST = Stop Signal Task, VAS = Visual Analogue Scale, MCR = Mental Contamination Report, DOCS = Dimensional Obsessive Compulsive Scale.

Statistical Analysis

Statistics were performed using SPSS (version 20; IBM Corp, 2011) and a significance level of 0.05 was used. Effect sizes are reported in the form of partial eta-squared (η_p^2). For outlier analysis, since the integration method already excludes outlier reaction times by selecting a specific point within the distribution of the reaction times, we only checked whether any participants had consistent scores higher than 3 standard deviations from the other participants. This resulted in the exclusion of one participant from the HCF group.

Differences between groups or inductions in age, impulsiveness, MASQ depression, ease to imagine the induction scenario, PI total scores, scores on the washing subscale of the PI, baseline positive and negative mood were analyzed using separate one way ANOVA's. Potential differences between groups or inductions in experienced previous non-consensual sexual encounters were analyzed using Fisher's exact test, since a difference in the experience of a previous non-consensual sexual encounter could influence the effectiveness of the induction.

As the effectiveness of the induction was crucial to our design, we investigated this with multiple measures such as the MCR, VAS negative and positive mood, DOCS, and time spent on washing hands. For the MCR, in line with Elliott and Radomsky

(2012), we performed separate ANOVA's on perceived kiss desirability and the difference score of pre- and post-physical dirtiness of the man as dependent variables and group and induction as independent variables. A multivariate ANOVA was conducted in order to assess the effects of the induction on feelings of mental contamination (i.e., feelings of dirtiness, urges to wash, internal negative emotions, and external negative emotions) as the dependent variables and group and induction as the independent variables. Furthermore, in order to assess the effect of the manipulation on positive and negative mood separate mixed ANOVA's with Time (pre- and post-induction) as a within-subject factor and group and inductions as between-subject factors was performed. Moreover, in order to assess the effect of the manipulation on the DOCS an ANOVA was performed on the DOCS scores with group and induction as the independent variables. Finally, in order to assess the effect of the manipulation on hand washing, as an analogue for compulsive behavior, an ANOVA was performed on the time spent on hand washing with group and induction as the independent variables.

The effectiveness of the reminder of the induction during the SST was assessed with a mixed ANOVA on the disgust VAS scales administered before and after the reminder with Time (pre-post induction) and Reminder (3 reminders in total) as within-subject factors and group and induction as between-subject factors.

In order to investigate the hypothesis that disorder-relevance and current OCD symptoms would have an effect on inhibition a mixed ANOVA was performed on the SSRTs with Time (pre- and post-induction) and Valence (negative, neutral, contamination-related) as within-subject factors and group and induction as between-subject factors.

Finally, in order to test whether baseline SSRTs would be able to predict an increase in symptoms after the induction separate linear regressions were performed per OCD symptoms measure after the induction (i.e., feelings of dirtiness, urge to wash, hand washing, internal negative emotions, external negative emotions, DOCS, VAS negative, and VAS positive) with baseline SSRT for OCD-relevant, negative and neutral pictures as independent variables. For the analysis of VAS positive and VAS negative we corrected for baseline VAS positive and negative scores. As we only expected an increase in symptoms after the OCD induction, we excluded participants that had received the neutral mood induction (n = 40) from these analyses.

Results

Sample Characteristics

See Table 1 for the means and standard deviations of the sample characteristics. Age, impulsiveness, MASQ depression, baseline positive mood, and ease to imagine the scenario were not significantly different between groups (HCF or LCF), inductions (OCD induction or neutral induction) or Group x Induction (all *F*'s(1,79) < 3.47, all *p*'s > .05). Moreover, in this sample 31% experienced a previous non-consensual sexual encounter, but this did not differ per group ($\chi^2(1) = .01$, $p = .92$), or induction ($\chi^2(1) = .06$, $p = .80$). Importantly, in line with the pre-selection, there was a significant difference between groups for PI-R washing ($F(1,79) = 327.72$, $p < .001$, $\eta_p^2 = .81$) and the PI total score ($F(1,79) = 117.44$, $p < .001$, $\eta_p^2 = .60$). Furthermore, there was a significant difference between groups for baseline negative mood $F(1,79) = 9.12$, $p = .003$, $\eta_p^2 = .10$), which was to be expected comparing subclinical to healthy participants.

Table 1.

Means and standard deviations on demographic and baseline ratings for each condition

	HCF/OCD induction (*n* = 20)		HCF/neutral induction (*n* = 19)		LCF/OCD induction (*n* = 23)		LCF/neutral induction (*n* = 21)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age	19.15[a]	2.06	18.63[a]	1.38	19.22[a]	2.75	19.00[a]	1.90
Impulsiveness	6.50[a]	3.98	3.79[a]	2.55	7.22[a]	4.91	6.29[a]	5.52
MASQ AD	27.70[a]	6.38	30.42[a]	9.00	26.78[a]	8.71	25.90[a]	8.29
Baseline pos	42.62[a]	21.46	34.77[a]	21.20	46.26[a]	14.36	42.21[a]	16.47
Baseline neg	27.73[a]	15.17	28.71[a]	19.08	16.77[b]	10.36	20.99[b]	10.75
Ease to imagine	64.00[a]	21.75	73.60[a]	10.10	70.41[a]	20.76	71.19[a]	13.44
PI-R washing	16.90[a]	5.90	18.95[a]	6.60	0.74[b]	1.14	0.48[b]	0.93
PI-R total	66.80[a]	21.38	66.00[a]	19.49	30.35[b]	13.13	22.62[b]	11.87

Note. HCF = high contamination fear group, LCF = low contamination fear group, MASQ AD = Mood and Anxiety Symptom Questionnaire anhedonic depression, baseline pos = baseline positive mood, baseline neg = baseline negative mood, PI-R = Padua Inventory-revised. For each row, variables that share the same subscript are not significantly different from each other ($p > .05$)

Manipulation Checks

In order to check whether the manipulation was successful we analyzed scores from the MCR, VAS negative and positive mood, DOCS, and hand washing as shown in Tables 2a en 2b. As expected, the MCR revealed significant differences between inductions: participants in the OCD induction reported more mental contamination, a larger difference between pre- and post-physical dirtiness of the man and less kiss desirability than participants in the neutral induction. Moreover, the VAS for positive and negative mood showed significant interaction effects between Time x Induction. Follow-up independent samples *t*-tests revealed that there was no difference between inductions before the induction (negative mood: $t(81) = 0.87$, $p = .39$; positive mood: $t(81) = 1.46$, $p = .15$), while there was a significant difference between the inductions after the induction (negative mood: $t(81) = 5.02$, $p < .001$, Cohen's $d = 1.10$; positive mood: $t(81) = 3.33$, $p = .001$, Cohen's $d = 0.73$). As expected, after the induction participants in the OCD induction reported more negative mood and less positive mood than participants in the neutral induction. Furthermore, contrary to our prediction, there were no significant effects of induction on the DOCS or time spent on hand washing at the end of the experiment. To conclude, participants reported more mental contamination and a change in their mood in the OCD symptom induction, while participants did not differ from the neutral induction on the DOCS or their time spent on washing their hands.

Moreover, these analyses showed some interesting group effects. Participants in the HCF group reported higher scores on the DOCS, more negative mood, less positive mood, and more feelings of mental contamination than participants in the LCF group. This finding is in line with the association between the contamination fear subtype and mental contamination (Rachman, 2004). Finally, there was a small significant interaction effect between Group x Induction for time spent on washing hands. Follow-up independent *t*-tests revealed that this interaction effect is due to the lack of a difference between inductions in the HCF group ($t(37) = 0.83$, $p = .41$), whereas the inductions differed significantly in the LCF group ($t(42) = 2.22$, $p = .03$, Cohen's $d = 0.67$). In the LCF group participants that received the OCD induction spent more time washing their hands than participants that received the neutral induction.

Table 2a.

Results manipulation check

Variables	Main effect of Induction				Main effect of Group				Induction x Group interaction			
	F	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2
Mental Contamination Report												
Perceived kiss desirability[a]	22.82	1, 79	**< .001**	.22	0.80	1, 79	.374	.01	0.17	1, 79	.680	< .01
Difference pre- and post-physical dirtiness of the man[a]	44.81	1, 79	**< .001**	.36	0.71	1, 79	.403	.01	0.54	1, 79	.467	.01
Feelings of Mental Contamination[b]	26.42	4, 76	**< .001**	.58	5.70	4, 76	**< .001**	.23	1.39	4, 76	.245	.07
VAS												
Negative Mood[c]	7.99	1, 79	**.006**	.09	10.65	1, 79	**.002**	.12	0.21	1, 79	.645	< .01
Positive Mood[c]	1.29	1, 79	.259	.02	4.32	1, 79	**.041**	.05	0.47	1, 79	.494	< .01
Other measures												
DOCS[a]	1.74	1, 79	.191	.02	24.71	1, 79	**< .001**	.24	1.44	1, 79	.234	.02
Time hand washing[a]	0.38	1, 79	.539	< .01	2.73	1, 79	.103	.03	4.00	1, 79	**.049**	.05

Note: DOCS = Dimensional Obsessive-Compulsive Scale. [a] Represents results of 2 (Induction) x 2 (Group) ANOVA's; [b] Represents results of a 2 (Induction) x 2 (Group) MANOVA with feelings of dirtiness, urges to wash, internal negative emotions, and external negative emotions as dependent variables representing feelings of mental contamination; [c] Represents results of 2 (Induction) x 2 (Group) x 2 (Time) Mixed ANOVA's.

Table 2b.

Results manipulation check continued

Variables	Main effect Time				Time x Induction interaction				Time x Group interaction				Time x Induction x Group interaction			
	F	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2
VAS																
Negative Mood[c]	0.04	1, 79	.837	< .01	47.10	1, 79	**< .001**	.37	0.13	1, 79	.720	< .01	0.02	1, 79	.899	< .01
Positive Mood[c]	0.18	1, 79	.672	< .01	43.99	1, 79	**< .001**	.36	2.75	1, 79	.101	.03	0.26	1, 79	.613	< .01

Note: [c] Represents results of 2 (Induction) x 2 (Group) x 2 (Time) Mixed ANOVA's.

Manipulation Check Reminder Induction

In order to assess the effect of the reminder of the induction during the second SST, a mixed ANOVA was performed on the disgust VAS scales administered before and after the reminder with Time (pre-post induction) and Reminder (3 reminders in total) as within-subject factors and group and induction as between-subject factors. This revealed a significant Reminder x Time interaction effect ($F(2,78) = 6.63$, $p = .003$, $\eta_p^2 = .14$) and a significant Time x Induction interaction effect ($F(1,79) = 47.56$, $p < .001$, $\eta_p^2 = .38$). Follow-up paired samples *t*-tests comparing reminder at the different time points for the increase pre-post induction at every reminder showed that the Reminder x Time interaction effect was due to a significant difference in the increase in disgust between the first reminder and the second reminder ($t(82) = 3.38$, $p = .001$, Cohen's $d = 0.18$), between the first reminder and the third reminder ($t(82) = 3.12$, $p = .002$, Cohen's $d = 0.20$), but not between the second and third reminder ($t(82) = 0.45$, $p = .65$). The difference between pre- and post-scores was larger after the first reminder ($M_{diff} = 17.42$, $SD_{diff} = 27.17$) than after the second (M_{diff}

= 12.64, SD_{diff} = 24.33) and third reminder (M_{diff} = 12.06, SD_{diff} = 24.59), indicating a habituation of the reminder of the induction. Furthermore, follow-up independent samples *t*-test showed that the Time x Induction effect was due to the absence of a difference between inductions before the reminder ($t(81) = 1.19$, $p = .24$), while the difference between inductions was significant after the reminder ($t(81) = 4.52$, $p < .001$, Cohen's $d = 0.99$). After the reminder participants in the OCD induction reported more disgust ($M = 49.83$, $SD = 25.76$) than participants in the neutral induction ($M = 26.08$, $SD = 21.73$), indicating that the reminder of the induction was successful.

Effects of Disorder-Relevance and Current OCD Symptoms on Inhibition

In order to reduce the positive skew of the SSRT distribution over participants the SSRT were transformed using a square root transformation. The mixed ANOVA on the transformed SSRT with Time (pre- and post-induction) and Valence (negative, neutral, and contamination-related) as within-subject factors and group and induction as between-subject factors revealed a significant main effect of Valence ($F(2,78) = 4.69$, $p = .01$, $\eta_p^2 = .11$). Follow-up paired *t*-test showed that there was no significant difference between contamination-related and negative pictures ($t(82) = 1.15$, $p = .25$) or contamination-related and neutral pictures ($t(82) = 1.60$, $p = .11$), but there was a significant difference between negative and neutral pictures ($t(82) = 2.95$, $p = .004$, Cohen's $d = 0.21$). Participants were faster after negative pictures (M = 208ms, SD = 39ms) than after neutral pictures (M = 217ms, SD = 46ms). Moreover, there was a significant main effect of Time ($F(1,79) = 4.62$, $p = .03$, $\eta_p^2 = .06$) in which participants were faster in the second SST (M = 208ms, SD = 39ms) than the first (M = 216ms, SD = 46ms). Furthermore, there was a significant main effect of Group ($F(1,79) = 4.60$, $p = .04$, $\eta_p^2 = .06$) in which participants in the HCF group were faster (M = 204ms, SD = 38ms) than participants in the LCF group (M = 220ms, SD = 38ms). There was also a main effect of induction ($F(1,79) = 5.32$, $p = .02$, $\eta_p^2 = .06$) in which participants receiving the OCD induction were faster (M = 203ms, SD = 32ms) than participants in the neutral induction (M = 222ms, SD = 43ms). As this effect did not interact with Time, this indicates a coincidental preexisting difference in SSRTs between inductions. The other predicted interaction effects were also not significant (F's < 1.84, p's > .16). Based on the current data, there was no effect of an OCD symptom induction on SSRTs and

disorder-relevant picture valence did not affect the HCF group and LCF group differently.

Predicting Symptoms based on Baseline Inhibition Capacity

The linear regressions did not reveal any significant effects (all *p*'s > .11). Baseline SSRTs after any type of picture were not able to predict the increase in symptoms after the OCD symptom induction.

Discussion

This study set out to test differential hypotheses of trait versus state models of inhibitory control in OCD. Moreover, we investigated whether underperformance in inhibitory control would be specific for OCD-related stimuli. State-related views such as the executive overload model of OCD (Abramovitch et al., 2012) predict changes in inhibition capacity after state manipulations of OCD symptoms, whereas the endophenotype (trait) view predicts little effect of such a manipulation. Moreover, as inhibition capacity would be a marker for vulnerability to develop OCD, the endophenotype view implies that baseline capacity to inhibit contamination-related stimuli would predict the magnitude of an increase of symptoms after an OCD symptom induction. Surprisingly, the current results failed to support either a trait or a state view on inhibitory deficits in OCD given the absence of baseline OCD-related inhibitory deficits as well as the absence of state influences on such deficits. We discuss these findings in more detail below.

First of all, the manipulation checks showed that for most outcome measures the induction proved successful in inducing OCD symptoms. The induction successfully elicited feelings of mental contamination and a change in general positive and negative mood. However there was no generalization of the induction effect to time spent on washing hands as an analogue of compulsive behavior or to an adapted version of the DOCS in order to measure current OCD symptoms. This suggests that although the induction was potent enough to induce feelings of mental contamination, which is strongly related to the contamination fear subtype of OCD (Rachman, 2004), it did not generalize to behavior and intrusive thoughts. However, it should be noted that the adapted DOCS used in this study after the induction enquired after symptoms

experienced during the experiment in general. In hindsight, this manner of enquiry may have been too broad. Indeed, a recent study using the same OCD symptom induction in which the adapted DOCS specifically enquired after symptoms experienced during induction found that participants receiving an OCD symptom induction reported more intrusive thoughts compared to participants receiving a neutral mood induction (De Putter & Koster, 2017). Moreover, the manipulation check of the reminder of the induction during the second inhibition task showed that reminder of the induction was successful in maintaining the effects of the induction. These findings are crucial as they imply that, according to the state view, one could expect interference effects of the induction during the second inhibition task.

According to the state view of Abramovitch et al. (2012) we had expected a change in inhibitory functioning after the OCD symptom induction and a differential effect of contamination-related, negative and neutral picture valence. In contrast, results showed that the induction had no effect on subsequent performance on inhibition and there was no effect of contamination-related picture valence. Here, in contrast to Verbruggen and De Houwer (2007), participants displayed faster SSRTs following negative pictures compared to neutral pictures. According to the trait endophenotype view we had expected differences between the subclinical HCF and control LCF group at baseline, no change in inhibitory functioning after an OCD symptom induction, and the ability of baseline inhibition to predict an increase in symptoms after an OCD symptom induction. Although there was indeed no change in inhibitory functioning after the induction, baseline performance on inhibition was not a significant predictor of an increase in symptoms after the OCD symptom induction. Moreover, the significant difference between the HCF and the LCF group was in the opposite direction than predicted by the endophenotype view. The HCF group actually performed better on inhibition than the LCF group. The endophenotype (trait) view regards underperformance in inhibition as a sign of increased genetic risk for developing OCD (Chamberlain et al., 2005). Therefore this finding is in contrast with the endophenotype view and meta-analyses showing a deficit in inhibition in OCD (e.g., Abramovitch et al., 2013; Snyder et al., 2014). However, this finding could be due to the choice of the subtype of OCD. Indeed, a meta-analysis on differences in neuropsychological performance between subtypes showed that the contamination

subtype generally outperforms the checking subtype with especially large effect sizes for response inhibition (Leopold & Backenstrass, 2015). Current evidence of differential performance in response inhibition according to subtype stems from studies using Stroop and go/no go tasks. The current study suggests that this effect may generalize to the SST in subclinical participants of the contamination subtype and that they may even outperform comparison participants low on contamination fear. Although further examination of the specificity of underperformance in response inhibition according to subtype with multiple subtypes included is necessary in order to confirm this. Importantly, although this effect was characterized by a medium effect size, the significant difference between groups should be interpreted with caution as the *p*-value (i.e., $p = .04$) only just fell below the threshold of significance. Moreover, the average difference was only 16 milliseconds which is unlikely to have any clinical significance. In conclusion, the current results are in contrast with the trait endophenotype view, but do not provide support for the state view either.

There are several limitations to the current study. First, this study used a subclinical population instead of a clinical OCD population. Yet, the utility of analogue samples in research on OCD has already been shown by Gibbs (1996) and Abramowitz et al. (2014). Moreover, as inhibition was suggested as an endophenotype of OCD we had expected decreased inhibition in at-risk participants. However, there might be protective factors at play preventing these participants to progress to a clinical level. For instance, intact inhibition capacity could be one of these protecting factors. Second, it is possible that the contamination-related pictures presented during the SST could also have served as an induction of state OCD symptoms. However, in that event we would have expected a strong effect of contamination-related picture valence, which we did not observe. Third, although the choice of the OCD symptom induction was based on its effectiveness in evoking OCD symptoms (De Putter et al., 2017), the inhibition task was independent of the nature of the induction. If the induction would have been relevant for the inhibition task, as is the case in real life for OCD patients, the results might have been different. Similarly, Linkovski et al. (2016) found that repeated checking only affected inhibition for previously checked stimuli. Relatedly, the OCD-related pictures used in the SSTs were selected based on their relevance for the contamination subtype in general. However, even within subtypes OCD is characterized

by substantial heterogeneity in what triggers their symptoms (Rufer, Grothusen, Maß, Peter, & Hand, 2005). Future research investigating the state-trait debate with an OCD symptom induction and disorder-relevant stimuli should therefore include idiosyncratic material and an induction that is more relevant for the subsequent information processing task.

Limitations notwithstanding, this study was one of the first studies investigating the differential hypotheses of the state-trait debate and taking valence-specificity into account with an experimental design. In conclusion, there is mixed evidence for the endophenotype view in which the absence of an effect of an OCD symptom induction or OCD-related picture valence is in line with this view, whereas baseline inhibition capacity not predicting any increase in symptoms after an OCD symptom induction is in contrast with this view. Moreover, the group difference between HCF and LCF was in the opposite direction than predicted by the endophenotype view. Based on the current data no evidence was found for state models such as the executive overload model (Abramovitch et al., 2012) as we did not find any difference in performance on inhibition after an OCD symptom induction or according to preceding OCD-related picture valence. Therefore, the results of this study are inconclusive regarding the state-trait debate, but are clearly in contrast with the idea of stable inhibitory deficits in contamination fear.

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References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163-1171. doi: 10.1016/j.cpr.2013.09.004

Abramovitch, A., & Cooperman, A. (2015). The cognitive neuropsychology of obsessive-compulsive disorder: A critical review. *Journal of Obsessive-Compulsive and Related Disorders, 5*, 24-36. doi: 10.1016/j.jocrd.2015.01.002

Abramovitch, A., Dar, R., Hermesh, H., & Schweiger, A. (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD. *Journal of Neuropsychology, 6*(2), 161-191. doi: 10.1111/j.1748-6653.2011.02021.x

Abramovitch, A., Dar, R., Schweiger, A., & Hermesh, H. (2011). Neuropsychological impairments and their association with obsessive-compulsive symptom severity in obsessive-compulsive disorder. *Archives of Clinical Neuropsychology*. doi: 10.1093/arclin/acr022

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., . . . Hale, L. R. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the dimensional obsessive-compulsive scale. *Psychological Assessment, 22*(1), 180-198. doi: 10.1037/a0018260

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Andrés, S., Lázaro, L., Salamero, M., Boget, T., Penadés, R., & Castro-Fornieles, J. (2008). Changes in cognitive dysfunction in children and adolescents with obsessive-compulsive disorder after treatment. *Journal of Psychiatric Research, 42*(6), 507-514. doi: 10.1016/j.jpsychires.2007.04.004

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Ball, S. G., Baer, L., & Otto, M. W. (1996). Symptom subtypes of obsessive-compulsive disorder in behavioral treatment studies: A quantitative review. *Behaviour Research and Therapy, 34*(1), 47-51. doi: 10.1016/0005-7967(95)00047-2

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive–compulsive disorder: State or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11-12), 1031-1038. doi: 10.1080/j.1440-1614.2006.01928.x

Broderick, J., Grisham, J. R., & Weidemann, G. (2013). Disgust and fear responding in contamination-based obsessive-compulsive disorder during pictorial exposure. *Behavior Therapy, 44*(1), 27-38. doi: 10.1016/j.beth.2012.05.005

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience & Biobehavioral Reviews, 29*(3), 399-419. doi: 10.1016/j.neubiorev.2004.11.006

De Putter, L. M. S., Van Yper, L., & Koster, E. H. W. (2017). Obsessions and compulsions in the lab: A meta-analysis of procedures to induce symptoms of obsessive-compulsive disorder. *Clinical Psychology Review, 52*, 137-147. doi: 10.1016/j.cpr.2017.01.001

De Putter, L. M. S., & Koster, E. H. W. (2017). *The effects of obsessive-compulsive symptoms and disorder-relevant stimuli on the dynamics of selective attention.* Manuscript in preparation.

Deacon, B., & Maack, D. J. (2008). The effects of safety behaviors on the fear of contamination: An experimental investigation. *Behaviour Research and Therapy, 46*(4), 537-547. doi: 10.1016/j.brat.2008.01.010

Elliott, C. M., & Radomsky, A. S. (2012). Mental contamination: The effects of imagined physical dirt and immoral behaviour. *Behaviour Research and Therapy, 50*(6), 422-427. doi: 10.1016/j.brat.2012.03.007

Eysenck, S. B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*(5), 613-619. doi: 10.1016/0191-8869(85)90011-x

Fairbrother, N., Newth, S. J., & Rachman, S. (2005). Mental pollution: Feelings of dirtiness without physical contact. *Behaviour Research and Therapy, 43*(1), 121-130. doi: 10.1016/j.brat.2003.12.005

Fisher, P. L., & Wells, A. (2005). How effective are cognitive and behavioral treatments for obsessive–compulsive disorder? A clinical significance analysis. *Behaviour Research and Therapy, 43*(12), 1543-1558. doi: 10.1016/j.brat.2004.11.007

Gibbs, N. A. (1996). Nonclinical populations in research on obsessive-compulsive disorder: A critical review. *Clinical Psychology Review, 16*(8), 729-773. doi: 10.1016/S0272-7358(96)00043-8

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160*(4), 636-645. doi: 10.1176/appi.ajp.160.4.636

Harkin, B., & Kessler, K. (2012). Deficient inhibition of return in subclinical ocd only when attention is directed to the threatening aspects of a stimulus. *Depression and Anxiety, 29*(9), 807-815. doi: 10.1002/da.21911

Herba, J. K., & Rachman, S. (2007). Vulnerability to mental contamination. *Behaviour Research and Therapy, 45*(11), 2804-2812. doi: 10.1016/j.brat.2007.07.010

IBM Corp. (2011). IBM SPSS statistics for windows, version 20.0. Armonk, NY: IBM Corp.

Kuelz, A. K., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: A critical review. *Biological Psychology, 65*(3), 185-236. doi: 10.1016/j.biopsycho.2003.07.007

Kuelz, A. K., Riemann, D., Halsband, U., Vielhaber, K., Unterrainer, J., Kordon, A., & Voderholzer, U. (2006). Neuropsychological impairment in obsessive-compulsive disorder—improvement over the course of cognitive behavioral treatment. *Journal of Clinical and Experimental Neuropsychology, 28*(8), 1273-1287. doi: 10.1080/13803390500507246

Lang, P. J., Bradley, M. M., & Cuthbert, B. (1997). *International affective picture system (IAPS): Instruction manual and affective ratings.* University of Florida, NIMH Center for the Study of Emotion and Attention, Gainesville, FL, USA.

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of Anxiety Disorders, 30*, 48-58. doi: 10.1016/j.janxdis.2014.12.016

Levitt, H. (1971). Transformed up-down methods in psychoacoustics. *The Journal of the Acoustical Society of America, 49*(2B), 467-477. doi: 10.1121/1.1912375

Lijffijt, M., Caci, H., & Kenemans, J. L. (2005). Validation of the Dutch translation of the I_7 questionnaire. *Personality and Individual Differences, 38*(5), 1123-1133. doi: 10.1016/j.paid.2004.07.010

Linkovski, O., Kalanthroff, E., Henik, A., & Anholt, G. E. (2016). Stop checking: Repeated checking and its effects on response inhibition and doubt. *Journal of Behavior Therapy and Experimental Psychiatry, 53*, 84-91. doi: 10.1016/j.jbtep.2014.12.007

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory and language*. San Diego, CA: Academic Press.

Markarian, Y., Larson, M. J., Aldea, M. A., Baldwin, S. A., Good, D., Berkeljon, A., . . . McKay, D. (2010). Multiple pathways to functional impairment in obsessive-compulsive disorder. *Clinical Psychology Review, 30*(1), 78-88. doi: 10.1016/j.cpr.2009.09.005

Mataix-Cols, D., Lawrence, N. S., Wooderson, S., Speckens, A., & Phillips, M. L. (2009). The Maudsley Obsessive–Compulsive Stimuli Set: Validation of a standardized paradigm for symptom-specific provocation in obsessive–compulsive disorder. *Psychiatry Research, 168*(3), 238-241. doi: 10.1016/j.psychres.2008.05.007

McDermott, L. M., & Ebmeier, K. P. (2009). A meta-analysis of depression severity and cognitive function. *Journal of Affective Disorders, 119*(1–3), 1-8. doi: 10.1016/j.jad.2009.04.022

McNair, D. M., Lorr, M., & Dropplemann, L. F. (1992). *Manual: Profile of mood states revised*. San Diego, CA: Educational and Industrial Testing Service.

Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C.-H., del Campo, N., . . . Bullmore, E. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*(12), 3223-3236. doi: 10.1093/brain/awm205

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Pauls, D. L., Abramovitch, A., Rauch, S. L., & Geller, D. A. (2014). Obsessive-compulsive disorder: An integrative genetic and neurobiological perspective. *Nature Reviews. Neuroscience, 15*(6), 410-424. doi: 10.1038/nrn3746

Presentation®, version 17.2, Neurobehavioral Systems, Inc., Berkeley, CA, www.neurobs.com

Rachman, S. (2004). Fear of contamination. *Behaviour Research and Therapy, 42*(11), 1227-1255. doi: 10.1016/j.brat.2003.10.009

Radomsky, A. S., Elliott, C. M., Rachman, S., Fairbrother, N., & Newth, S. J. (2008). *Mental Contamination Report*. Unpublished questionnaire, Concordia University, Montreal, QC.

Rossi, V., & Pourtois, G. (2012). Transient state-dependent fluctuations in anxiety measured using STAI, POMS, PANAS or VAS: A comparative review. *Anxiety, Stress, & Coping, 25*(6), 603-645. doi: 10.1080/10615806.2011.582948

Rufer, M., Grothusen, A., Maß, R., Peter, H., & Hand, I. (2005). Temporal stability of symptom dimensions in adult patients with obsessive–compulsive disorder. *Journal of Affective Disorders, 88*(1), 99-102. doi: 10.1016/j.jad.2005.06.003

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/mp.2008.94

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: A meta-analysis. *Psychological Medicine, 44*(6), 1121-1130. doi: 10.1017/S0033291713001803

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2014). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science*. doi: 10.1177/2167702614534210

Trivedi, J. K., Dhyani, M., Goel, D., Sharma, S., Singh, A. P., Sinha, P. K., & Tandon, R. A. (2008). Neurocognitive dysfunction in patients with obsessive compulsive disorder. *African Journal of Psychiatry, 11*(3), 204-209.

Van Oppen, P., Hoekstra, R. J., & Emmelkamp, P. M. G. (1995). The structure of obsessive-compulsive symptoms. *Behaviour Research and Therapy, 33*(1), 15-23. doi: 10.1016/0005-7967(94)E0010-G

Verbruggen, F., Chambers, C. D., & Logan, G. D. (2013). Fictitious inhibitory differences: How skewness and slowing distort the estimation of stopping latencies. *Psychological Science, 24*(3), 352-362. doi: 10.1177/0956797612457390

Verbruggen, F., & De Houwer, J. (2007). Do emotional stimuli interfere with response inhibition? Evidence from the stop signal paradigm. *Cognition and Emotion, 21*(2), 391-403. doi: 10.1080/02699930600625081

Voderholzer, U., Schwartz, C., Freyer, T., Zurowski, B., Thiel, N., Herbst, N., . . . Kuelz, A. K. (2013). Cognitive functioning in medication-free obsessive-compulsive patients treated with cognitive-behavioural therapy. *Journal of Obsessive-Compulsive and Related Disorders, 2*(3), 241-248. doi: 10.1016/j.jocrd.2013.03.003

Wardenaar, K. J., van Veen, T., Giltay, E. J., de Beurs, E., Penninx, B. W. J. H., & Zitman, F. G. (2010). Development and validation of a 30-item short adaptation of the Mood and Anxiety Symptoms Questionnaire (MASQ). *Psychiatry Research, 179*(1), 101-106. doi: 10.1016/j.psychres.2009.03.005

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*(1), 15–25. doi: 10.1037/0021-843X.104.1.15

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3-14. doi: 10.1037/0021-843X.104.1.3

CHAPTER 4

THE EFFECTS OF OBSESSIVE-COMPULSIVE SYMPTOMS AND DISORDER-RELEVANT STIMULI ON THE DYNAMICS OF SELECTIVE ATTENTION[1]

ABSTRACT

Two studies were conducted in order to examine the link between selective attention and trait and state OCD symptoms. Selective attention was both considered as a dynamic process in time by investigating attentional bias scores at trial level and as a stable concept by investigating traditional attentional bias scores. In the first study we investigated the difference in selective attention between a group scoring high (*n* = 32) and a group scoring low (*n* = 32) on contamination fear at a cross-sectional level. In the second study we administered a dot probe task before and after an experimental manipulation of OCD symptoms (*n* = 35) or a neutral induction (*n* = 33) in order to determine the effects of state OCD symptoms on selective attention. In the current studies we found no evidence for either a trait-related presence of attention bias nor for influences of experimentally induced contamination fear. Furthermore, baseline selective attention did not predict symptoms after an OCD symptom induction. These results point to either a more complex relationship between OCD and selective attention than an unidirectional relationship or suggest that selective attention may not be as important for obsessive-compulsive disorders as it is for anxiety disorders.

[1] Based on De Putter, L. M. S., & Koster, E. H. W. (2017). *The effects of obsessive-compulsive symptoms and disorder-relevant stimuli on the dynamics of selective attention.* Manuscript submitted for publication.

Introduction

Obsessive-Compulsive Disorder (OCD) is a persisting and severe disorder which consists of recurrent intrusive thoughts and/or compulsions (American Psychiatric Association, 2013). It's lifetime prevalence is 2-3.5 %, making it the fourth most common mental disorder (Angst et al., 2004; Ruscio, Stein, Chiu, & Kessler, 2010). Some cognitive models have proposed that attentional bias to threat is one of the mechanisms contributing to the development and maintenance of OCD (e.g., Bar-Haim et al., 2007; Muller & Roberts, 2005). Attentional bias refers to the tendency to selectively attend to threatening stimuli over nonthreatening stimuli. For instance, the integrative model of Bar-Haim et al. (2007) is a model of threat processing comprising four stages: preattentively evaluating stimuli in the environment; allocating cognitive resources to threat stimuli; comparing threat with memory, assessing context of threat and available coping resources; and interrupting current goals and orienting attention towards threat. This model was based on a meta-analysis in which the results for OCD were not significantly different from anxiety disorders, which suggests that the integrative models applies to OCD.

Other models have suggested a mutually reinforcing relation between attentional bias towards threat and anxiety. For instance, Eysenck, Derakshan, Santos, and Calvo (2007) proposed the attentional control theory (ACT). This model poses that attentional control is governed by bottom-up capture and top-down control (Corbetta & Shulman, 2002). Bottom-up capture is activated by threat stimuli that can be internal (such as intrusive thoughts) and external stimuli (such as pictures of threatening stimuli) whereas top-down control is goal-oriented and enables to focus on the task at hand. Applied to OCD, ACT implies increased bottom-up capture in the context of obsessive thoughts and threatening external stimuli. Since resources in working memory are limited, increased bottom-up capture would result in decreased top-down control, causing decreased efficiency (e.g., slower reaction times) in the performance on cognitive tasks. Therefore, ACT predicts that an induction of OCD symptoms would enhance bottom-up capture and thus attentional bias towards threat. Indeed, Cohen, Lachenmeyer, and Springer (2003) found a significant deterioration on performance on a non-emotional Stroop Task after an OCD symptom induction. The view of a mutual reinforcing relation between attentional bias towards threat and anxiety was further

corroborated by Van Bockstaele et al. (2014), where their review demonstrated that a unidirectional cause-effect model between attentional bias to threat and anxiety is unlikely.

Some research has indeed shown an attentional bias for OCD-related stimuli in subclinical or clinical OCD (e.g., Amir, Najmi, & Morrison, 2009; Lavy, Van Oppen, & Van Den Hout, 1994; Moritz, Von Muehlenen, Randjbar, Fricke, & Jelinek, 2009; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996). However, other studies failed to find an attentional bias for OCD (e.g., Harkness, Harris, Jones, & Vaccaro, 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008). Moreover, Summerfeldt and Endler (1998) argued in their review that, in contrast to anxiety disorders, attentional bias in OCD has only been demonstrated in OCD with contamination concerns. In contrast, Bar-Haim et al. (2007) found no significant difference between OCD and anxiety disorders in attentional bias in their meta-analysis.

The inconsistencies in the current literature could be due to two major limitations. First, the current research on attentional bias to threat in the context of OCD has often viewed attentional bias as a stable bias. However, Rodebaugh et al. (2016) argued that one of the reasons for the unreliability of most of the measures capturing attentional bias could be that attentional bias is not a stable trait, but rather a dynamic process. Moreover, recently a novel way to express attentional variability has been developed in order to capture attentional bias as a dynamic process in time (Zvielli, Bernstein, & Koster, 2015). These scores are calculated by repeatedly estimating trial-level attentional bias by subtracting temporally contiguous incongruent-congruent trial pairs at the level of trials instead of at the level of the entire task. In line with the view of attentional bias as a dynamic process, Bradley et al. (2016) found no evidence of OCD symptoms predicting vigilance or delayed disengagement, but OCD symptoms did predict the tendency to repeatedly re-orient and fixate upon OCD stimuli over time as measured with eyetracking. As there is very little research on the variability of attentional bias in the context of OCD, the current studies considered attentional bias not only with the traditional bias scores as a stable concept, but also with the new trial-level bias scores (TL-BS) approach considering attentional bias as a dynamic process.

Secondly, from the current research it is still unclear whether attentional bias has an influence on OCD symptoms or whether state OCD symptoms can also influence

attentional bias. For instance, a study that more explicitly examined the nature of the relationship between attentional bias and OCD showed that an experimental reduction of attentional bias resulted in increased behavioral approach towards contamination stimuli in subclinical contamination fear participants, suggesting a link between attentional bias and behavioral avoidance in contamination fear (Najmi & Amir, 2010). However, it is noteworthy that there is limited research using prospective designs to examine whether attentional bias influences the presence and expression of OCD symptoms.

Furthermore, in a meta-analysis Pergamin-Hight, Naim, Bakermans-Kranenburg, van Ijzendoorn, and Bar-Haim (2015) showed that attentional bias was specific for disorder-congruent stimuli in anxiety disorders. However, only four studies on OCD were included. Therefore, further research on the specificity of attentional bias in OCD is warranted.

In order to further elucidate the link between attentional bias specifically to OCD-related stimuli and OCD symptoms two studies were conducted. In the first study we investigated the relationship between trait OCD and attentional bias for OCD-related stimuli using a cross-sectional design, whereas in the second study we tested whether attentional bias for OCD-related stimuli is influenced by state OCD-related concerns. Moreover, in the second study we checked whether attentional bias for OCD-related stimuli at baseline can predict an increase in symptoms after an OCD symptom induction, which we would expect if attentional bias would contribute to OCD symptoms.

Study 1

The first study set out to examine the relationship between attentional bias towards contamination stimuli on the one hand and on the other hand subclinical OCD participants scoring high (HCF) on the cleaning subscale of the Maudsley Obsessional-Compulsive Inventory (Hodgson & Rachman, 1977) versus participants scoring low on contamination fear (LCF). Contamination fear consists of the fear of being contaminated or contaminating someone else and is one of the most common symptom dimensions of OCD (Ball, Baer, & Otto, 1996; Markarian et al., 2010). As attentional bias to threat has been put forward a mechanism to develop OCD symptoms, we expected to observe

an attentional bias towards contamination-related stimuli in HCF. This study used a subclinical sample as the meta-analysis of Bar-Haim et al. (2007) did not show a difference between clinical patients and participants with high self-reported anxiety in attentional bias. Furthermore, the utility of analogue samples in research on the mechanisms of OCD has been demonstrated previously by Gibbs (1996) and Abramowitz et al. (2014).

In this study selective attention was measured using a dot probe task including, contamination-related, neutral and positive (i.e., representing cleanliness) pictures. Previous research on selective attention to OCD-related and positive words in OCD found no effect of positive words (Lavy et al., 1994). However, Moritz et al. (2008) argued that words may not be strong enough to elicit an attentional bias.

Method

Participants

According to a power analysis based on d = 0.38 (the effect size found for between-group comparisons of threat-related bias in the dot probe task; Bar-Haim et al., 2007), with $\alpha = 0.05$ and a power of 0.8, we needed a minimum of 60 participants in total. The total sample included 64 participants with ages ranging from 17 to 51 years (M = 19.42, SD = 5.16; 50 females). Undergraduate students of Ghent University interested in participating could subscribe to the website http://www.screeningpsychologie.be/, where they filled out the cleaning subscale of the Maudsley Obsessive-Compulsive Inventory online (MOCI; Hodgson & Rachman, 1977). Participants were invited to the laboratory when they scored 5 or higher on the cleaning subscale (i.e., the mean of OCD patients on the cleaning subscale; Hodgson & Rachman, 1977) for the HCF group and when they scored 0 on the cleaning subscale for the LCF group. This resulted in 32 participants in the LCF group and 32 participants in the HCF fear group. The study was approved by the ethical committee at Ghent University. Informed consent was obtained from all individual participants included in the study. Participants received course credit for their contribution.

Measures and Materials

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID). In order to check diagnostic status the OCD-section of the Dutch version of the SCID was used (First, Spitzer, Gibbon, & Williams, 1998). The SCID is a widely used semi-structured clinical interview developed to asses psychopathology according to the DSM-IV.

Maudsley Obsessive-Compulsive Inventory (MOCI). The cleaning subscale of the MOCI (Hodgson & Rachman, 1977) was used in order to preselect participants on contamination fear. This subscale consists of several statements regarding cleanliness (e.g., "My hands do not feel dirty after touching money"). Participants responded by selecting "true" or "false". The MOCI has good psychometric properties (Hodgson & Rachman, 1977).

Padua Inventory-revised (PI-R). In order to assess OCD symptoms the PI-R (Van Oppen, Hoekstra, & Emmelkamp, 1995) was used. The subscales of the PI-R assess impulses, washing, checking, rumination, and precision. Participants rated the 41 items on a Likert scale form 0 (never/not at all) to 4 (very often). The PI-R has good psychometric properties (Van Oppen et al., 1995).

Obsessive Compulsive Inventory-revised (OCI-R). In addition to the PI-R, the OCI-R (Foa et al., 2002; Foa, Kozak, Salkovskis, Coles, & Amir, 1998) was used to assess OCD symptoms. The OCI-R consists of six subscales: washing, checking, ordering, obsessing, hoarding, and neutralizing. The 18 items were rated on a Likert scale from 0 (not at all) to 4 (extremely). The OCI-R has good psychometric properties (Hajcak, Huppert, Simons, & Foa, 2004).

Dimensional Obsessive-Compulsive Scale (DOCS). An adaptation of the contamination subscale of the DOCS (Abramowitz et al., 2010) was used in order to compare momentary OCD symptoms during the experiment between the LCF and the HCF group. Participants rated the items on a Likert scale from 0 (none at all/not at all difficult) to 4 (most of the time/extremely difficult). The three adapted questions were: "How much time have you spent during the experiment on washing or cleaning behaviors because of feelings of contamination?", "How difficult was it for you during the experiment to disregard thoughts about contamination and refrain from behaviors such as washing, showering, cleaning and other decontamination routines when you try

to do so?", and "How much time have you spent during the experiment on thinking about contamination?".

Disgust Scale-Revised (DS-R). As disgust sensitivity is elevated in the contamination fear symptom dimension of OCD (Broderick, Grisham, & Weidemann, 2013), the DS-R (Haidt, McCauley, & Rozin, 1994; Olatunji et al., 2009; Olatunji et al., 2007) was used to assess disgust sensitivity. The DS-R consists of three subscales: core disgust, animal reminder disgust, and contamination disgust. The 25 items were rated on a Likert scale from 0 (completely disagree/not disgusting at all) to 4 (completely agree/very disgusting). The DS-R has good psychometric properties (Olatunji et al., 2009; Olatunji et al., 2007).

Mood scales For ethical reasons, Visual Analogue Scales (VAS) assessing mood were administered before and after the dot probe task in order to ensure that participants were not negatively impacted by the experiment. This was done by three VAS scales assessing happiness, sadness, and anxiety on a scale from "neutral" to "as happy/sad/anxious as I can imagine". At the end of the experiment momentary experience of disgust was assessed by asking how much disgust they experienced on a Likert scale from 1 (not at all) to 9 (very much). In order to cancel out any negative impact from the experiment a short movie was shown as a means of a positive mood induction when these mood scales showed a large decrease in positive mood or increase in negative mood and anxiety. As these scales were only used for ethical reasons we did not include these data in the analyses.

Dot probe task. In order to assess selective attention the dot probe task (MacLeod, Mathews, & Tata, 1986) was used. The dot probe was programmed using Inquisit Millisecond 3 software (2011). The dot probe task consisted of three trial types: contamination-related vs. neutral, safety vs. neutral and neutral vs. neutral. There were 64 trials per trial type resulting in a total of experimental 192 trials. These trials were preceded by 12 practice trials in which participants received feedback on their performance. Half of the contamination-related vs. neutral and safety vs. neutral trials were congruent, in which the dot appeared at the location previously occupied by the contamination-related or safety picture. The other half of the trials were incongruent, in which the target appeared at the location previously occupied by the neutral picture. The task was programmed so that each picture category was presented equally often in

each location and that each picture within the picture category was presented equally often. The order of the trial types was randomized for each participant.

All stimuli were presented against a white background. A trial started with a black fixation cross presented in the middle of the screen. After 500 ms two pictures (384 x 288 pixels) appeared above and below the fixation cross for 500 ms. Subsequently the pictures were erased and a black dot appeared at the same location as one of the previous pictures. The dot remained on the screen until the participant responded with a press on the "Q" key when the dot was above the fixation cross and a press on the "M" key when the dot was below the fixation cross on an AZERTY keyboard.

A total of 64 neutral (e.g., a bus), 16 contamination-relevant (e.g., a dirty toilet) and 16 pictures representing safety (e.g., a bottle of soap) were selected for this task. The neutral pictures were selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997). The contamination-related pictures were selected from the IAPS, the Maudsley Obsessive-Compulsive Stimuli Set (Mataix-Cols, Lawrence, Wooderson, Speckens, & Phillips, 2009), the picture set of Vogt, Lozo, Koster, and De Houwer (2011), and publically available online sources. The safety pictures were selected from the cleanliness picture set of Vogt et al. (2011) and publically available online sources.

Procedure

At the start of the experiment participants read and signed the informed consent form. Afterwards participants were interviewed with the OCD-section of the SCID. Subsequently, participants filled out the PI-R, DS-R, OCI-R, and the VAS scales. After the questionnaires the dot probe task was administered. Finally, participants filled out the mood scales, adapted DOCS and, if necessary, they received a positive mood induction with a short movie. At the end of the study participants received a full debriefing about the experiment. An overview of the procedure is depicted in Fig. 1.

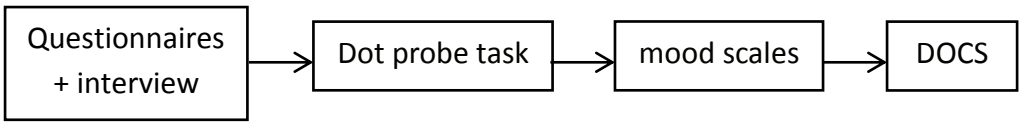

Figure 1. Overview of the procedure of study 1.

Statistical Analysis

SPSS (version 20; IBM Corp, 2011) was used in order to perform statistics with the significance level set at $p < .05$. Partial eta-squared (η_p^2) was used for effect sizes. Continuous sample characteristics such as age, state OCD symptoms experienced during the experiment, DS-R, washing subscales, and total scores of the PI-R and OCI-R were analyzed with separate *t*-tests. Subsequently, the difference between groups in gender was analyzed using Fisher's exact test.

As a first step in the dot probe data preparation, in line with previous research (e.g., Zvielli, Bernstein, & Koster, 2014), all trials with errors and reaction times (RT) faster than 200 and slower than 1500 ms were removed (2.16%). Generally accuracy was high (*M* = 97.98%, *SD* = 1.70%, range = 92% - 100%). Subsequently, all RTs deviating more than three standard deviations from the participant's individual mean per trialtype (i.e., safety congruent, safety incongruent, threat incongruent, threat congruent, and neutral) and from the sample mean RT per trialtype were removed (3.50%). Finally, attentional bias for threat was calculated by subtracting mean threat-congruent trials from mean threat-incongruent trials and attentional bias for safety by likewise subtracting mean safety-congruent trials from mean safety-incongruent trials. Positive attentional bias scores refer to attentional bias towards threat/safety and negative attentional bias scores refer to attentional avoidance away from threat/safety. Attentional interference for threat was calculated by subtracting mean neutral trials from mean threat-incongruent trials and attentional interference for safety by likewise subtracting mean neutral trials from mean safety-incongruent trials. Attentional interference scores above zero refer to interference by threat/safety pictures. Attentional bias variability[2] for threat and safety was calculated using the computation

[2] As other TL-BS measures (i.e., mean attentional bias towards or away) correlated highly with attentional bias variability (all *r*'s > .81, all *p*'s < .001), we did not repeat analyses with these other TL-BS measures in order to avoid an inflation of type I error.

code of Zvielli and Bernstein (2016) as used in Zvielli et al. (2014). This code subtracts RTs between temporally contiguous matched trials (incongruent vs. congruent) so that attentional bias can be estimated at trial-level.

Group differences were investigated using *t*-tests and Fisher's exact test when applicable. In order to test the main hypothesis of this study that there is a difference between HCF and LCF in selective attention towards threat and safety, separate mixed ANOVA's were performed on the different indices of selective attention for threat and safety (i.e., attentional bias, attentional interference, and attentional bias variability) with Valence (threat or safety) as a within-subject factor and Group as a between-subject factor.

Results

Sample Characteristics

Age did not differ between groups ($t(62) = 0.07$, $p = .943$). There were significant more women in the HCF group ($n = 29$), than in the LCF group ($n = 21$; $\chi^2(1) = 5.85$, $p = .032$). Moreover, there was no difference between experienced state OCD symptoms during the experiment ($t(62) = 1.63$, $p = .109$). Importantly, in line with the pre-selection, there were significant differences between groups in the DS-R, washing subscales, and total scores of the OCI-R and PI-R (all t's > 3.86, all p's < .001), in which the HCF group scored higher than the LCF group (for means see Table 1). Of the HCF group the SCID identified six participants with clinical levels of OCD, while no participants were identified with OCD in the LCF group.

Table 1.

Means and standard deviations on demographic for HCF and LCF from study 1

	HCF (*n* = 32)		LCF (*n* = 32)	
	M	*SD*	*M*	*SD*
Age	19.38[a]	4.43	19.47[a]	5.87
DS-R	62.69[a]	14.32	45.44[b]	14.21
OCI-R washing subscale	4.56[a]	3.04	0.75[b]	1.57
OCI-R total	24.88[a]	11.84	14.75[b]	8.97
PI-R washing subscale	15.09[a]	8.51	4.00[b]	5.24
PI-R total	58.03[a]	22.18	33.63[b]	13.19
DOCS	2.63[a]	2.25	1.72[a]	2.20

Note. HCF = high contamination fear group, LCF = low contamination fear group, DS-R = Disgust Scale-Revised, OCI-R = Obsessive Compulsive Inventory-revised, PI-R = Padua Inventory-revised, DOCS = Dimensional Obsessive Compulsive Scale. For each row, variables that share the same subscript are not significantly different from each other ($p < .05$).

HCF versus LCF in Selective Attention to Threat and Safety

The results of the mixed ANOVA's are represented in Table 2. Contrary to predictions, for all measures of selective attention (i.e., attentional bias, attentional interference, and attentional bias variability) analyses revealed no significant interaction effect between Valence x Group or a main effect of Group. However, there was a significant main effect of Valence for every measure, in which participants generally showed more attentional bias and attentional interference for threat than for safety and higher attentional bias variability in the presence of threat-related pictures.

Table 2.

HCF versus LCF in selective attention to threat and safety

	Main effect of Valence				Main effect of Group				Valence x Group interaction			
Variables	*F*	*df*	*P*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2
Attentional Bias	18.33	1, 62	**< .001**	.23	0.92	1, 62	.340	.01	0.87	1, 62	.354	.01
Attentional Interference	50.61	1, 62	**< .001**	.45	0.02	1, 62	.878	< .01	0.07	1, 62	.786	< .01
Attentional Bias Variability	5.64	1, 62	**.021**	.08	3.66	1, 62	.060	.06	0.18	1, 62	.676	< .01

Note. Mixed ANOVA with Valence (threat or safety) as a within-subject factor and Group as a between-subject factor.

In order to test whether attentional bias or interference differed from zero (i.e., no attentional preference, interference or variability), one sample *t*-tests were performed. One sample *t*-tests showed that for safety attentional avoidance and variability were significantly different from zero (zero represents no bias). For threat all measures of selective attention differed significantly from zero (see Table 3). Participants generally showed attentional bias towards threat, attentional interference after threat, and attentional bias variability, whereas they showed a slight attentional avoidance from safety pictures.

Table 3.

One-samples t-tests from zero

	M	*SD*	*t*(63)	*p*
Attentional Bias Safety	-3.54	13.57	-2.09	**.041**
Attentional Bias Threat	7.20	16.68	3.45	**.001**
Attentional Interference Safety	-1.62	12.70	-1.02	.313
Attentional Interference Threat	15.25	18.40	6.63	**< .001**
Attentional Bias Variability Safety	78.66	20.67	30.45	**< .001**
Attentional Bias Variability Threat	85.29	25.15	27.13	**< .001**

Discussion

The first study set out to investigate selective attention towards contamination-related stimuli in a HCF and LCF group. Results indicated a general effect of attentional bias and interference towards threat, attentional avoidance from safety and attentional bias variability. However, contrary to predictions, this was not specific for HCF.

Study 2

Provided that we failed to observe trait influences of HCF we examined whether a state induction of contamination fear influenced selective attention. Moreover, we examined whether attentional bias at baseline influences the response to a contamination symptoms induction. The hypothesis, main methods, and analyses of this study have been preregistered at https://aspredicted.org/ (#1076).

The current study used a convenience sample, since previous research has shown that symptoms similar to OCD can effectively be induced in healthy participants (De Putter, Van Yper, & Koster, 2017). Moreover, Moritz et al. (2009) found that OCD patients did not rate OCD-related stimuli as more negative than healthy control subjects. Therefore, a convenience sample lends itself to investigate the effect of an OCD symptom induction on OCD-related stimuli. Furthermore, as contamination fear is best construed as dimensional rather than categorical (Mataix-Cols, do Rosario-Campos, & Leckman, 2005), it is likely at least some stimuli will elicit contamination fear in healthy participants. In order to make the stimuli more idiosyncratic, participants rated their anxiety following a range of contamination-related pictures. Only the pictures eliciting most anxiety were presented in the dot probe task. In the current study the dot probe task included contamination-related, neutral, and generally negative pictures. Including generally negative pictures allowed for investigating whether an effect would be specific for contamination-related stimuli or for negative stimuli in general.

Method

Participants

According to an a priori power analysis based on the effect size *d* = 0.38 (the effect size found for between-group comparisons of threat-related bias in the dot probe; Bar-Haim et al., 2007), with α = 0.05 and a power of 0.8, we needed a minimum of 60 participants in total. In line with our preregistration, we tested 70 healthy participants. All participants were female as our OCD symptom induction was specifically designed for women. Participants age ranged from 17 to 37 years (*M* = 22.56, *SD* = 3.26). Most participants were undergraduate students from Ghent University. The study was approved by the ethical committee at Ghent University. Informed consent was obtained from all individual participants included in the study. Participants received 10 euro for their participation.

Measures

PI-R. The PI-R as described in study 1 was used to assess OCD symptoms.

Impulsiveness–Venturesomeness–Empathy questionnaire (I_7). As attentional bias has previously been associated with impulsivity (e.g., Coskunpinar & Cyders, 2013;

Hou et al., 2011), the impulsiveness subscale of the I_7 (Eysenck, Pearson, Easting, & Allsopp, 1985; Lijffijt, Caci, & Kenemans, 2005) was used to check for any group differences in levels of impulsivity. This subscale consists 19 dichotomous (yes/no) items.

Mood and Anxiety Symptoms Questionnaire (MASQ-D30). As depression levels have also been associated with attentional bias (e.g., Koster, De Raedt, Goeleven, Franck, & Crombez, 2005), the anhedonic depression scale of the short adaptation of the MASQ (Wardenaar et al., 2010; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) was used in order to check for group differences in levels of depression. The 10 items of the anhedonic depression scale were rated on a Likert scale from 1 (not at all) to 5 (extremely).

Yale-Brown Obsessive–Compulsive Scale (Y-BOCS). In order to measure severity of any obsessive-compulsive symptoms the Y-BOCS severity self-report as designed by Baer (1991) was used. This Y-BOCS is very similar to the interview and has good psychometric properties (Steketee, Frost, & Bogart, 1996). The questionnaire included an explanation of what obsessions and compulsions entail. Participants indicated the time spent, interference, distress, resistance, and control over obsessions and compulsions separately on a scale from 0 (none) to 4 (extreme).

VAS. In line with Rossi and Pourtois (2012), seven VAS were adopted from the Profile of Mood States (McNair, Lorr, & Dropplemann, 1992) as a means of a manipulation check for neutral or OCD symptom induction. As in study 1, the scale "disgusted" was added because of its relevance to the contamination symptom dimension of OCD (Broderick et al., 2013). The mean of the scales "tense", "angry", "depressed", and "disgusted" was used to estimate negative mood. The mean of the scales "happy", "energetic", and "satisfied" was used to estimate positive mood. Finally, one scale was used to estimate fatigue. The VAS scales were administered before and after neutral or OCD symptom induction. The VAS scales were also administered at the end of the experiment in order to check participants' mood before leaving the experiment for ethical reasons. As preregistered, the data of the VAS scales at the end of the experiment were therefore not included in the data analyses.

Mental Contamination Report (MCR). As a means of a manipulation check, the MCR as developed by Radomsky, Elliott, Rachman, Fairbrother, and Newth (2008) was

administered after neutral or OCD symptom induction. Radomsky et al. (2008) modified this version from the mental contamination report as used by previous studies (Fairbrother, Newth, & Rachman, 2005; Herba & Rachman, 2007). The 21 items were rated on a scale from 0 (not at all) to 100 (completely). The MCR assessed internal negative emotions (e.g., guilt), external negative emotions (e.g., anger), feelings of dirtiness, urge to wash (e.g., face), ease to imagine the scenario, desirability of the kiss, and the man's morality before and after the kiss.

DOCS. The same adapted version of the DOCS as used in study 1 was used in study 2 as a means of a manipulation check after neutral or OCD symptom induction. All questions of the DOCS were phrased so that they specifically referred to how participants felt during the induction. The DOCS was also administered at the end of the experiment. As preregistered, only the data of the DOCS after neutral or OCD symptom induction were included in the analysis, as the measurement at the end of the experiment was solely meant to check participants' mood before leaving the experiment for ethical reasons.

Hand washing. Washing behavior was included as a means of a manipulation check of neutral or OCD symptom induction. We asked all participants to wash their hands using hand sanitizer at the end of the study in order to have a continuous measure of washing behavior. The time spent on washing hands was recorded, unbeknownst to the participants, using a stopwatch.

Materials

Dot probe task. The dot probe task ran using Inquisit Millisecond 4 software (2016). The dot probe task in study 2 was similar to the dot probe task in study 1 with a few adaptations. In this dot probe task the trial type safety vs. neutral was replaced with negative vs. neutral in order to assess any incremental selective attention of contamination-relevant stimuli above and beyond negative stimuli in general. Moreover, the number of experimental trials was increased to 240 trials in total, resulting in 80 trials per trial type. The dot probe task was administered before and after neutral or OCD symptom induction.

In total 60 neutral (e.g., a leaf) and 60 negative (e.g., a gun) pictures were selected from the International Affective Picture System (IAPS; Lang, Bradley, &

Cuthbert, 1997). The 60 contamination-relevant pictures (e.g., a dirty toilet) were selected from the IAPS, the Maudsley Obsessive-Compulsive Stimuli Set (Mataix-Cols, Lawrence, Wooderson, Speckens, & Phillips, 2009), the picture set of Morein-Zamir et al. (2013) and publically available online sources. An independent sample (*n* = 28) rated these pictures in order to match negative and disorder-relevant pictures on arousal and how much fear and disgust the pictures elicited on a Likert scale ranging from 1 (none) to 9 (very much). Moreover, they rated the valence of the pictures on a Likert scale ranging from 1 (negative) to 9 (positive)[3]. Forty neutral pictures were selected from the IAPS to form 20 neutral-neutral pairs. In order to enhance the relevance of the contamination-related pictures to the participants, participants rated all contamination-relevant pictures on how much fear these pictures elicited before the dot probe task. Only the 16 pictures most relevant to the participant were used in the dot probe task in order to mimic the idiosyncratic nature of OCD.

Non-Consensual Kiss (NCK) induction. Participants were randomly allocated to either a NCK induction or a neutral induction. The NCK induction was chosen based on a meta-analysis on induction procedures of OCD symptoms (De Putter et al., 2017). The NCK induction was one of the strongest inductions for OCD symptoms in healthy participants. The audio script for the NCK induction was translated into Dutch from the script of the non-consensual physically dirty condition of Elliott and Radomsky (2012). The induction consists of listening to a scenario through headphones that describes a party and at the end of the party participants imagine being kissed non-consensual by a physically dirty man. For the neutral induction the audio script of the consensual physically clean condition of Elliott and Radomsky (2012) was adjusted by substituting the consensual kiss on the mouth by a kiss on the cheek as a means of saying goodbye. A kiss on the cheek is a common informal way of saying goodbye to friends in Belgium. Before listening to the scenario participants were instructed to imagine being the

[3] *M* arousal OCD pictures = 4.17, *SD* arousal OCD pictures = 0.94, *M* arousal negative pictures = 4.90, *SD* arousal negative pictures = 0.73; *M* fear OCD pictures = 2.56, *SD* fear OCD pictures = 0.91, *M* fear negative pictures = 4.29, *SD* fear negative pictures = 1.38; *M* disgust OCD pictures = 4.51, *SD* disgust OCD pictures = 1.44, *M* disgust negative pictures = 3.01, *SD* disgust negative pictures = 1.06; *M* valence OCD pictures = 3.63, *SD* valence OCD pictures = 0.60, *M* valence negative pictures = 3.01, *SD* valence negative pictures = 0.63

woman described in the scenario as vividly as possible. The experimenters conducting the experiment were blind to the condition (NCK or neutral) participants were randomized to.

Reminder Induction. Participants were reminded of the induction in a short break after 120 trials in the second dot probe task. Participants rated their current disgust and anxiety level, right before and after being asked to focus on the scenario again on the moment they received a kiss. This was done in order to prevent that the effects of the OCD induction would subside during the duration of the second dot probe task.

Procedure

At the beginning of the experiment participants read and signed the informed consent form. After that, the PI-R, I_7 impulsiveness scale, MASQ, and Y-BOCS were administered. Subsequently participants performed the first dot probe task. After the first dot probe task participants filled out the VAS scales. Then participants were randomly assigned to either the OCD induction or the neutral induction. After the induction, participants filled out the manipulation check questionnaires (VAS scales, MCR, and DOCS). Subsequently participants performed the second dot probe task. During the second dot probe task there was a short break in the middle of the task in which participants rated their current disgust and anxiety level, right before and after being reminded of the induction. Afterwards, all participants were asked to wash their hands as a last manipulation check of the OCD induction. The hand washing was postponed to the end of the experiment in order to prevent it from cancelling out any effects of the OCD induction. Finally, participants filled out the last VAS scales and DOCS and if necessary received a positive mood induction by means of a short movie. All participants were fully debriefed at the end of the experiment. For an overview of the study see Fig. 2.

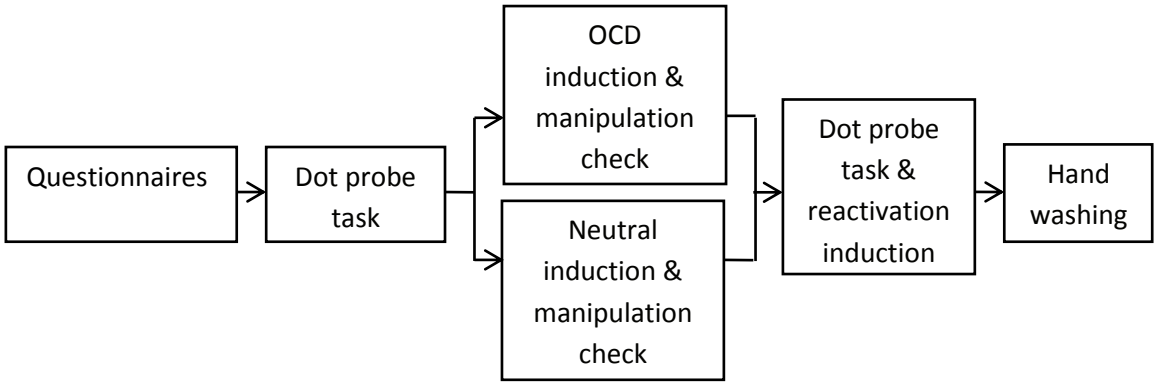

Figure 2. Overview of the procedure of study 2.

Statistical Analysis

All sample characteristics were analyzed using separate *t*-tests. Since a difference between induction groups in previous experienced non-consensual sexual encounter could influence the effectiveness of the induction, potential differences between induction groups in encounters were analyzed using Fisher's exact test. Dot probe analysis was done in the same manner as in study 1. Two participants had average accuracy rates of below 80% and were excluded from further analyses. After exclusion of these participants general accuracy was high (*M* = 96.28%, *SD* = 3.11%, range = 84% - 100%).

In order to check whether the manipulation was successful, we used multiple measures such as the MCR, VAS negative and positive mood, DOCS, and time spent on washing hands. In line with Elliott and Radomsky (2012), we performed separate ANOVA's on perceived kiss desirability and the difference score of pre- and post-physical dirtiness of the man as measured by the MCR as dependent variables and induction group as the independent variable. In order to test for the effects of the induction on feelings of mental contamination (i.e., feelings of dirtiness, urges to wash, internal negative emotions, and external negative emotions), a multivariate ANOVA was conducted on feelings of mental contamination as dependent variables and induction group as independent variable. Moreover, in order to test for the effect of the induction on positive and negative mood, separate mixed ANOVA's with Time (pre- and post-induction) as a within-subject factor and induction group as between-subject factor was performed. Furthermore, in order to test for the effect of the manipulation on the

DOCS and time spent hand washing separate a ANOVA's were performed on the DOCS scores and time spent on washing hands with induction group as the independent variable. Finally, the effect of the reminder of the induction during the second dot probe task was assessed using separate mixed ANOVA's on the disgust and anxiety VAS scales administered before and after the reminder with Time (pre-post induction) as the within-subject factor and induction group as the between-subject factor.

In order to test the hypothesis that current OCD symptoms affect selective attention, we performed a separate mixed ANOVA for each selective attention measure (i.e., attentional bias, attentional interference, and attentional bias variability) with Time (pre- and post-induction) and Valence (OCD-related or generally negative) as within-subject factors and induction group as a between-subject factor.

Finally, in order to test whether baseline selective attention is able to predict an increase in symptoms after the OCD induction, separate linear regressions were performed per OCD symptoms measure after the induction (i.e., feelings of dirtiness, urge to wash, time spent on hand washing, internal negative emotions, external negative emotions, DOCS, VAS negative, and VAS positive) with baseline selective attention (i.e., attentional bias, attentional interference, and attentional bias variability) for OCD-related and negative pictures as independent variables. In the analyses with VAS positive and negative mood we corrected for baseline scores. In these analyses only participants in the OCD symptom induction group were included, as we only expected an increase in symptoms after this induction.

Results

Sample Characteristics

See Table 4 for the means and standard deviations of the sample characteristics. None of the baseline sample characteristics were significantly different between groups (all t's(66) < 1.58, all p's > .120). Furthermore, in this sample 50% experienced a previous non-consensual sexual encounter, but this did not differ per group ($\chi^2(1) = 0.06$, $p = .808$).

Table 4.

Means and standard deviations on demographic and baseline ratings for OCD symptom induction (OCDI) and neutral induction (NI) from study 2

	OCDI (*n* = 35)		NI (*n* = 33)	
	M	*SD*	*M*	*SD*
Age	22.60	3.81	22.24	2.41
Impulsiveness	5.77	3.10	4.52	3.47
MASQ depression	27.17	9.06	27.85	8.26
Baseline positive mood	56.83	17.09	52.59	16.14
Baseline negative mood	15.62	16.27	18.24	14.88
Ease to imagine the scenario	72.75	14.80	75.11	17.18
PI-R washing subscale	5.46	5.14	5.64	4.11
PI-R total	35.46	17.79	37.39	16.31
Y-BOCS	5.06	4.62	4.73	4.02

Note. MASQ = Mood and Anxiety Symptom Questionnaire, PI-R = Padua Inventory-revised, Y-BOCS = Yale-Brown Obsessive–Compulsive Scale.

Manipulation Checks

In order the check whether the manipulation was successful scores from the MCR, VAS negative and positive mood, DOCS and time spent on hand washing were analyzed (see Table 5). There was a significant difference in induction groups for all measures of the mental contamination report and the DOCS, in which participants consistently reported less kiss desirability, a larger difference between pre- and post-physical dirtiness of the man and more symptoms in the OCD induction group than in the neutral induction group. Furthermore, repeated measures ANOVA's showed that there was a significant interaction between induction group and time. Follow-up paired samples *t*-tests showed that there was no difference in induction groups in reported positive or negative mood before the induction (positive mood: $t(66) = 1.05$, $p = .296$; negative mood: $t(66) = 0.69$, $p = .491$), but there was a significant difference between induction groups after the induction (positive mood: $t(66) = 2.21$, $p = .031$; negative mood: $t(47.77) = 5.31$, $p < .001$). After the induction participants in the OCD induction group reported less positive ($M = 42.98$, $SD = 20.31$) and more negative mood (M =

31.80, *SD* = 23.07) than the neutral induction group (positive: *M* = 53.16, *SD* = 17.47; negative: *M* = 8.98, *SD* = 10.36). The only measure that did not reveal a significant difference between induction groups was time spent on hand washing.

Table 5.

Results manipulation check

Variables	Main effect of Induction				Main effect of Time				Induction x Time interaction			
	F	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2
Mental Contamination Report												
Perceived kiss desirability[a]	65.93	1, 66	**< .001**	.50								
Difference pre- and post-physical dirtiness of the man[a]	104.63	1, 66	**< .001**	.61								
Feelings of Mental Contamination[b]	38.16	4, 63	**< .001**	.71								
VAS												
Negative Mood[c]	7.48	1, 66	**.008**	.10	3.80	1, 66	.056	.05	51.45	1, 66	**< .001**	.44
Positive Mood[c]	0.58	1, 66	.448	.01	12.14	1, 66	**.001**	.16	14.34	1, 66	**< .001**	.18
Other measures												
DOCS[a]	36.81	1, 66	**< .001**	.36								
Time hand washing[a]	1.25	1, 66	.268	.02								

Note: DOCS = Dimensional Obsessive-Compulsive Scale. [a] Represents results of univariate ANOVA's; [b] Represents results of MANOVA with feelings of dirtiness, urges to wash, internal negative emotions, and external negative emotions as dependent variables representing feelings of mental contamination; [c] Represents results of 2 (Induction group) x 2 (Time) Mixed ANOVA's.

Table 6.

Effects of disorder-relevance and current OCD symptoms on selective attention

Variables	Main effect of Valence				Induction x Time interaction				Valence x Induction x Time interaction			
	F	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2	*F*	*df*	*p*	η_p^2
Attentional Bias	1.59	1, 66	.212	.02	0.04	1, 66	.834	< .01	0.48	1, 66	.491	< .01
Attentional Interference	14.07	1, 66	**< .001**	.18	0.21	1, 66	.651	< .01	0.87	1, 66	.354	.01
Attentional Bias Variability	0.87	1, 66	.355	.01	1.71	1, 66	.196	.03	1.30	1, 66	.258	.02

Note: Represents results of hypothesis testing within a 2 (Induction group) x 2 (Time) x 2 (Valence) Mixed ANOVA's.

Manipulation Check Reminder Induction

In order to determine whether the Reminder of the induction was successful, a separate mixed ANOVA was performed on the anxiety VAS scales and disgust VAS scales administered before and after the reminder with Time (pre-post reminder) as a within-subject factor and induction group as a between-subject factor. These analyses showed significant Time x Induction group interactions (disgust: $F(1,64) = 70.20$, $p < .001$, $\eta_p^2 = .52$; anxiety: $F(1,64) = 30.53$, $p < .001$, $\eta_p^2 = .32$). Follow-up paired samples *t*-tests showed that this effect was due to no significant change in disgust or anxiety for the neutral induction group (anxiety: $t(31) = 0.13$, $p = .896$; disgust: $t(31) = 0.55$, $p = .589$) while there was a significant increase in anxiety ($M_{diff} = 22.85$, $SD_{diff} = 21.79$) and disgust ($M_{diff} = 37.12$, $SD_{diff} = 22.93$) in the OCD induction group (anxiety: $t(33) = 6.12$, $p < .001$; disgust: $t(33) = 9.44$, $p < .001$). There was also a main effect of Time (disgust: $F(1,64) = 61.76$, $p < .001$, $\eta_p^2 = .49$; anxiety: $F(1,64) = 29.38$, $p < .001$, $\eta_p^2 = .31$) and induction group (disgust: $F(1,64) = 39.17$, $p < .001$, $\eta_p^2 = .38$; anxiety: $F(1,64) = 13.23$, $p = .001$, $\eta_p^2 = .17$). These effects were qualified by the Time x Group interaction effect.

Effects of Disorder-Relevance and Current OCD Symptoms on Selective Attention

The results of the 2 (Induction group) x 2

(Time) x 2 (Valence) ANOVA's are presented in Table 6. The predicted effect of an influence of symptoms on selective attention by an Induction x Time interaction was not significant for any measure of selective attention. Furthermore there were no Valence x Induction x Time interaction effects and there was only an effect of the Valence of the pictures (OCD-related or generally negative) for attentional interference. On average participants showed more attentional interference after OCD-related pictures (M = 8.45, SD = 10.05) than after generally negative pictures (M = 3.02, SD = 9.32). Interestingly, there was also a Time x Valence interaction effect for attentional interference. Follow-up paired t-tests showed that the effect of valence was significant during the first dot probe task ($t(67) = 4.87$, $p < .001$), but not during the second dot probe task ($t(67) = 1.20$, $p = .235$)[4].

Predicting Symptoms based on Baseline Selective Attention

Linear regressions performed on feelings of dirtiness, urge to wash, external negative emotions, internal negative emotions, DOCS scores, time spent on washing hands and positive and negative mood did not show any significant effects (all p's > .117). Baseline selective attention (i.e. attentional bias, attentional interference and attentional bias variability) for any type of picture did not predict the increase in symptoms after OCD symptom induction.

Discussion

The second study set out to examine the effects of an OCD symptom induction on subsequent selective attention to contamination-related stimuli and the ability of baseline selective attention to predict an increase in symptoms after OCD symptom induction. Importantly, the manipulation checks showed that the OCD symptom induction was successful for every measure except time spent on hand washing. Thus, the induction was successful in inducing feelings of mental contamination and intrusive thoughts, but this effect did not generalize to washing behavior in the lab. Moreover, the manipulation check of the reminder of the induction during the second task showed

[4] Including Padua contamination scores did not result in any state (group) x trait (PI-R scores) interactions.

that this reminder was successful in maintaining the effects of the induction. These findings are important as they imply that, if selective attention is influenced by increased state contamination fear, we can expect increased selective attention to OCD-related stimuli after this induction.

The predicted increase in selective attention after OCD symptom induction was not significant. Therefore, the current study does not provide evidence for the view that selective attention to threat is highly responsive to state manipulation in the context of contamination fear. Furthermore, contrary to the view that attentional bias contributes to OCD symptoms, baseline selective attention was not able to predict an increase in symptoms after OCD symptom induction. Interestingly, participants showed more attentional interference for OCD-related stimuli than generally negative stimuli. Similarly, Morein-Zamir et al. (2013) found selective attention towards idiosyncratic pictorial stimuli in nonanxious individuals. Moreover, this finding corresponds to Pergamin-Hight et al. (2015) who found that attentional bias is specific for disorder-related stimuli. This valence-specific effect for attentional interference was only present during the first dot probe task (i.e., before OCD or neutral induction).

General Discussion

The current studies investigated the link between OCD symptoms and selective attention. Research regarding an attentional bias to OCD-related stimuli in the context of OCD has been mixed and characterized by several limitations. First, to date little research has been done on attentional bias as a dynamic process which can change over time. Second, from the current literature it is unclear whether attentional bias has an influence on OCD symptoms or whether state OCD symptoms influence attentional bias. Some cognitive models have proposed that attentional bias to threat is one of the mechanisms contributing to the development and maintenance of OCD (e.g., Bar-Haim et al., 2007; Muller & Roberts, 2005), while other models such as the ACT have proposed a mutually reinforcing relation between attentional bias towards threat and anxiety (Eysenck et al., 2007). Therefore, selective attention to threat may increase after the induction of OCD symptoms. These limitations were addressed in two studies. The first study examined the difference between a HCF and LCF group in selective attention using a cross-sectional design. In the second study an experimental design

was used in which selective attention was assessed before and after an induction designed to elicit symptoms similar to OCD. Furthermore, in the second study we investigated whether selective attention for OCD-related stimuli at baseline could predict an increase in symptoms after an OCD symptoms induction. In the current studies we found no evidence for either a trait-related presence of selective attention nor for influences of experimentally induced contamination fear. Moreover, baseline selective attention had no impact on subsequent OCD induction.

The findings that there was no effect of trait OCD and that baseline selective attention is not able to predict changes in OCD symptoms are in line with other studies that did not find an effect of trait OCD symptoms on selective attention (e.g., Harkness et al., 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008). However, the results are in contrast with Bar-Haim et al. (2007) and other studies who did find an effect of selective attention in OCD (e.g., Amir et al., 2009; Moritz et al., 2009). The absence of a relationship between trait OCD and selective attention is also in contrast with cognitive models proposing that attentional bias to threat is one of the mechanisms contributing to the development and maintenance of OCD (e.g., Bar-Haim et al., 2007; Muller & Roberts, 2005). The finding that there was no effect of an OCD symptom induction on subsequent selective attention is in contrast with Cohen et al. (2003), who found a decrease in performance after OCD symptom induction. Furthermore, this finding suggests models such as the ACT (Eysenck et al., 2007) proposing a mutually reinforcing relation between attentional bias towards threat and anxiety may not apply to OCD.

It is important to note that the sample size for these studies was based on a priori power analyses. These power analyses were based on meta-analytic findings on attentional bias where a medium effect size was observed (Bar-Haim et al., 2007). Thus we were underpowered to demonstrate small effect sizes, yet sufficiently powered to find medium effect sizes. Therefore it is unlikely that the current results are due to a lack of power. These results suggest, in line with Summerfeldt and Endler (1998), that selective attention may not play a pivotal role in the context of OCD. Another possibility is that the relationship between selective attention is more complex than a unidirectional relationship from either selective attention to OCD symptoms or from OCD symptoms to selective attention. For instance, Muller and Roberts (2005) have

suggested cognitive variables might interact to influence OCD. Future research is necessary in order to determine whether the relationship between OCD and selective attention is more complex or whether selective attention is not as important for OCD as it is for other anxiety disorders (Summerfeldt & Endler, 1998).

A strength of the current studies was that they investigated attentional bias both as a dynamic process and as stable attentional bias and interference scores. Contrary to the traditional attentional bias and interference measures, the TL-BS measure of attentional bias variability has demonstrated good to excellent reliability and validity (Rodebaugh et al., 2016; Zvielli et al., 2015). Interestingly, we largely found the same results regardless of the specific measure of selective attention in our studies. Previous research has highlighted the need for the use of idiosyncratic stimuli in the investigation to attention (Muller & Roberts, 2005). Therefore, a specific strength of the second study was that it included a procedure for idiosyncratic picture selection.

These studies are characterized by several limitations. First and foremost, these studies used either a subclinical sample (study 1) or a convenience sample (study 2). Interestingly however, one-sample *t*-tests showed that all participants displayed an attentional bias (regardless of HCF or LCF), suggesting the possibility to examine attentional bias in a convenience sample. Moreover, the utility of analogue samples in research on the mechanisms of OCD has been demonstrated elaborately by Gibbs (1996) and Abramowitz et al. (2014). A second limitation is that although the induction of OCD symptoms was successful, it is possible that the pictures themselves already acted as an OCD symptom induction. However, it is likely that a separate OCD induction in study 2 would have a stronger effect on selective attention than pictures alone. A third limitation is that these studies focused on the contamination symptom dimension of OCD, which limits the generalizability of these findings to other symptom dimensions of OCD. Indeed, Harkness et al. (2009) suggested selective attention to be specific for the contamination symptom dimension. Future research would benefit from an comprehensive study including clinical OCD patients with multiple symptom dimensions, in order to check whether any found effects apply to OCD in general or only to specific symptom dimensions.

Limitations notwithstanding, the current studies were among the first investigating the link between OCD symptoms and selective attention considered as a

dynamic process in time. In conclusion, there was little evidence for selective attention as a mechanism influencing OCD symptoms since selective attention to contamination-related stimuli was found in participants regardless of scoring high or low on contamination fear. Moreover, baseline selective attention did not predict increased OCD symptoms after an OCD symptom induction. Finally, we did not find evidence for an influence of state OCD symptoms on selective attention, since an OCD symptom induction did not affect subsequent selective attention. These results suggest that selective attention may not be as important for OCD as it is for other anxiety disorders or that the relation between OCD and selective attention is more complex than an unidirectional relationship.

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References

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., . . . Hale, L. R. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the dimensional obsessive-compulsive scale. *Psychological Assessment, 22*(1), 180-198. doi: 10.1037/a0018260

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive–compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157. doi: 10.1016/j.brat.2008.10.020

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Baer, L. (1991). *Getting control: Overcoming your obsessions and compulsions*. New York, NY: Penguin.

Ball, S. G., Baer, L., & Otto, M. W. (1996). Symptom subtypes of obsessive-compulsive disorder in behavioral treatment studies: A quantitative review. *Behaviour Research and Therapy, 34*(1), 47-51. doi: 10.1016/0005-7967(95)00047-2

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1-24. doi: 10.1037/0033-2909.133.1.1

Bradley, M. C., Hanna, D., Wilson, P., Scott, G., Quinn, P., & Dyer, K. F. W. (2016). Obsessive–compulsive symptoms and attentional bias: An eye-tracking methodology. *Journal of Behavior Therapy and Experimental Psychiatry, 50*, 303-308. doi: 10.1016/j.jbtep.2015.10.007

Broderick, J., Grisham, J. R., & Weidemann, G. (2013). Disgust and fear responding in contamination-based obsessive-compulsive disorder during pictorial exposure. *Behavior Therapy, 44*(1), 27-38. doi: 10.1016/j.beth.2012.05.005

Cohen, Y., Lachenmeyer, J. R., & Springer, C. (2003). Anxiety and selective attention in obsessive–compulsive disorder. *Behaviour Research and Therapy, 41*(11), 1311-1323. doi: 10.1016/S0005-7967(03)00037-8

Coskunpinar, A., & Cyders, M. A. (2013). Impulsivity and substance-related attentional bias: A meta-analytic review. *Drug and Alcohol Dependence, 133*(1), 1-14. doi: 10.1016/j.drugalcdep.2013.05.008

De Putter, L. M. S., Van Yper, L., & Koster, E. H. W. (2017). Obsessions and compulsions in the lab: A meta-analysis of procedures to induce symptoms of obsessive-compulsive disorder. *Clinical Psychology Review, 52*, 137-147. doi: 10.1016/j.cpr.2017.01.001

Elliott, C. M., & Radomsky, A. S. (2012). Mental contamination: The effects of imagined physical dirt and immoral behaviour. *Behaviour Research and Therapy, 50*(6), 422-427. doi: 10.1016/j.brat.2012.03.007

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353. doi: 10.1037/1528-3542.7.2.336

Eysenck, S. B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*(5), 613-619. doi: 10.1016/0191-8869(85)90011-x

Fairbrother, N., Newth, S. J., & Rachman, S. (2005). Mental pollution: Feelings of dirtiness without physical contact. *Behaviour Research and Therapy, 43*(1), 121-130. doi: 10.1016/j.brat.2003.12.005

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (1998). *User's guide for the Structured clinical interview for DSM-IV axis I disorders SCID-I: clinician version.* (M. A. C. van Groenestijn, G. W. Akkerhuis, R. W. Kupka, N. Schneider & W. A. Nolen, Trans.). Amsterdam, NL: Pearson Assessment and Information BV.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory: Development and validation of

a short version. *Psychological Assessment, 14*(4), 485-496. doi: 10.1037/1040-3590.14.4.485

Foa, E. B., Kozak, M. J., Salkovskis, P. M., Coles, M. E., & Amir, N. (1998). The validation of a new obsessive–compulsive disorder scale: The Obsessive–Compulsive Inventory. *Psychological Assessment, 10*(3), 206-214. doi: 10.1037/1040-3590.10.3.206

Gibbs, N. A. (1996). Nonclinical populations in research on obsessive-compulsive disorder: A critical review. *Clinical Psychology Review, 16*(8), 729-773. doi: 10.1016/S0272-7358(96)00043-8

Haidt, J., McCauley, C., & Rozin, P. (1994). Individual differences in sensitivity to disgust: A scale sampling seven domains of disgust elicitors. *Personality and Individual Differences, 16*(5), 701-713. doi: 10.1016/0191-8869(94)90212-7

Hajcak, G., Huppert, J. D., Simons, R. F., & Foa, E. B. (2004). Psychometric properties of the OCI-R in a college sample. *Behaviour Research and Therapy, 42*(1), 115-123. doi: 10.1016/j.brat.2003.08.002

Harkness, E. L., Harris, L. M., Jones, M. K., & Vaccaro, L. (2009). No evidence of attentional bias in obsessive compulsive checking on the dot probe paradigm. *Behaviour Research and Therapy, 47*(5), 437-443. doi: 10.1016/j.brat.2009.02.004

Herba, J. K., & Rachman, S. (2007). Vulnerability to mental contamination. *Behaviour Research and Therapy, 45*(11), 2804-2812. doi: 10.1016/j.brat.2007.07.010

Hodgson, R. J., & Rachman, S. (1977). Obsessional-compulsive complaints. *Behaviour Research and Therapy, 15*(5), 389-395. doi: 10.1016/0005-7967(77)90042-0

Hou, R. H., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite, 56*(2), 424-427. doi: 10.1016/j.appet.2011.01.019

Inquisit 3 [Computer software]. (2011). Retrieved from http://www.millisecond.com.

Inquisit 4 [Computer software]. (2015). Retrieved from http://www.millisecond.com.

Koster, E. H. W., De Raedt, R., Goeleven, E., Franck, E., & Crombez, G. (2005). Mood-congruent attentional bias in dysphoria: Maintained attention to and impaired disengagement from negative information. *Emotion, 5*(4), 446-455. doi: 10.1037/1528-3542.5.4.446

Lang, P. J., Bradley, M. M., & Cuthbert, B. (1997). *International affective picture system (IAPS): Instruction manual and affective ratings.* University of Florida, NIMH Center for the Study of Emotion and Attention, Gainesville, FL, USA.

Lavy, E., Van Oppen, P., & Van Den Hout, M. (1994). Selective processing of emotional information in obsessive compulsive disorder. *Behaviour Research and Therapy, 32*(2), 243-246. doi: 10.1016/0005-7967(94)90118-X

Lijffijt, M., Caci, H., & Kenemans, J. L. (2005). Validation of the Dutch translation of the I7 questionnaire. *Personality and Individual Differences, 38*(5), 1123-1133. doi: 10.1016/j.paid.2004.07.010

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*(1), 15-20. doi: 10.1037/0021-843X.95.1.15

Markarian, Y., Larson, M. J., Aldea, M. A., Baldwin, S. A., Good, D., Berkeljon, A., . . . McKay, D. (2010). Multiple pathways to functional impairment in obsessive-compulsive disorder. *Clinical Psychology Review, 30*(1), 78-88. doi: 10.1016/j.cpr.2009.09.005

Mataix-Cols, D., do Rosario-Campos, M. C., & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *American Journal of Psychiatry, 162*(2), 228-238. doi: 10.1176/appi.ajp.162.2.228

Mataix-Cols, D., Lawrence, N. S., Wooderson, S., Speckens, A., & Phillips, M. L. (2009). The Maudsley Obsessive–Compulsive Stimuli Set: Validation of a standardized paradigm for symptom-specific provocation in obsessive–compulsive disorder. *Psychiatry Research, 168*(3), 238-241. doi: 10.1016/j.psychres.2008.05.007

McNair, D. M., Lorr, M., & Dropplemann, L. F. (1992). *Manual: Profile of mood states revised*. San Diego, CA: Educational and Industrial Testing Service.

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Moritz, S., Fischer, B.-K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., & Jelinek, L. (2008). Words may not be enough! No increased emotional Stroop effect in obsessive–compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104. doi: 10.1016/j.brat.2008.05.005

Moritz, S., Von Muehlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive–compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371. doi: 10.1017/S1355617709090511

Moritz, S., & von Mühlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229. doi: 10.1002/da.20294

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive–Compulsive Disorder: A review. *Journal of Anxiety Disorders, 19*(1), 1-28. doi: 10.1016/j.janxdis.2003.12.001

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive-compulsive symptoms. *Journal of Abnormal Psychology, 119*(1), 136-142. doi: 10.1037/a0017549

Olatunji, B. O., Moretz, M. W., McKay, D., Bjorklund, F., de Jong, P. J., Haidt, J., . . . Schienle, A. (2009). Confirming the three-factor structure of the Disgust Scale—Revised in eight countries. *Journal of Cross-Cultural Psychology, 40*(2), 234-255. doi: 10.1177/0022022108328918

Olatunji, B. O., Williams, N. L., Tolin, D. F., Abramowitz, J. S., Sawchuk, C. N., Lohr, J. M., & Elwood, L. S. (2007). The Disgust Scale: Item analysis, factor structure, and suggestions for refinement. *Psychological Assessment, 19*(3), 281-297. doi: 10.1037/1040-3590.19.3.281

Pergamin-Hight, L., Naim, R., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., & Bar-Haim, Y. (2015). Content specificity of attention bias to threat in anxiety disorders: A meta-analysis. *Clinical Psychology Review, 35*, 10-18. doi: 10.1016/j.cpr.2014.10.005

Radomsky, A. S., Elliott, C. M., Rachman, S., Fairbrother, N., & Newth, S. J. (2008). *Mental Contamination Report*. Unpublished questionnaire, Concordia University, Montreal, QC.

Rodebaugh, T. L., Scullin, R. B., Langer, J. K., Dixon, D. J., Huppert, J. D., Bernstein, A., . . . Lenze, E. J. (2016). Unreliability as a threat to understanding psychopathology:

The cautionary tale of attentional bias. *Journal of Abnormal Psychology, 125*(6), 840-851. doi: 10.1037/abn0000184

Rossi, V., & Pourtois, G. (2012). Transient state-dependent fluctuations in anxiety measured using STAI, POMS, PANAS or VAS: A comparative review. *Anxiety, Stress, & Coping, 25*(6), 603-645. doi: 10.1080/10615806.2011.582948

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/mp.2008.94

Steketee, G., Frost, R., & Bogart, K. (1996). The Yale-Brown Obsessive Compulsive Scale: Interview versus self-report. *Behaviour Research and Therapy, 34*(8), 675-684. doi: 10.1016/0005-7967(96)00036-8

Summerfeldt, L. J., & Endler, N. S. (1998). Examining the evidence for anxiety-related cognitive biases in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 12*(6), 579-598. doi: 10.1016/S0887-6185(98)00035-8

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in Obsessional Compulsive Disorder. *Behaviour Research and Therapy, 34*(1), 53-60. doi: 10.1016/0005-7967(95)00041-U

Van Bockstaele, B., Verschuere, B., Tibboel, H., De Houwer, J., Crombez, G., & Koster, E. H. W. (2014). A review of current evidence for the causal impact of attentional bias on fear and anxiety. *Psychological Bulletin, 140*(3), 682-721. doi: 10.1037/a0034834

Van Oppen, P., Hoekstra, R. J., & Emmelkamp, P. M. G. (1995). The structure of obsessive-compulsive symptoms. *Behaviour Research and Therapy, 33*(1), 15-23. doi: 10.1016/0005-7967(94)E0010-G

Vogt, J., Lozo, L., Koster, E. H. W., & De Houwer, J. (2011). On the role of goal relevance in emotional attention: Disgust evokes early attention to cleanliness. *Cognition and Emotion, 25*(3), 466-477. doi: 10.1080/02699931.2010.532613

Wardenaar, K. J., van Veen, T., Giltay, E. J., de Beurs, E., Penninx, B. W. J. H., & Zitman, F. G. (2010). Development and validation of a 30-item short adaptation of the Mood and Anxiety Symptoms Questionnaire (MASQ). *Psychiatry Research, 179*(1), 101-106. doi: 10.1016/j.psychres.2009.03.005

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*(1), 15-25. doi: 10.1037/0021-843X.104.1.15

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 3-14. doi: 10.1037/0021-843X.104.1.3

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2014). Dynamics of Attentional Bias to Threat in Anxious Adults: Bias towards and/or Away? *PLoS ONE, 9*(8), e104025. doi: 10.1371/journal.pone.0104025

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2015). Temporal dynamics of attentional bias. *Clinical Psychological Science, 3*(5), 772–788. doi: 10.1177/2167702614551572

CHAPTER 5

CAN SELECTIVE ATTENTION AND INHIBITION CAPACITY (INTERACTIVELY) PREDICT FUTURE OCD SYMPTOMS? A PROSPECTIVE STUDY[1]

ABSTRACT

The current study set out to investigate whether obsessive beliefs, selective attention, inhibition, and the interaction between selective attention and inhibition can prospectively predict OCD symptoms. Obsessive beliefs, inhibition, and selective attention were assessed in a student sample (*n* = 89) during a baseline session in the beginning of the first semester. Their predictive value was examined by assessing symptoms after an OCD symptoms induction in the lab and by assessing OCD symptoms during a period of increased stress (the examination period) 68 to 80 days after baseline. Results showed that obsessive beliefs did not consistently predict OCD symptoms and there was no predictive effect of attentional bias, attentional bias variability, and inhibition capacity in isolation. However, attentional bias variability and inhibition capacity in the context of contamination-related stimuli interacted, in which only the combination of poor inhibition capacity and large attentional bias variability predicted contamination OCD symptoms during the examination period. These results support the notion that information processing biases act in concert rather than in isolation in predicting contamination OCD symptoms.

[1] Based on De Putter, L. M. S., & Koster, E. H. W. (2017). *Can selective attention and inhibition (interactively) predict future obsessive compulsive symptoms? A prospective study in undergraduates.* Manuscript submitted for publication.

Introduction

Patients with obsessive-compulsive disorder (OCD) suffer from recurrent intrusive thoughts and/or repetitive compulsions (American Psychiatric Association, 2013). With a lifetime prevalence rate of 2-3.5% this persistent and debilitating disorder has been identified as the fourth most common mental disorder (Angst et al., 2004; Ruscio, Stein, Chiu, & Kessler, 2010).

One of the mechanisms that has been put forward to explain the development and maintenance of OCD are cognitive beliefs (Obsessive Compulsive Cognitions working Group; OCCWG, 1997; Rachman, 1997, 1998; Salkovskis, 1985). For instance, the OCCWG (2005) identified three factors of obsessive beliefs: (1) responsibility and threat estimation, (2) perfectionism and intolerance of uncertainty, and (3) importance and control of thoughts. These theories suggest that overestimation of threat and feeling responsible for possible harm can exacerbate OCD symptoms, such as repeatedly checking whether they did not cause accidental harm. Similarly, the need for things to be perfect and an intolerance of feelings of uncertainty can lead to obsessions over symmetry or checking repetitively. Moreover, by believing a thought can increase the likelihood of an event or that a thought alone has implications for one's moral character (i.e., thought-action fusion), patients can feel an increased need to neutralize these thoughts.

To date, the results of the few studies that prospectively investigated the predictive value of these obsessive beliefs have been inconsistent. For instance, Abramowitz, Khandker, Nelson, Deacon, and Rygwall (2006) followed 85 participants during child birth and postpartum. They found that obsessive beliefs predicted the development of OCD symptoms after an average interval of seven to eight months. Similarly, Coles and Horng (2006) followed 377 students and found that obsessive beliefs significantly predicted OCD symptoms after six weeks. In contrast, Novara et al. (2011) conducted a longitudinal study in which they followed 99 students for five years. Although obsessive beliefs were associated with symptoms at baseline, obsessive beliefs did not influence OCD symptoms at one, three or five year follow-up. Similarly, Coles, Pietrefesa, Schofield, and Cook (2008) followed 572 students and found no

predictive value of obsessive beliefs when negative life events were entered in the model.

Another mechanism that has been put forward as contributing to the development and maintenance of OCD is selective attention to threat (Muller & Roberts, 2005). Selective attention is defined as a tendency to selectively attend to threatening stimuli over nonthreatening stimuli. Selective attention to threat could lead to increased perception of threat in the environment, which could subsequently exacerbate OCD symptoms (Muller & Roberts, 2005). Although some research has found an association between attentional bias for OCD-related stimuli and OCD (e.g., Amir, Najmi, & Morrison, 2009; Lavy, Van Oppen, & Van Den Hout, 1994; Moritz, Von Muehlenen, Randjbar, Fricke, & Jelinek, 2009; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996), other studies found no such association (e.g., Harkness, Harris, Jones, & Vaccaro, 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008). Interestingly, an experimental study of Najmi and Amir (2010) demonstrated that decreasing attentional bias can result in more behavioral approach towards contamination stimuli in subclinical contamination fear participants. However, to our knowledge, no studies have examined the relationship between selective attention and OCD symptoms prospectively.

Response inhibition, the ability to inhibit a prepotent motor response (Logan, 1994), is a third factor that has often been associated with OCD (Abramovitch, Abramowitz, & Mittelman, 2013). Response inhibition has been put forward as an endophenotype of OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). In this view, a response inhibition deficit is linked to the genetics and the neurobiology of OCD and plays a key factor in the vulnerability to develop OCD. Indeed, some studies found similar underperformance in inhibition in OCD patients and their healthy relatives and in OCD patients in remission (Bannon, Gonsalvez, Croft, & Boyce, 2006; Menzies et al., 2007). However, to our knowledge, this assumption has not been tested prospectively.

Muller and Roberts (2005) have suggested that to date too much research focusses on information processing factors in isolation. Another interesting approach to vulnerability factors in OCD is looking at the interaction between information processing factors. Similarly, Hirsch, Clark, and Mathews (2006) proposed the

"combined cognitive biases hypothesis", which states that information processing factors will likely act in concert rather than in isolation in predicting symptoms. For instance, the attentional control theory (ACT; Eysenck, Derakshan, Santos, & Calvo, 2007) poses that attentional control is governed by bottom-up capture as well as top-down control (Corbetta & Shulman, 2002). Threatening information is associated with the bottom-up capture of attention where in many situations attention is selectively oriented towards threatening information. Inhibition is related to the top-down control system, which is goal-oriented and enables us to focus on the task at hand. It is plausible these systems interact in the prediction of OCD symptoms. For instance, one could expect that having a tendency to selectively attend to threatening stimuli is particularly harmful when it is combined with weakened top-down control by difficulty in response inhibition after encountering such stimuli. The merit of the combined cognitive biases hypothesis has already been demonstrated in other disorders such as social anxiety (Hirsch et al., 2006), but has yet to be demonstrated in the context of OCD.

While there is much research available investigating the link between information processing factors and OCD, there is little research investigating interactive effects between information processing factors and even fewer research doing so prospectively. The current study addressed this issue by investigating whether cognitive beliefs, selective attention, response inhibition, and the interaction between selective attention and response inhibition can prospectively predict OCD symptoms. For this purpose selective attention, inhibition, and obsessive beliefs were assessed during a baseline session in a student sample in the beginning of the first semester. In order to investigate whether these factors could predict an increase in symptoms in the lab, we used an OCD symptom induction (see method section). General stress has been associated with elevated OCD symptoms (Coles & Horng, 2006). In a student sample the examination period is an ecologically valid stressor. Therefore, we assessed OCD symptoms during a second session in the examination period.

Since the OCD symptom induction in the lab is closely related to the contamination fear symptom dimension of OCD, the OCD-relevant material within this study was focused on the contamination symptom dimension. Contamination fear consists of a fear of being contaminated or contaminating others and is one of the most

common symptom dimensions in OCD (Ball, Baer, & Otto, 1996; Markarian et al., 2010). The induction in the lab consisted of an induction based on mental contamination, as mental contamination emerged as one of the most effective induction procedures of OCD symptoms in a meta-analysis (De Putter, Van Yper, & Koster, 2017). Mental contamination is often characterized by a moral element and consists of a sense of internal dirtiness (Rachman, 2004).

Method

Participants

The sample included 99 participants (21 males, 78 females) ranging in age from 17 to 40 years (*M* = 19.76, *SD* = 3.23). Participants from Ghent University were recruited online. Two participants reported suicidal ideation and were therefore not subjected to the OCD symptom induction. One participant could not report a specific memory for the OCD symptom induction (see method section) and was similarly excluded from analyses. Six participants did not respond to the follow-up assessment call during the examination period and one participant did not have any exams. These participants were excluded from analyses. The final sample included 89 participants. The study was approved by the local ethical committee at Ghent University. Informed consent was obtained from all participants included in the study. Participants were either paid 25 euro or course credit and 15 euro for their contribution.

Measures

MINI-screen. In order to check for psychopathology such as clinical OCD, the Dutch version of the MINI International Neuropsychiatric Interview-screen was used (MINI-screen; Sheehan et al., 1998). The MINI is a structured interview and consists of questions assessing psychopathology based on the DSM-IV. The MINI has good psychometric properties (Sheehan et al., 1998).

Disgust Scale-Revised (DS-R). The DS-R (Haidt, McCauley, & Rozin, 1994; Olatunji et al., 2009; Olatunji et al., 2007) was used to assess disgust sensitivity, which is associated with contamination fear OCD (Broderick, Grisham, & Weidemann, 2013). The DS-R consists of 25 items that were rated on a scale from 0 (completely disagree/not disgusting at all) to 4 (completely agree/very disgusting). The scale is

comprised of three subscales (core disgust, animal reminder disgust and contamination disgust). The DS-R has good psychometric properties (Olatunji et al., 2009; Olatunji et al., 2007).

Impulsiveness–Venturesomeness–Empathy questionnaire (I_7). Since both inhibition capacity and attentional bias have previously been associated with impulsivity (e.g., Coskunpinar & Cyders, 2013; Hou et al., 2011), the impulsiveness subscale of the I_7 (Eysenck, Pearson, Easting, & Allsopp, 1985; Lijffijt, Caci, & Kenemans, 2005) was administered. The impulsiveness subscale consists 19 dichotomous (yes/no) items and has good psychometric properties (Lijffijt et al., 2005).

Mood and Anxiety Symptoms Questionnaire (MASQ-D30). The anhedonic depression scale of the short adaptation of the MASQ (Wardenaar et al., 2010; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995) was administered, since depression has been associated with cognitive functioning (McDermott & Ebmeier, 2009). This scale consisted of 10 items and were rated on a scale from 1 (not at all) to 5 (very much). The short adaptation of the MASQ has good psychometric properties (Wardenaar et al., 2010).

Obsessive Beliefs Questionnaire (OBQ-44). The OBQ-44 was administered in order to assess beliefs considered critical in the development and maintenance of OCD (OCCWG, 2005). The OBQ-44 consists of 44 items and three subscales: overestimation of responsibility and threat, perfectionism and the need for certainty, and importance and control of thoughts. Items were rated on a scale from 1 (disagree very much) to 7 (agree very much). The OBQ-44 has good psychometric properties (OCCWG, 2005).

Yale-Brown Obsessive–Compulsive Scale (Y-BOCS). The Y-BOCS severity self-report as designed by Baer (1991) was one of the three measures that were administered both at baseline and during the examination period in order to assess OCD symptoms. The self-report version of the Y-BOCS is also characterized by good psychometric properties (Steketee, Frost, & Bogart, 1996). As in the Y-BOCS interview, the self-report Y-BOCS included an explanation on the nature of obsessions and compulsions. The Y-BOCS consisted of 10 items of which 5 items assessed obsessions and 5 items assessed compulsions. The items assessed time spent, interference, distress, resistance, and control over obsessions or compulsions on a scale from 0 (none) to 4 (extreme).

Obsessive Compulsive Inventory-revised (OCI-R). The OCI-R (Foa et al., 2002; Foa, Kozak, Salkovskis, Coles, & Amir, 1998) was the second of the three measures that were administered both at baseline and during the examination period in order to assess OCD symptoms. The OCI-R consisted of 18 items, which were rated on a Likert scale from 0 (not at all) to 4 (extremely). The six subscales assessed washing, checking, ordering, obsessing, hoarding, and neutralizing. The OCI-R has good psychometric properties (Hajcak, Huppert, Simons, & Foa, 2004).

Padua Inventory-revised (PI-R). The PI-R (Van Oppen, Hoekstra, & Emmelkamp, 1995) was the third of three measures that were administered both at baseline and during the examination period in order to assess OCD symptoms. The PI-R consisted of 41 items comprising five subscales: impulses, washing, checking, rumination, and precision. Items were rated on a Likert scale form 0 (never/not at all) to 4 (very often). The PI-R has good psychometric properties (Van Oppen et al., 1995).

Visual Analogue Scales (VAS). VAS scales were used in order to assess a change between pre- and post-OCD symptom induction. Seven VAS were adopted from the Profile of Mood States (McNair, Lorr, & Dropplemann, 1992). Positive mood was estimated using the VAS scales “energetic”, “satisfied”, and “happy” and negative mood was estimated using the VAS scales “angry”, “tense”, and “depressed”. The VAS scales “anxious” and “guilty” were added for the purpose of the OCD symptom induction. A separate scale was used to assess fatigue. VAS scales assessing disgust, urge to wash, feelings of dirtiness in located in the body and dirtiness in general were added because of the relevance of disgust for contamination fear (Broderick, Grisham, & Weidemann, 2013). Finally, VAS scales assessing the vividness of the memory and the ease to imagine the memory were added in order to determine whether the process of imagining the memory for the OCD symptom induction was successful.

Dimensional Obsessive-Compulsive Scale (DOCS). In order to determine whether the OCD symptom induction was successful in eliciting contamination fear, we adapted three items of the contamination subscale of the DOCS (Abramowitz et al., 2010). The adapted questions consisted of: “How much time have you spent during the experiment on thinking about contamination?”, “How much time have you spent during the experiment on washing or cleaning behaviors because of contamination?”, and “How difficult was it for you during the experiment to disregard thoughts about

contamination and refrain from behaviors such as washing, showering, cleaning and other decontamination routines when you tried to do so?". Participants rated these items on a scale from 0 (none at all/not at all difficult) to 4 (most of the time/extremely difficult). These items were administered before and after the OCD symptom induction. The DOCS before the induction enquired about symptoms experienced during the first part of the experiment. The DOCS after the induction enquired about symptoms experienced specifically during the induction.

Materials and Experimental Tasks

Picture selection procedure. In total 60 contamination-related pictures (e.g., a dirty toilet) were selected from the picture set of Morein-Zamir et al. (2013), the Maudsley Obsessive-Compulsive Stimuli Set (Mataix-Cols, Lawrence, Wooderson, Speckens, & Phillips, 2009), the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997), and publically available online sources. The 60 negative (e.g., a gun) and neutral (e.g., a leaf) pictures were selected from the IAPS. The negative and contamination-related pictures were matched on arousal based on ratings of an independent sample (*N* = 28) on a Likert scale ranging from 1 (none) to 9 (very much) on arousal and how much fear and disgust the pictures elicited. Moreover, the valence of the pictures were rated on a Likert scale ranging from 1 (negative) to 9 (positive)[2]. In order to enhance the personal relevance of the contamination-related pictures for the participant, participants in the current study rated these pictures on how much anxiety they elicited.

Stop-Signal Task (SST). The SST (Logan, 1994) as adapted by Verbruggen and De Houwer (2007) was used in order to assess inhibition capacity in the context of contamination-related pictures. The SST was programmed using Presentation® software (version 17.2, Neurobehavioral Systems). Each trial started with a fixation cross

[2] *M* arousal OCD pictures = 4.17, *SD* arousal OCD pictures = 0.94, *M* arousal negative pictures = 4.90, *SD* arousal negative pictures = 0.73; *M* fear OCD pictures = 2.56, *SD* fear OCD pictures = 0.91, *M* fear negative pictures = 4.29, *SD* fear negative pictures = 1.38; *M* disgust OCD pictures = 4.51, *SD* disgust OCD pictures = 1.44, *M* disgust negative pictures = 3.01, *SD* disgust negative pictures = 1.06; *M* valence OCD pictures = 3.63, *SD* valence OCD pictures = 0.60, *M* valence negative pictures = 3.01, *SD* valence negative pictures = 0.63

presented for 500ms (70 x 100 pixels) followed by a picture for 500ms (384 x 288 pixels) and finally the target ("#" or "@", 100 x 100 pixels). A response was required within 1250ms by pressing the "D" key for the "#" and the "K" key for the "@" on an AZERTY keyboard. This mapping rule was counterbalanced between participants. Participants were instructed to respond as quickly as possible. There was an intertrial interval of 1500ms. In 30% of the trials a clearly audible stop-signal (75ms) was presented through headphones. Participants were instructed to inhibit their response following a stop-signal. In order to obtain a probability of stopping of 50% per participant, the stop-signal delay (SSD) started at 250ms and was continuously adjusted using a separate staircase tracking procedure (Levitt, 1971). The SSD was increased by 25ms when participants participants were succesful in inhibiting their response after a stop-signal, and the SSD was decreased by 25ms when participants failed to inhibit their response after a stop-signal. The task consisted of nine blocks. The first block consisted of a practice phase of 30 trials in which participants received immediate feedback on their performance. The other blocks consisted of 60 trials. Participants received feedback at the end of every block (accuracy, mean reaction time, and mean probability of stopping). In total, there were 160 trials per picture type (neutral, negative or contamination-related) and 48 stop trials per picture type. In the SST only the 40 contamination-related pictures eliciting most anxiety were presented based on the anxiety ratings of the pictures of each participant. Every picture was presented four times.

Dot probe task. The dot probe task (MacLeod, Mathews, & Tata, 1986) was used in order to measure selective attention. The task ran using Inquisit Millisecond 4 software (2015). There were three trial types: contamination-related vs. neutral, negative vs. neutral and neutral vs. neutral. In total there were 204 trials, including 12 practice trials and 64 experimental trials per trial type. In the practice trials participants received immediate feedback.

The trials started with a fixation cross presented in the middle of the screen for 500ms, followed by two pictures (384 x 288 pixels) above and below the fixation cross for 500 ms. Finally, a dot appeared on the same location as one of the pictures, which remained until the participant responded on an AZERTY keyboard. Participants were instructed to press on the "M" key when the dot was below the fixation cross and press

on the "Q" key when the dot was above the fixation cross. Of the contamination-related vs. neutral and negative vs. neutral trials, half were congruent (i.e., the dot appeared on the location of the contamination-related or negative picture) and half were incongruent (i.e., the picture appeared on the location of the neutral picture). The order of the trials was randomized between participants.

In the dot probe task only the 16 contamination-related pictures eliciting most anxiety were presented based on the anxiety ratings of the pictures of each participant. For the neutral-neutral picture pairs another 32 pictures were selected from the IAPS (Lang et al., 1997). Every picture was presented four times.

Mental contamination induction. The selection of the induction of OCD symptoms was based on a meta-analysis (De Putter et al., 2017), in which mental contamination emerged as one of the most effective induction procedures for healthy participants. One of the methods of inducing mental contamination is to induce guilt. Similar to Mancini, Gangemi, Perdighe, and Marini (2008) participants were instructed remember the moment in their in life which they felt extremely guilty and to write this memory down. After they finished writing the story, participants were instructed to close their eyes and to imagine the events as vividly as possible. They were instructed to experience the events again and to focus on the emotions they felt at the time.

Procedure

An overview of the procedure is depicted in Figure 1. The experiment started after participants had read and signed the informed consent form. After the informed consent, the MINI-screen was administered. Subsequently, participants filled out several questionnaires (i.e., DS-R, I_7, OBQ-44, OCI-R, PI-R, Y-BOCS, MASQ-D30). Then participants rated the 60 contamination-related pictures on how much anxiety they elicited. Afterwards the SST and dot probe were administered (in counterbalanced order). After these tasks, participants filled out the mood scales and the DOCS. Subsequently the OCD symptom induction was administered. Change in symptoms after the induction was assessed using the mood scales and DOCS again. The time between the first session and the session during the examination period varied from 68 to 80 days (*M* = 72.65 days, *SD* = 3.33 days). The second session was administered online through Limesurvey and included the OCI-R, PI-R, Y-BOCS. It also included manipulation

check questions such as how much stress they experienced due to the exams, when their last exam took place and when they would have their next exam.

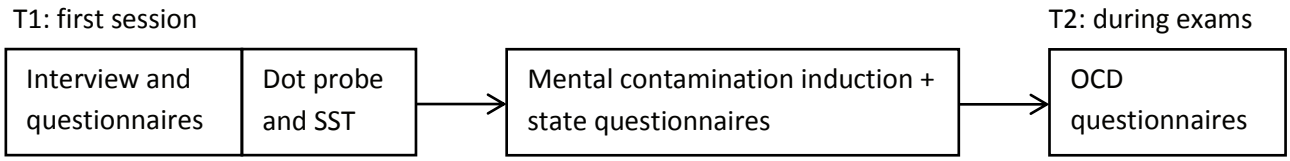

Figure 1. Overview of the procedure. SST = Stop-Signal Task

Statistical Analysis

SPSS (version 20; IBM Corp, 2011) was used in order to analyze the data with a significance level of $p < .05$. In order to estimate the Stop-Signal Reaction Times (SSRTs) of the stop-signal task the integration method was used. In this method the n^{th} reaction time of the distribution of the trials in which there was no stop-signal is equal to the point at which the stop process finishes. In order to determine the n^{th} reaction time, the point in the distribution at which the integral equals the probability of responding after a stop-signal is taken. Subsequently the SSRT was calculated by subtracting the SSD from the finishing time (Verbruggen, Chambers, & Logan, 2013).

In order to prepare the dot probe data, in line with previous research (e.g., Zvielli, Bernstein, & Koster, 2014), all trials with errors and reaction times (RT) faster than 200ms and slower than 1500ms were removed (5.55%). Two participants had an accuracy score below 80% and were excluded from analysis. After exclusion, accuracy was generally high (M = 96.19%, SD = 2.75%, range = 85%-100%). Furthermore, all RTs that differed more than three standard deviation from the sample mean RT per trialtype (i.e., negative congruent, negative incongruent, contamination-related congruent, contamination-related incongruent, and neutral) and from the participant's individual mean per trialtype were removed (1.34%). After data preparation, two participants deviated more than three standard deviations from the average RT of the sample and were excluded from analysis. For the dot probe data traditional attentional bias and attentional bias variability (ABV) were calculated. Attentional bias for contamination-related stimuli was calculated by subtracting mean contamination-related-congruent trials from mean contamination-related-incongruent trials and

attentional bias for negative stimuli by subtracting mean negative-congruent trials from mean negative-incongruent trials. Positive scores for attentional bias refer to attentional bias towards contamination-related or negative stimuli while negative scores for attentional bias refer to attentional bias away from contamination-related or negative stimuli. Finally, ABV was calculated by assessing attentional bias at trial-level by subtracting RT's between temporally contiguous matched trials (incongruent vs. congruent). For this purpose the computation code of Zvielli et al. (2014) was applied.

In order to reduce the chance of Type I errors it was tested whether the dependent variables from the main analyses could be combined into factors. As a first step principal component analysis (PCA) with oblique rotation (direct oblimin) was used in order to check how the added VAS scales should integrate with the VAS scales derived from the POMS (i.e., positive and negative affect). Subsequently, a PCA with oblique rotation (direct oblimin) was done in order to check whether the total scores of the OCD measures and the contamination subscales of the OCI-R and PI-R could be merged. Subsequently, Pearson inter-correlations were run between the study variables at baseline and post-measurement.

In order to test whether measures of selective attention, inhibition for contamination-related stimuli and their interaction could predict change in symptoms after the induction and during the examination period after correcting for baseline symptoms and obsessive beliefs, separate hierarchical linear regressions were conducted with baseline symptoms in step 1, the OBQ-44 in step 2, attentional bias, ABV and SSRT for contamination-related pictures in step 3, and the interactions between the different measures of selective attention and SSRT in step 4. Since we used interaction terms between measures of selective attention and inhibition as predictors, we mean centered the measures of selective attention and inhibition prior to analyses. We tested for multicollinearity by inspecting the variance inflation factor (VIF) and tolerance statistics. After inclusion of all predictors, tolerance statistics varied from .45 to .96 and VIF values from 1.04 to 2.21, suggesting that none of the predictors were problematic (Field, 2009).

Results

Clustering of Scales

As a first step, we tested whether we could combine the dependent variables from the main analyses in order to reduce the chance of Type I error.

Dependent variables induction in the lab. PCA was conducted on the baseline VAS scales energetic, satisfied, happy, angry, tense, depressed, anxious, guilty, disgust, urge to wash, feelings of dirtiness located in the body and dirtiness in general with oblique rotation (direct oblimin). The Kaiser-Meyer-Olkin measure verified the sampling adequacy, KMO = .81. Correlations between items were sufficiently large for PCA (Bartlett's test of sphericity $\chi^2(66) = 554.00$, $p < .001$). Three components had eigenvalues over Kaiser's criterion of 1 and combined explained 67.79% of the variance. The scree plot confirmed three components. Items that cluster on the same components suggest that the first component represents mental contamination (i.e., urge to wash, dirtiness in general, dirtiness located in the body, and disgust), the second component represents positive affect (i.e., energetic, satisfied, and happy) and the third component represents negative affect (i.e., angry, tense, depressed, anxious, and guilty). PCA on the VAS scales post-induction revealed the same results[3]. The reliability of these factors proved acceptable both at baseline and post-induction (range $\alpha = .75$ to $\alpha = .88$). Tables S1 and S2 in the supplementary material show factor loadings after rotation.

Dependent variables naturalistic stress induction. PCA with oblique rotation was conducted on the baseline OCI-R, PI-R and Y-BOCS total scores. The Kaiser-Meyer-Olkin measure verified the sampling adequacy, KMO = .71. Correlations between items were sufficiently large for PCA (Bartlett's test of sphericity $\chi^2(3) = 186.78$, $p < .001$). One component had an eigenvalue over Kaiser's criterion of 1, which was confirmed by the scree plot. The component explained 84.02% of the variance. PCA on the OCI-R, PI-R, and Y-BOCS during the examination period revealed the same results[4]. Based on this

[3] KMO = .82, Bartlett's test of sphericity χ^2 (66) = 551.52, $p < .001$, 69.86% of variance explained by the three components.

[4] KMO = .66, Bartlett's test of sphericity χ^2 (3) = 130.15, $p < .001$, 76.18% of variance explained by the component.

analysis, the OCI-R, PI-R and Y-BOCS total scores were summed together for the next analyses.

Finally, in order to assess effects more specifically for the contamination symptom dimension of OCD a PCA was performed in order to check whether the contamination subscales of the PI-R and OCI-R could be combined. The Kaiser-Meyer-Olkin measure verified the sampling adequacy, KMO = .5. Correlations between items were sufficiently large for PCA (Bartlett's test of sphericity $\chi^2(1) = 120.68$, $p < .001$). One component had an eigenvalue over Kaiser's criterion of 1, which was confirmed by the scree plot. The component explained 93.36% of the variance. PCA on the contamination subscales of the OCI-R and PI-R during the examination period revealed the same results[5]. Based on this analysis, the contamination subscales of the OCI-R and PI-R were summed together for the next analyses.

Sample Characteristics

Means, standard deviations, and correlations between measures at time 1 and time 2 are presented in Table 1. Interestingly, attentional bias and ABV for contamination-related stimuli did not show a significant correlation, indicating that they likely measure different facets of selective attention and can be included in the same analyses. Furthermore, the OBQ-44 correlated significantly with almost all dependent measures at post-measurement (except VAS positive) and almost all dependent variables at baseline (except DOCS baseline). None of the selective attention and inhibition measures correlated significantly with OCD symptoms, except for attentional bias for contamination-related stimuli which correlated significantly with contamination-related OCD symptoms during the examination period. At baseline only inhibition capacity correlated significantly with VAS mental contamination. Only 1 of 89 participants included in the analyses was identified with clinical OCD, while 32 participants were identified with other current mental disorders according to the MINI-screen.

[5] KMO = .50, Bartlett's test of sphericity χ^2 (1) = 84.61, $p < .001$, 89.50% of variance explained by the component.

Table 1.

Intercorrelations, means, and standard deviations among measures at baseline below the diagonal and after OCD induction (DOCS and VAS positive, negative, and mental contamination) or during examination period (general OCD and contamination-related OCD) above the diagonal.

Measure	General OCD T1	Contam OCD T1	DOCS T1	VAS pos T1	VAS neg T1	VAS mental contam T1	OBQ-44	DS-R	I_7	MASQ-D30	SSRT contam	AB contam	ABV contam	Post-measurement	
														M	*SD*
General OCD T2	.80**	.57**	.14	-.23*	.43**	.38**	.57**	.22*	.21*	.39**	-.13	-.08	.03	48.64	34.55
Contam OCD T2	.49**	.76**	.23*	-.10	.16	.40**	.23*	.33**	.24*	.22*	-.09	.22*	.05	6.12	7.47
DOCS T2	.32**	.34**	.41**	-.09	.10	.10	.25*	.03	.23*	.12	.05	.05	-.13	.63	1.20
VAS pos T2	-.32**	-.19	-.12	.60**	-.27*	-.17	-.20	-.29**	.07	-.43**	-.11	-.03	-.06	29.92	19.08
VAS neg T2	.37**	.19	.18	-.32**	.53**	.34**	.35**	.17	.15	.30**	.04	.09	.09	33.90	20.86
VAS mental contam T2	.54**	.47**	.32**	-.29**	.49**	.74**	.44**	.17	.24*	.37**	-.11	-.02	-.01	12.18	15.49
OBQ-44	.73**	.37**	.17	-.33**	.43**	.40**	1.00								
DS-R	.22*	.37**	.25*	-.20	.11	.26*	.27*	-							
I_7	.26*	.29**	.07	.16	-.03	.10	.28**	.18	-						
MASQ-D30	.35**	.21	.16	-.40**	.43**	.28**	.18	.04	-.06	-					
SSRT contam	-.20	-.15	.01	-.13	-.13	-.23*	-.10	-.07	-.02	.05	-				
AB contam	-.13	.18	.16	.01	.07	.02	-.16	.09	-.04	-.04	.03	-			
ABV contam	.04	.05	-.01	-.20	.09	.04	.06	.17	.06	.07	.07	.15	-		
Baseline *M*	55.73	6.57	1.79	49.24	12.86	9.47	145.53	46.21	6.20	28.56	196.99	4.12	83.35		
Baseline *SD*	36.30	8.12	1.98	17.69	12.83	14.24	34.59	13.83	3.98	8.08	40.35	18.69	24.51		

Note. Contam = contamination, pos = positive, neg = negative, DOCS = Dimensional Obsessive-Compulsive Scale**,** DS-R = Disgust Scale-Revised, I_7 = impulsiveness subscale of Impulsiveness–Venturesomeness–Empathy questionnaire, OBQ-44 = Obsessive Beliefs Questionnaire, MASQ-D30 = Mood and Anxiety Symptoms Questionnaire anhedonic depression, SSRT = Stop-Signal Reaction Time, AB = Attentional Bias, ABV = Attentional Bias Variability.

** $p < .01$, * $p < .05$

Predicting Symptoms after Lab Induction

The mean of the corresponding VAS scales was used in order to form the three components (i.e., VAS positive affect, VAS negative affect, and VAS mental contamination). VAS positive affect, VAS negative affect, VAS mental contamination, and DOCS were measured before and after OC symptom induction in the lab. We first tested the hypothesis that cognitive beliefs, selective attention, inhibition, and their interaction in the context of contamination-related stimuli could predict OC symptoms after OC symptom induction in the lab. Specifically, separate linear regressions were conducted for the three components and for the DOCS. Hierarchical linear regression was conducted with baseline VAS or DOCS in step 1, the OBQ-44 in step 2, attentional bias, ABV and SSRT for contamination-related pictures in step 3, and the interactions between the different measures of selective attention and SSRT in step 4. Baseline VAS or DOCS was a significant predictor and explained 17% to 54% of the variance depending on the specific analysis (see Table 2). Interestingly, obsessive beliefs explained a significant amount (3%) of additional variance of VAS mental contamination following the induction after correcting for baseline VAS mental contamination. This effect was specific for VAS mental contamination. Contrary to predictions, inhibition, measures of selective attention, and their interactions did not predict VAS or DOCS scores following the induction after correcting for baseline VAS or DOCS and obsessive beliefs.

Table 2.

Results of linear regression analysis predicting VAS and DOCS following the mental contamination induction.

	VAS positive				VAS negative				VAS mental contamination				DOCS			
	B	*SE B*	β	*p*	*B*	*SE B*	β	*p*	*B*	*SE B*	β	*p*	*B*	*SE B*	β	*p*
Step 1																
Baseline	.64	.09	.60	< .001	.86	.15	.53	< .001	.80	.08	.74	< .001	.25	.06	.41	< .001
Step 2																
OBQ-44	< .01	.05	< .01	.960	.09	.06	.15	.132	.08	.03	.18	.023	.01	< .01	.18	.069
Step 3																
SSRT contam	-.02	.04	-.04	.655	.06	.05	.12	.204	.03	.03	.07	.347	< .01	< .01	.08	.435
AB contam	-.05	.09	-.05	.593	.09	.11	.08	.406	< .01	.06	< .01	.951	< .01	.01	.04	.669
ABV	.06	.07	.07	.417	.01	.08	.02	.861	-.03	.05	-.05	.478	-.01	< .01	-.15	.127
Step 4																
AB x SSRT contam	< .01	< .01	-.04	.649	< .01	< .01	-.02	.870	< .01	< .01	-.05	.509	< .01	< .01	.05	.615
ABV x SSRT contam	< .01	< .01	.09	.360	< .01	< .01	-.04	.641	< .01	< .01	.07	.326	< .01	< .01	-.17	.100

Note. DOCS = Dimensional Obsessive-Compulsive Scale, OBQ-44 = Obsessive Beliefs Questionnaire, contam = contamination, SSRT = Stop-Signal Reaction Time, AB = Attentional Bias, ABV = Attentional Bias Variability.

VAS positive: R^2 = .36 for step 1 (p < .001); Δ R^2 < .01 for step 2 (p = .960); Δ R^2 < .01 for step 3 (p = .798); Δ R^2 < .01 for step 4 (p = .634). VAS negative: R^2 = .28 for step 1 (p < .001); Δ R^2 = .02 for step 2 (p = .132); Δ R^2 = .02 for step 3 (p = .472); Δ R^2 < .01 for step 4 (p = .858). VAS mental contamination: R^2 = .54 for step 1 (p < .001); Δ R^2 = .03 for step 2 (p = .023); Δ R^2 < .01 for step 3 (p = .727); Δ R^2 < .01 for step 4 (p = .559). DOCS: R^2 = .17 for step 1 (p < .001); Δ R^2 = .03 for step 2 (p = .069); Δ R^2 = .03 for step 3 (p = .418); Δ R^2 = .03 for step 4 (p = .256).

Predicting Symptoms after Naturalistic Stress Induction

We tested the hypothesis that cognitive beliefs, selective attention, inhibition, and their interaction in the context of contamination-related stimuli could prospectively predict OC symptoms during the examination period. Specifically, a separate linear regression was performed for general OCD symptoms (i.e., sum score of PI-R, OCI-R, and Y-BOCS) and OCD symptoms of the contamination symptom dimension (i.e., sum score of washing subscales of PI-R and OCI-R). Hierarchical linear regression was conducted with general or contamination OCD symptoms at baseline in step 1, the OBQ-44 in step 2, attentional bias, ABV and SSRT for contamination-related pictures in step 3, and the interactions between the different measures of selective attention and SSRT in step 4 (see Table 3).

Table 3.

Results of linear regression analysis predicting OCD symptoms at follow-up during the examination period.

	General OCD				Contamination OCD			
	B	*SE B*	β	*p*	*B*	*SE B*	β	*p*
Step 1								
Baseline OCD symptoms	.76	.06	.80	< .001	.70	.06	.76	< .001
Step 2								
OBQ-44	-.03	.09	-.03	.783	-.01	.02	-.06	.417
Step 3								
SSRT contam	.03	.06	.03	.607	< .01	.01	.02	.802
AB contam	.05	.12	.02	.713	.03	.03	.07	.370
ABV contam	< .01	.09	< .01	.992	< .01	.02	.00	.981
Step 4								
AB x SSRT contam	< .01	< .01	-.03	.662	< .01	< .01	.08	.300
ABV x SSRT contam	< .01	< .01	.14	.038	< .01	< .01	.15	.042

Note. OBQ-44 = Obsessive Beliefs Questionnaire, contam = contamination, SSRT = Stop-Signal Reaction Time, AB = Attentional Bias, ABV = Attentional Bias Variability.

General OCD symptoms: R^2 = .64 for step 1 (p < .001); Δ R^2 < .01 for step 2 (p = .783); Δ R^2 < .01 for step 3 (p = .938); Δ R^2 = .02 for step 4 (p = .113). Contamination OCD symptoms: R^2 = .57 for step 1 (p < .001); Δ R^2 < .01 for step 2 (p = .417); Δ R^2 < .01 for step 3 (p = .823); Δ R^2 = .034 for step 4 (p = .034).

Baseline OCD symptoms were a significant predictor and explained most of the variance in both analyses. Contrary to Abramowitz et al. (2006), obsessive beliefs did not predict symptoms during the examination period. Likewise, inhibition and measures of selective attention did not predict symptoms. Interestingly, the interaction between ABV and SSRTs for contamination-related stimuli explained a significant amount (3.4%) of additional variance of contamination OCD symptoms during the examination period after correcting for baseline symptoms. Adding the interaction term did not result in a significant improvement of the model for general OCD symptoms. Therefore, the effect of the interaction between ABV and inhibition for contamination-related stimuli seems more specific for predicting contamination OCD symptoms.

In order to further interpret the significant ABV x SSRT interaction a moderation model was tested with ABV as the moderator of effect X on contamination symptoms during the examination period as Y and SSRT as M using Process (command model 1; Hayes, 2012). After controlling for heteroscedasticity, the conditional effect of X on Y at different values of the moderator showed that when SSRT for contamination-related stimuli was low (i.e., good inhibitory functioning) or average there was no effect of ABV on contamination symptoms during the examination period (SSRT low: B = -0.05, $SE\ B$ = .03, $t(81)$ = -1.38, p = .170; SSRT average: B < .01, $SE\ B$ = .02, $t(81)$ = -0.03, p = .979). However, when SSRT for contamination-related stimuli was high (i.e., poor inhibitory functioning) the effect of ABV on contamination symptoms during the examination period was significant (B = 0.05, $SE\ B$ = .02, $t(81)$ = 1.99, p = .049). Therefore, it was only when participants showed poor inhibitory functioning in the context of contamination-related pictures that larger attentional bias variability in the context of contamination-related pictures predicted contamination OCD symptoms prospectively.

Discussion

The current study set out to investigate whether obsessive beliefs, selective attention, inhibition, and the interaction between selective attention and inhibition can prospectively predict OCD symptoms. Obsessive beliefs, inhibition, and selective attention were assessed in a student sample during a baseline session in the beginning of the first semester. Within this baseline session an OCD symptom induction was administered consisting of a mental contamination induction. The examination period

was used as an ecologically valid stress induction, in which OCD symptoms were assessed again 68 to 80 days after baseline. The main results were that there was no predictive value of inhibition capacity, attentional bias or attentional bias variability in the context of contamination-related stimuli over and above baseline symptoms for OCD symptoms after an induction in the lab or during the examination period. This finding is in line with other studies that do not find an association between inhibition capacity or selective attention and OCD (e.g., Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Harkness et al., 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008; Rasmussen, Siev, Abramovitch, & Wilhelm, 2016). Moreover, obsessive beliefs were only a significant predictor for a change in state feelings of mental contamination after the induction. Participants with more obsessive beliefs reported more feelings of mental contamination after the induction. As we did not consistently find that obsessive beliefs predicted symptoms, these results question the predictive value of obsessive beliefs for OCD symptoms. This finding is in line with other studies that do not find a consistent predictive effect of obsessive beliefs on OCD symptoms (e.g., Coles et al., 2008; Novara et al., 2011).

Interestingly, specifically for contamination OCD symptoms, there was a significant effect of the interaction between inhibition capacity and attentional bias variability in the context of contamination-related pictures after controlling for the other variables. The model including the interaction terms accounted for 3.4% of additional explained variance above the other models. There was only an effect of attentional bias variability on contamination OCD symptoms during the examination period if inhibitory functioning in the context of contamination-related pictures was poor. This suggests that inhibition capacity and attentional bias variability interact to predict contamination OCD symptoms. Having poor inhibitory functioning or a large attentional bias variability in the context of contamination-related pictures only made participants more vulnerable for contamination OCD symptoms when both were present at the same time. This result supports the merit of looking at interactions between factors as suggested by Muller and Roberts (2005) and the combined cognitive biases hypothesis of Hirsch et al. (2006). Future research is warranted in order to investigate whether these effects extend to interactive effects between other

information processing factors such as task switching and visual memory and interactive effects between obsessive beliefs and information processing factors.

We consider this study as an important step towards more comprehensive prediction models of OCD where examining effects on lab stressors as well as real life is crucial. At present it is unclear why the effects observed with regard to the real-life stressor were not observed in the lab. Potential reasons can be the differences between the type of stressor, intervening influences of other life events, or resilience factors that reduced the impact of problematic information-processing factors. To further assess the clinical influence of combined risk factors it would be advantageous to match lab and real-life stressors more closely.

There are several other limitations to this study. First, OCD symptoms were investigated in a convenience sample of undergraduate students. However, this study did not investigate clinical OCD, but rather mechanisms through which OCD symptoms could develop. The utility of using analogue samples in the study of mechanisms in OCD has been demonstrated elegantly by Abramowitz et al. (2014) and Gibbs (1996). Second, the follow-up period varied between 68 to 80 days. It is possible that different mechanisms predict OCD symptoms at a longer time period of six months. Third, it would be interesting to check whether obsessive beliefs interact with inhibition capacity or selective attention. However, our sample size rendered such analyses underpowered.

Limitations notwithstanding, to our knowledge this study is the first to investigate the interactive effects of inhibition capacity and selective attention prospectively. Moreover, this study went beyond the traditional attentional bias scores, which characterizes attentional bias as a stable concept, and included attentional bias variability, a measure of attentional bias as a dynamic process in time. In contrast to the traditional attentional bias scores, attentional bias variability has shown good to excellent reliability (Rodebaugh et al., 2016; Zvielli, Bernstein, & Koster, 2015). Moreover, we used idiosyncratic stimuli which is considered an important methodological aspect for OCD research (Muller & Roberts, 2005).

To conclude, obsessive beliefs did not consistently predict OCD symptoms after an OCD symptom induction or during the examination period. There was no predictive value of attentional bias, attentional bias variability and inhibition capacity in the

context of contamination-related stimuli in isolation. However, attentional bias variability and inhibition capacity in the context of contamination-related stimuli did interact to predict contamination OCD symptoms during the examination period. A large attentional bias variability and poor inhibition capacity proved to be a toxic combination and predicted contamination OCD symptoms during a period of increased stress (i.e., the examination period). These results support the notion that information processing biases act in concert rather than in isolation in predicting contamination OCD symptoms.

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References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163-1171. doi: 10.1016/j.cpr.2013.09.004

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., . . . Hale, L. R. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the dimensional obsessive-compulsive scale. *Psychological Assessment, 22*(1), 180-198. doi: 10.1037/a0018260

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

Abramowitz, J. S., Khandker, M., Nelson, C. A., Deacon, B. J., & Rygwall, R. (2006). The role of cognitive factors in the pathogenesis of obsessive–compulsive symptoms: A prospective study. *Behaviour Research and Therapy, 44*(9), 1361-1374. doi: 10.1016/j.brat.2005.09.011

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive–compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157. doi: 10.1016/j.brat.2008.10.020

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Baer, L. (1991). *Getting control: Overcoming your obsessions and compulsions*. New York, NY: Penguin.

Ball, S. G., Baer, L., & Otto, M. W. (1996). Symptom subtypes of obsessive-compulsive disorder in behavioral treatment studies: A quantitative review. *Behaviour Research and Therapy, 34*(1), 47-51. doi: 10.1016/0005-7967(95)00047-2

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive–compulsive disorder: State or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11-12), 1031-1038. doi: 10.1080/j.1440-1614.2006.01928.x

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive–compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141-150. doi: 10.1016/j.jpsychires.2006.11.008

Broderick, J., Grisham, J. R., & Weidemann, G. (2013). Disgust and fear responding in contamination-based obsessive-compulsive disorder during pictorial exposure. *Behavior Therapy, 44*(1), 27-38. doi: 10.1016/j.beth.2012.05.005

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience & Biobehavioral Reviews, 29*(3), 399-419. doi: 10.1016/j.neubiorev.2004.11.006

Coles, M. E., & Horng, B. (2006). A prospective test of cognitive vulnerability to obsessive-compulsive disorder. *Cognitive Therapy and Research, 30*(6), 723-734. doi: 10.1007/s10608-006-9033-x

Coles, M. E., Pietrefesa, A. S., Schofield, C. A., & Cook, L. M. (2008). Predicting changes in obsessive compulsive symptoms over a six-month follow-up: A prospective test of cognitive models of obsessive compulsive disorder. *Cognitive Therapy and Research, 32*(5), 657-675. doi: 10.1007/s10608-007-9132-3

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201-215.

Coskunpinar, A., & Cyders, M. A. (2013). Impulsivity and substance-related attentional bias: A meta-analytic review. *Drug and Alcohol Dependence, 133*(1), 1-14. doi: 10.1016/j.drugalcdep.2013.05.008

De Putter, L. M. S., Van Yper, L., & Koster, E. H. W. (2017). Obsessions and compulsions in the lab: A meta-analysis of procedures to induce symptoms of obsessive-compulsive disorder. *Clinical Psychology Review, 52*, 137-147. doi: 10.1016/j.cpr.2017.01.001

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353. doi: 10.1037/1528-3542.7.2.336

Eysenck, S. B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*(5), 613-619. doi: 10.1016/0191-8869(85)90011-x

Field, A. (2009). *Discovering statistics using SPSS* (third ed.). London, UK: Sage.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment, 14*(4), 485-496. doi: 10.1037/1040-3590.14.4.485

Foa, E. B., Kozak, M. J., Salkovskis, P. M., Coles, M. E., & Amir, N. (1998). The validation of a new obsessive–compulsive disorder scale: The Obsessive–Compulsive Inventory. *Psychological Assessment, 10*(3), 206-214. doi: 10.1037/1040-3590.10.3.206

Gibbs, N. A. (1996). Nonclinical populations in research on obsessive-compulsive disorder: A critical review. *Clinical Psychology Review, 16*(8), 729-773. doi: 10.1016/S0272-7358(96)00043-8

Haidt, J., McCauley, C., & Rozin, P. (1994). Individual differences in sensitivity to disgust: A scale sampling seven domains of disgust elicitors. *Personality and Individual Differences, 16*(5), 701-713. doi: 10.1016/0191-8869(94)90212-7

Hajcak, G., Huppert, J. D., Simons, R. F., & Foa, E. B. (2004). Psychometric properties of the OCI-R in a college sample. *Behaviour Research and Therapy, 42*(1), 115-123. doi: 10.1016/j.brat.2003.08.002

Harkness, E. L., Harris, L. M., Jones, M. K., & Vaccaro, L. (2009). No evidence of attentional bias in obsessive compulsive checking on the dot probe paradigm. *Behaviour Research and Therapy, 47*(5), 437-443. doi: 10.1016/j.brat.2009.02.004

Hayes, A. F. (2012). *PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling* [White paper]. Retrieved from http://www.afhayes.com/public/process2012.pdf

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223-236. doi: 10.1016/j.beth.2006.02.001

Hou, R. H., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite, 56*(2), 424-427. doi: 10.1016/j.appet.2011.01.019

IBM Corp. (2011). IBM SPSS statistics for windows, version 20.0. Armonk, NY: IBM Corp.

Inquisit 4 [Computer software]. (2015). Retrieved from http://www.millisecond.com.

Lang, P. J., Bradley, M. M., & Cuthbert, B. (1997). *International affective picture system (IAPS): Instruction manual and affective ratings.* University of Florida, NIMH Center for the Study of Emotion and Attention, Gainesville, FL, USA.

Lavy, E., Van Oppen, P., & Van Den Hout, M. (1994). Selective processing of emotional information in obsessive compulsive disorder. *Behaviour Research and Therapy, 32*(2), 243-246. doi: 10.1016/0005-7967(94)90118-X

Levitt, H. (1971). Transformed up-down methods in psychoacoustics. *The Journal of the Acoustical Society of America, 49*(2B), 467-477. doi: 10.1121/1.1912375

Lijffijt, M., Caci, H., & Kenemans, J. L. (2005). Validation of the Dutch translation of the I7 questionnaire. *Personality and Individual Differences, 38*(5), 1123-1133. doi: 10.1016/j.paid.2004.07.010

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory and language*. San Diego, CA: Academic Press.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*(1), 15-20. doi: 10.1037/0021-843X.95.1.15

Mancini, F., Gangemi, A., Perdighe, C., & Marini, C. (2008). Not just right experience: Is it influenced by feelings of guilt? *Journal of Behavior Therapy and Experimental Psychiatry, 39*(2), 162-176. doi: 10.1016/j.jbtep.2007.02.002

Markarian, Y., Larson, M. J., Aldea, M. A., Baldwin, S. A., Good, D., Berkeljon, A., . . . McKay, D. (2010). Multiple pathways to functional impairment in obsessive–compulsive disorder. *Clinical Psychology Review, 30*(1), 78-88. doi: 10.1016/j.cpr.2009.09.005

Mataix-Cols, D., Lawrence, N. S., Wooderson, S., Speckens, A., & Phillips, M. L. (2009). The Maudsley Obsessive–Compulsive Stimuli Set: Validation of a standardized paradigm for symptom-specific provocation in obsessive–compulsive disorder. *Psychiatry Research, 168*(3), 238-241. doi: 10.1016/j.psychres.2008.05.007

McDermott, L. M., & Ebmeier, K. P. (2009). A meta-analysis of depression severity and cognitive function. *Journal of Affective Disorders, 119*(1–3), 1-8. doi: 10.1016/j.jad.2009.04.022

McNair, D. M., Lorr, M., & Dropplemann, L. F. (1992). *Manual: Profile of mood states revised*. San Diego, CA: Educational and Industrial Testing Service.

Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C.-H., del Campo, N., . . . Bullmore, E. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*(12), 3223-3236. doi: 10.1093/brain/awm205

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Moritz, S., Fischer, B.-K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., & Jelinek, L. (2008). Words may not be enough! No increased emotional Stroop effect in obsessive–compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104. doi: 10.1016/j.brat.2008.05.005

Moritz, S., Von Muehlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive–compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371. doi: 10.1017/S1355617709090511

Moritz, S., & von Mühlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229. doi: 10.1002/da.20294

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive–Compulsive Disorder: a review. *Journal of Anxiety Disorders, 19*(1), 1-28. doi: 10.1016/j.janxdis.2003.12.001

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive-compulsive

symptoms. *Journal of Abnormal Psychology, 119*(1), 136-142. doi: 10.1037/a0017549

Novara, C., Pastore, M., Ghisi, M., Sica, C., Sanavio, E., & McKay, D. (2011). Longitudinal aspects of obsessive compulsive cognitions in a non-clinical sample: A five-year follow-up study. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(3), 317-324. doi: 10.1016/j.jbtep.2011.01.007

OCCWG. (1997). Cognitive assessment of obsessive-compulsive disorder. *Behaviour Research and Therapy, 35*(7), 667-681. doi: 10.1016/S0005-7967(97)00017-X

OCCWG. (2005). Psychometric validation of the obsessive belief questionnaire and interpretation of intrusions inventory—Part 2: Factor analyses and testing of a brief version. *Behaviour Research and Therapy, 43*(11), 1527-1542. doi: 10.1016/j.brat.2004.07.010

Olatunji, B. O., Moretz, M. W., McKay, D., Bjorklund, F., de Jong, P. J., Haidt, J., . . . Schienle, A. (2009). Confirming the three-factor structure of the Disgust Scale—Revised in eight countries. *Journal of Cross-Cultural Psychology, 40*(2), 234-255. doi: 10.1177/0022022108328918

Olatunji, B. O., Williams, N. L., Tolin, D. F., Abramowitz, J. S., Sawchuk, C. N., Lohr, J. M., & Elwood, L. S. (2007). The Disgust Scale: Item analysis, factor structure, and suggestions for refinement. *Psychological Assessment, 19*(3), 281-297. doi: 10.1037/1040-3590.19.3.281

Presentation®, version 17.2, Neurobehavioral Systems, Inc., Berkeley, CA, www.neurobs.com

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy, 35*(9), 793-802. doi: 10.1016/S0005-7967(97)00040-5

Rachman, S. (1998). A cognitive theory of obsessions: Elaborations. *Behaviour Research and Therapy, 36*(4), 385-401. doi: 10.1016/S0005-7967(97)10041-9

Rachman, S. (2004). Fear of contamination. *Behaviour Research and Therapy, 42*(11), 1227-1255. doi: 10.1016/j.brat.2003.10.009

Rasmussen, J., Siev, J., Abramovitch, A., & Wilhelm, S. (2016). Scrupulosity and contamination OCD are not associated with deficits in response inhibition. *Journal of Behavior Therapy and Experimental Psychiatry, 50*, 120-126. doi: 10.1016/j.jbtep.2015.06.004

Rodebaugh, T. L., Scullin, R. B., Langer, J. K., Dixon, D. J., Huppert, J. D., Bernstein, A., . . . Lenze, E. J. (2016). Unreliability as a threat to understanding psychopathology: The cautionary tale of attentional bias. *Journal of Abnormal Psychology, 125*(6), 840-851. doi: 10.1037/abn0000184

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/mp.2008.94

Salkovskis, P. M. (1985). Obsessional-compulsive problems: A cognitive-behavioural analysis. *Behaviour Research and Therapy, 23*(5), 571-583. doi: 10.1016/0005-7967(85)90105-6

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., . . . Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (MINI): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry, 59*, 22-33.

Steketee, G., Frost, R., & Bogart, K. (1996). The Yale-Brown Obsessive Compulsive Scale: Interview versus self-report. *Behaviour Research and Therapy, 34*(8), 675-684. doi: 10.1016/0005-7967(96)00036-8

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in Obsessional Compulsive Disorder. *Behaviour Research and Therapy, 34*(1), 53-60. doi: 10.1016/0005-7967(95)00041-U

Van Oppen, P., Hoekstra, R. J., & Emmelkamp, P. M. G. (1995). The structure of obsessive-compulsive symptoms. *Behaviour Research and Therapy, 33*(1), 15-23. doi: 10.1016/0005-7967(94)E0010-G

Verbruggen, F., Chambers, C. D., & Logan, G. D. (2013). Fictitious inhibitory differences: How skewness and slowing distort the estimation of stopping latencies. *Psychological Science, 24*(3), 352-362. doi: 10.1177/0956797612457390

Verbruggen, F., & De Houwer, J. (2007). Do emotional stimuli interfere with response inhibition? Evidence from the stop signal paradigm. *Cognition and Emotion, 21*(2), 391-403. doi: 10.1080/02699930600625081

Wardenaar, K. J., van Veen, T., Giltay, E. J., de Beurs, E., Penninx, B. W. J. H., & Zitman, F. G. (2010). Development and validation of a 30-item short adaptation of the

Mood and Anxiety Symptoms Questionnaire (MASQ). *Psychiatry Research, 179*(1), 101-106. doi: 10.1016/j.psychres.2009.03.005

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*(1), 15-25. doi: 10.1037/0021-843X.104.1.15

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 3-14. doi: 10.1037/0021-843X.104.1.3

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2014). Dynamics of attentional bias to threat in anxious adults: Bias towards and/or away? *PLoS ONE, 9*(8), e104025. doi: 10.1371/journal.pone.0104025

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2015). Temporal dynamics of attentional bias. *Clinical Psychological Science, 3*(5), 772–788. doi: 10.1177/2167702614551572

Supplementary Material

Table S1.

Factor loadings based on a principal components analysis with oblimin rotation for baseline VAS scales (*N* = 89)

	Positive affect	Negative affect	Mental contamination	Communality
Urge to wash			.95	.80
Dirtiness general			.86	.84
Dirtiness located in the body			.81	.86
Disgust			.55	.57
Happy	.86			.77
Energetic	.79			.62
Satisfied	.74			.70
Angry		.78		.53
Anxious		.72		.71
Depressed		.68		.59
Guilty		.62		.73
Tense		.51		.40

Note. Factor loadings < .4 are suppressed

Table S2.

Factor loadings based on a principal components analysis with oblimin rotation for post-induction VAS scales (*N* = 89)

	Positive affect	Negative affect	Mental contamination	Communality
Urge to wash			.96	.83
Dirtiness general			.88	.81
Dirtiness located in the body			.80	.74
Disgust			.48	.51
Happy	.73			.79
Energetic	.87			.70
Satisfied	.78			.82
Angry		.84		.63
Anxious		.78		.66
Depressed		.54		.58
Guilty		.77		.69
Tense		.66		.61

Note. Factor loadings < .4 are suppressed

CHAPTER 6

GENERAL DISCUSSION

Recapitulation of the Research Aims

To date, research regarding the role of response inhibition and selective attention in OCD is characterized by substantial heterogeneity, which demonstrates the need for more research on moderators explaining this heterogeneity. The current doctoral dissertation set out to examine three research aims in order to further clarify the link between selective attention, response inhibition, and OCD symptoms.

The first aim was to investigate whether the effects of response inhibition and selective attention in the context of OCD were stable or context-dependent. Within response inhibition there has been a long standing *state-trait debate*. Proponents of the trait view argue underperformance in inhibition is an endophenotype of OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). In contrast, proponents of the state view argue that inhibition capacity can be influenced by current symptoms (Abramovitch, Dar, Hermesh, & Schweiger, 2012). In the context of selective attention and OCD there has also been discussion in the literature regarding the nature of this relationship. Van Bockstaele et al. (2014) and Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, and van Ijzendoorn (2007) argue that selective attention is a vulnerability factor for anxiety. However, Van Bockstaele et al. (2014) also argue that symptoms may influence attention as well. While there is a wealth of research on the influence of selective attention on OCD, there is little research on the influence of OCD symptoms on selective attention.

The second aim was to examine whether the effects of response inhibition and selective attention in the context of OCD were general or valence-specific. The Attentional Control Theory (ACT; Eysenck, Derakshan, Santos, & Calvo, 2007) predicts that threat-related distractors will impair efficiency on tasks involving inhibition. This implies that underperformance on inhibition could be specific for disorder-relevant

stimuli compared to neutral stimuli. However, to date, there is little research on the valence-specificity of response inhibition. Based on the literature on OCD and selective attention, it is still unclear whether OCD symptoms are associated with a bias to negative stimuli in general or specifically to disorder-relevant stimuli. Pergamin-Hight, Naim, Bakermans-Kranenburg, van Ijzendoorn, and Bar-Haim (2015) found that in anxiety, attentional bias was specific for disorder-relevant stimuli over generally negative stimuli, however this meta-analysis only included four studies on OCD with mixed results. If an attentional bias is specific for disorder-relevant stimuli over generally negative stimuli, this would imply that selective attention is affected by previous learning and memory.

Finally, the third aim was to investigate whether OCD symptoms were best predicted by single or multiple information processing biases. Muller and Roberts (2005) and Hirsch, Clark, and Mathews (2006) pose that information processing biases could interact and therefore have a greater impact on disorders than the information processing factors in isolation. Attentional Control Theory (Eysenck et al., 2007) poses that bottom-up capture and top-down control bidirectionally influence each other. Applied to OCD, the bottom-up system (e.g., selective attention) could interact with top-down control (e.g., response inhibition capacity) in the maintenance and development of OCD symptoms. For instance, it is plausible that selective attention towards threat-related stimuli in combination with difficulties in response inhibition after threat-related stimuli exacerbate OCD symptoms.

Integration of the Main Findings

Stable versus Context-Dependent Response Inhibition and Selective Attention

As a first step in the investigation of whether response inhibition and selective attention are stable or context-dependent in the context of OCD, a meta-analysis was conducted on existing methods of OCD symptom inductions in chapter 2. Based on this meta-analysis mental contamination was found as one of the most potent induction procedures of OCD symptoms in nonclinical participants (Hedges's g = 0.8). Therefore, in the next studies inductions based on mental contamination were used in order to examine the effect of current OCD symptoms.

In chapter 3 the influence of current symptoms was investigated in the context of response inhibition. In this study the performance on a stop signal task of participants scoring high on contamination fear (HCF) was compared to participants scoring low on contamination fear (LCF). The stop signal task was administered before and after either a neutral induction or an OCD symptom induction. If underperformance in response inhibition is dependent on current symptoms, we would expect performance to deteriorate after an OCD symptom induction compared to a neutral induction. Although the OCD symptom induction was largely successful, this study showed no effect of an OCD symptom induction on response inhibition capacity. If underperformance in response inhibition would be a stable trait that makes someone more vulnerable to develop OCD, we would expect (1) that LCF would outperform HCF, (2) no effect of an OCD symptom induction, and (3) that baseline response inhibition capacity could predict an increase in symptoms after an OCD symptom induction. Although there was indeed no effect of an OCD symptom induction, baseline response inhibition capacity did not predict an increase in symptoms after an OCD symptom induction. More importantly, in contrast to the view of underperformance in inhibition being stable in OCD, participants with subclinical levels of OCD actually marginally outperformed participants scoring low on OCD. This finding is contrary to meta-analyses that find a deficit in inhibition in OCD compared to healthy controls (Abramovitch, Abramowitz, & Mittelman, 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2014). However, Leopold and Backenstrass (2015) showed that neuropsychological functioning can differ between symptom dimensions. In their meta-analysis they found that participants from the contamination symptom dimension generally outperformed participants from the checking symptom dimension. Therefore, our choice to focus on the contamination symptom dimension could explain this dissonance with other results in the literature.

In chapter 4 stability versus context-dependence was examined in selective attention and OCD in two studies. In the first study HCF was compared to LCF on selective attention towards safety and threat. If selective attention towards threat is stable, we would expect more selective attention in subclinical participants compared to participants scoring low on OCD symptoms especially to contamination-related stimuli. Instead, all participants showed selective attention towards contamination-

related stimuli regardless of group. The lack of a group difference in selective attention is in line with other studies that did not find an association between selective attention and OCD symptoms (e.g., Harkness, Harris, Jones, & Vaccaro, 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008), although some other studies did find an association between OCD and selective attention (e.g., Amir, Najmi, & Morrison, 2009; Moritz, Von Muehlenen, Randjbar, Fricke, & Jelinek, 2009; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996). In the second study the effect of current OCD symptoms was examined by administering a dot probe task before and after an OCD symptom induction. If selective attention would be context-dependent, we would expect increased selective attention towards OCD-related stimuli after an OCD symptom induction compared to a neutral induction. Although the OCD symptom induction was successful, in contrast to Cohen, Lachenmeyer, and Springer (2003) who found decreased performance on a Stroop task after an OCD symptom induction, there was no effect on subsequent selective attention. If selective attention would influence OCD symptoms, we would expect that selective attention would be able to predict the increase in symptoms after an OCD symptom induction. Instead, we found no predictive effect of baseline selective attention on OCD symptoms after an OCD symptom induction.

Conclusion. Both in response inhibition and selective attention we found no effect of current OCD symptoms on performance as there was no effect of an OCD symptom induction. Besides the absence of an effect of current symptoms, there was also no evidence for an association between stable selective attention or response inhibition and OCD symptoms. These baseline information processing factors were not able to predict an increase in symptoms after an induction and there was either no association between subclinical OCD and performance (in selective attention) or subclinical OCD performed better (in response inhibition). These results question the role of response inhibition and selective attention in OCD for subclinical OCD.

General versus Content-Specific Response Inhibition and Selective Attention

The second research aim consisted of testing whether the effects of response inhibition and selective attention were content-specific (i.e., only for OCD-related stimuli) or general. In chapter 3 the content-specificity of response inhibition was

investigated by administering an adapted stop signal task including neutral, negative and OCD-related pictures. Contrary to predictions, there was no effect of OCD-related stimuli, yet there was an effect of generally negative stimuli compared to neutral stimuli. In contrast to Verbruggen and De Houwer (2007), participants showed better response inhibition capacity in the context of negative stimuli compared to neutral stimuli. This contrast could be due to the nature of our negative stimuli. In order to be able to match our OCD-related and negative stimuli on arousal, our final selection of negative stimuli were characterized by a higher overall valence (mean valence = 3.26 compared to valence = 2.41 in Verbruggen & De Houwer, 2007) and lower overall arousal (mean arousal = 4.10 compared to arousal = 6.16 in Verbruggen & De Houwer, 2007). According to Pessoa (2009) emotional items that are relatively low in threat would enhance cognitive performance, while emotional items that are relatively high in threat, would impair cognitive performance. This could be an explanation for the contrast with Verbruggen and De Houwer (2007). Indeed, Pessoa, Padmala, Kenzer, and Bauer (2012) found that mild emotional material enhanced inhibition whereas strong emotional stimuli impaired inhibition capacity. The fact that we did not find an effect of OCD-related stimuli is partly in line with Linkovski, Kalanthroff, Henik, and Anholt (2016) who found no effect of OCD-related stimuli on stop signal *reaction times*. They did find an effect of OCD-related stimuli on accuracy in stopping trials. However, we did not analyse stopping accuracy as in our study this accuracy was artificially held around 50% by adapting the stop signal delay dependent on performance in order to ensure the calculation of valid stop signal reaction times.

In chapter 4 the content-specificity of selective attention was investigated by two studies. The first study compared selective attention towards threat to selective attention towards safety (i.e., cleanliness). As expected, participants showed more selective attention towards threat compared to safety, however this effect did not differ for HCF or LCF. In the second study we compared selective attention towards OCD-related stimuli to generally negative stimuli. Only for attentional interference there was a significant difference between OCD-related and negative stimuli. In line with a meta-analysis on content-specificity of selective attention (Pergamin-Hight et al., 2015), participants showed more interference after OCD-related stimuli compared to generally negative stimuli. However, this effect faded with time. Similarly, Morein-Zamir

et al. (2013) found selective attention towards idiosyncratic OCD-related stimuli in nonanxious participants.

Conclusion. There was an effect of increased attentional interference of OCD-related stimuli compared to generally negative stimuli. However, this effect was temporary and did not generalize to other measures of selective attention. Similarly, for response inhibition there was no significant difference in performance after OCD-related stimuli compared to negative stimuli. Taken together, these results question the role of content-specificity in information processing in the context of OCD.

The Effects of Combined Response Inhibition and Selective Attention

The predictive value of combined selective attention towards threat and response inhibition on OCD symptoms was investigated in chapter 5. In a baseline session selective attention, response inhibition and OCD symptoms were assessed at the beginning of the semester. In order to determine the predictive value of selective attention and response inhibition on OCD symptoms, an OCD symptom induction was administered in the lab. As a more naturalistic stressor, OCD symptoms were also assessed during the examination period (68 to 80 days later). Response inhibition after OCD-related stimuli and selective attention towards OCD-related stimuli neither alone nor their interaction predicted OCD symptoms following the OCD symptom induction in the lab, after correcting for baseline symptoms and obsessive beliefs. There was also no significant predictive effect of these factors after correcting for baseline symptoms and obsessive beliefs on general OCD symptoms during the examination period. The lack of an association between response inhibition, selective attention and OCD is in line with other studies finding no such association (e.g., Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Harkness et al., 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008; Rasmussen, Siev, Abramovitch, & Wilhelm, 2016). However, adding the interaction between response inhibition after OCD-related stimuli and selective attention towards OCD-related stimuli did significantly improve the predictive model for contamination fear OCD symptoms during the examination period after correcting for baseline symptoms, obsessive beliefs and the information processing factors in isolation. This effect was specific for the interaction between attentional bias variability (ABV) and response inhibition. It was only when participants

showed poor inhibitory functioning after OCD-related stimuli that ABV in the context of OCD-related stimuli prospectively predicted contamination OCD symptoms. This finding supports the merit of looking at interactions between information processing factors in the context of OCD rather than information processing factors in isolation (Hirsch et al., 2006; Muller & Roberts, 2005).

Conclusion. Neither response inhibition nor selective attention towards OCD-related stimuli was able to predict OCD symptoms after a lab stressor or naturalistic stressor in isolation. The interaction between response inhibition and ABV in the context of OCD-related stimuli significantly predicted additional variance in contamination OCD symptoms experienced during a naturalistic stressor (i.e., examination period). ABV only predicted contamination OCD symptoms when response inhibition was poor. This finding is in line with the combined cognitive biases hypothesis (Hirsch et al., 2006).

Implications

Theoretical Implications

The findings in this doctoral dissertation have several theoretical implications. Within theories on inhibition and OCD, our findings did not support the executive overload model (Abramovitch et al., 2012). This model poses that an overflow of obsessive thoughts consumes cognitive resources, which leads to an overload of the executive system and subsequent impairments in inhibition capacity. This will lead to fear of impulsivity when these impairments become evident for the OCD patient. In order to cope with the fear of impulsivity, OCD patients will increase their efforts to control automatic processes. This increased control of automatic processes will subsequently lead to fronto-striatal hyperactivation and more overflow of obsessive thoughts, making the vicious cycle complete. In our results we found no effects of induced OCD symptoms on inhibition, which we would have expected in the case of executive overload. This could imply that the executive overload model only applies to clinical OCD or executive functions other than response inhibition. However, Abramovitch, Shaham, Levin, Bar-Hen, and Schweiger (2015) did find decreased response inhibition in subclinical participants compared to participants scoring low on OCD. This finding suggests that effects on response inhibition are not limited to clinical

OCD patients. As OCD symptom severity was not a consistent moderator for neuropsychological performance (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014) and in our studies we failed to find an effect of an induction of OCD symptoms on subsequent response inhibition performance, it is also plausible that obsessive thoughts do not result in an overload of the executive system as the executive overload theory poses.

Other authors have considered inhibition as an *endophenotype* of OCD (e.g., Chamberlain et al., 2005). This implies that underperformance in inhibition would function as a genetic risk factor. In the results of this doctoral dissertation we found mixed evidence for this theory. On the one hand there was no effect of current symptoms on response inhibition. Moreover, in interaction with selective attention response inhibition capacity was able to predict contamination symptoms during a naturalistic stressor. However, in contrast to the endophenotype view, participants scoring high on contamination fear actually performed slightly better on response inhibition and response inhibition did not predict OCD symptoms when considered in isolation. Consequently, based on the results of this dissertation, no definitive conclusions can be drawn regarding the endophenotype view in OCD.

Within selective attention Bar-Haim et al. (2007) have proposed the integrative model. This model includes four stages of threat processing. First, stimuli in the environment are evaluated pre-attentively. Then, when stimuli are labeled as a threat, cognitive resources are allocated to the threat stimuli. Subsequently, in a conscious anxious state, the context and available coping resources are assessed and the threat is compared with memory. After, if the stimulus is still labeled as a threat, current goals are interrupted and attention is focused on the treat. Anxiety disorders can stem from abnormalities in processing at these different stages (Bar-Haim et al., 2007). The finding that attentional interference was larger for idiosyncratic OCD-related stimuli compared to generally negative stimuli in the first dot probe task in chapter 4 provides some evidence for the role of memory in selective attention, as suggested by the integrative model. Without memory such a distinction would be unlikely. However, this effect was not replicated for other measures of selective attention. There was also little evidence for selective attention as a vulnerability factor for OCD. Selective attention by itself did not predict OCD symptoms in any study and there was no difference in selective

attention between participants scoring high on contamination fear and participants scoring low on contamination fear. Moreover, the integrative model was based on findings on attentional bias in anxiety. Yet, in this dissertation no effects of attentional bias were found in the context of OCD. In contrast, attentional bias variability was able to predict contamination fear during a naturalistic stressor in interaction with response inhibition. This suggests that selective attention in OCD is not a stable trait, but rather a dynamic process. Considered as a dynamic process, there may be a role for selective attention as a vulnerability factor in OCD, especially in interaction with other information processing factors.

Van Bockstaele et al. (2014) have proposed a bidirectional model in which attentional bias affects anxiety and anxiety can also affect attentional bias. Based on the results of this dissertation we did not find evidence for an effect of OCD symptoms on attentional bias. This suggests that the role of selective attention in OCD could be qualitatively different from anxiety.

ACT (Eysenck et al., 2007) takes the interaction between selective attention and inhibition into account. ACT poses there is a difference between bottom-up capture, which is influenced by internal and external threatening stimuli (e.g., obsessive thoughts or threat-related stimuli) and top-down control, which is influenced by goals, expectations and knowledge (Corbetta & Shulman, 2002). Selective attention plays a pivotal role in bottom-up capture and inhibition is one of the main functions involved in top-down control. Bottom-up capture and top-down control influence each other bidirectionally. For instance, bottom-up capture decreases top-down control and decreased top-down control is more susceptible to the influence of bottom-up capture. These effects should be most evident under stressful conditions. Due to compensation strategies, effects of decreased top-down control will be most evident on performance efficiency rather than the quality of performance. ACT implies that the presence of current OCD symptoms would enhance selective attention towards OCD-related stimuli and decrease inhibition capacity. It also suggests that underperformance in inhibition could be specific for threat-related stimuli. Moreover, ACT implies an interaction between selective attention and inhibition in which selective attention towards threat would be particularly harmful for anxiety in the context of poor inhibition. The current dissertation found mixed evidence for the application of ACT to OCD. On the one hand,

current OCD symptoms neither affected selective attention towards OCD-related stimuli nor response inhibition capacity. Furthermore, there was no effect of including OCD-related stimuli in a task assessing response inhibition. On the other hand, the interaction between attentional bias variability and response inhibition did predict contamination OCD symptoms during a natural stressor. Based on these mixed findings, no definitive conclusions can be drawn on the applicability of ACT to OCD.

Clinical Implications

To some extent it is not surprising that there is only an effect on OCD symptoms when both response inhibition capacity and attentional bias variability in the context of OCD-related stimuli are affected. Many OCD patients try to divert their attention away from their triggers, but if there is little control over attention and attention is repeatedly focused on different triggers, this would provide ample opportunity for failures to inhibit compulsions. This vicious cycle would be broken if patients could exert attentional or inhibitory control and therefore it would only be problematic when both are impaired simultaneously.

However, it is important to note that the effect of the interaction between response inhibition and selective attention was statistically significant yet small: The model with the interaction only explained 3.4% of the additional variance. This raises an important and critical question: Is this effect clinically significant? Based on the current results this is a difficult question to answer. We showed that the interaction between response inhibition and ABV was stronger than the effect of obsessive beliefs in the prediction of contamination fear during an ecological stressor. Obsessive beliefs had little predictive value for OCD symptoms, yet targeting these beliefs has proved effective in treatment (e.g., Ougrin, 2011; Rosa-Alcazar, Sanchez-Meca, Gomez-Conesa, & Marin-Martinez, 2008). This illustrates that showing that a particular factor can predict symptoms, does not provide information on the presence or size of the reversed effect: the effect of simultaneously decreasing ABV and improving response inhibition on subsequent OCD symptoms. In this line of reasoning, it would be necessary to test whether a computerized training to simultaneously address ABV and response inhibition could have an effect on OCD symptoms. Moreover, even if such training would prove to have merit, it would still need to be investigated if and how this

could optimize treatments. For instance, what is the effect of a standalone training compared to waitlist or other effective treatments? Also the study of the potential benefits of adding this training to current treatments would be an important step forward, in which it is possible that for instance Cognitive Behavior Therapy (CBT) and computerized training would interact and thereby accelerate beneficial treatment results. So far research on training cognitive functions in OCD has been mixed. For instance, Najmi and Amir (2010) showed that training aimed to reduce attentional bias subsequently increased approach behavior to feared stimuli in subclinical contamination OCD, suggesting that computerized training could accelerate beneficial treatment results in exposure therapy. However, other trainings have been less successful. For instance, Calkins and Otto (2013) found no effects of a cognitive control training of three sessions on OCD symptoms. Moreover, Grisham, Becker, Williams, Whitton, and Makkar (2014) found no effects on OCD symptoms with a single session interpretation bias modification training in a subclinical sample. Amir, Kuckertz, Najmi, and Conley (2015) investigated the utility of a combination of self-directed exposure and response prevention with three sessions of attention bias modification, attention control training, interpretation bias modification, and working memory training. They found that only interpretation bias modification and attentional control training resulted in a significant reduction in OCD symptoms. Since chapter 5 showed that information processing factors can interact, it would be interesting for future research to investigate whether these effects might be enhanced by training multiple information processing factors and cognitive functions simultaneously.

To conclude, the current results provide a more fine-grained understanding of the mechanisms underlying OCD, however more research is needed in order to draw conclusions regarding the potential of computerized training of response inhibition and selective attention as a possible standalone or add-on to current treatments.

Strengths and Limitations

The current doctoral dissertation is characterized by several strengths. First, the current studies were among the first to systematically examine the effects of current OCD symptoms on response inhibition and selective attention and the effect of interacting response inhibition and selective attention on OCD symptoms using OCD-

related stimuli. Second, a strength of the current studies was that selective attention was conceptualized both as a stable process (i.e., attentional bias and interference) and a dynamic process in time (i.e., attentional bias variability). While attentional bias and interference scores have often been criticized for their low reliability, attentional bias variability has shown good to excellent reliability (Rodebaugh et al., 2016; Zvielli, Bernstein, & Koster, 2015). Finally, another strength of the current studies was that chapters 4 and 5 used a procedure to enhance the personal relevance of OCD-related stimuli for participants (Muller & Roberts, 2005). Even within symptom dimensions OCD patients show substantial heterogeneity in their triggers (Hermans, Martens, De Cort, Pieters, & Eelen, 2003). The Obsessive-Compulsive Cognition Working Group (OCCWG, 1997) identified this heterogeneity as one of the main obstacles for the study of selective attention in OCD. Since OCD symptoms are likely dimensional rather than categorical (Abramowitz et al., 2014), it is plausible that subclinical or normal participants will also respond to specific OCD-related triggers and not to other OCD-related triggers. Therefore, enhancing the personal relevance of the stimuli is an important methodological aspect in OCD research.

The studies in this doctoral dissertation are also characterized by several limitations. First, the empirical studies were all based on subclinical or healthy participants and may not generalize to clinical OCD. However, this population was well-suited for research on the mechanisms of OCD as OCD symptoms are dimensional rather than categorical, phenomenologically similar in non-clinical and clinical populations, and have similar etiological and maintenance factors in clinical and non-clinical populations (Abramowitz et al., 2014). Second, this doctoral dissertation only used mental contamination based inductions in order to investigate the effect of OCD symptoms on information processing, yet OCD symptoms evoked by mental contamination could be qualitatively different from other OCD symptoms and may have different effects on information processing. For instance, Leopold and Backenstrass (2015) showed that neuropsychological functioning can differ between symptom dimensions in OCD. Third, since OCD-related pictures can evoke OCD symptoms (De Putter, Van Yper, & Koster, 2017) it is possible that the computer tasks including these pictures did not only assess the effect of disorder-relevant pictures on response inhibition and selective attention but also the effect of current OCD symptoms.

However, the mental contamination induction was a more potent induction (De Putter et al., 2017) and this induction had no effect on subsequent performance. Therefore it is unlikely that there was an effect of the valence of the stimuli in the task due to evoked OCD symptoms. Finally, the nature of the OCD symptom induction was independent of the information processing tasks. Here it would be useful to match the OCD symptom induction more closely to the stimuli used in the information processing tasks so that the evoked symptoms are more relevant to the task at hand.

Directions for Future Research

Based on this doctoral dissertation several suggestions can be made for future research. First, in order to draw definitive conclusions on the role of current OCD symptoms on selective attention and response inhibition, more research is necessary with different kinds OCD symptom inductions. This would further elucidate if and specifically which symptoms (dimensions) affect information processing. In this line of research, it would also be interesting to compare the effects of evoked symptoms on information processing factors between subclinical or healthy to clinical OCD, as it is possible that evoked symptoms are not strong enough in healthy or subclinical participants in order to affect information processing. However, here it is important that the symptom provocation is potent yet manageable and in accordance with ethical standards.

Second, in order to further investigate valence-specific effects in information processing in OCD, it would be useful to investigate the effect of idiosyncratically selected pictures taken of their specific triggers in information processing tasks in clinical OCD patients compared to generally negative pictures that are not related to their OCD and neutral pictures.

Moreover, as chapter 5 was one of the first studies investigating interacting effects between information processing variables in OCD, more research on the effect of interacting information processing factors and cognitive factors on OCD would be an important step forward. For instance, it would be interesting to investigate whether this effect generalizes to clinical OCD over different follow-up periods and whether interacting information processing factors can not only be used in order to predict OCD symptoms in the context of life events, but also whether these interacting information

processing factors can predict response to specific treatments. To date, research on whether information processing factors predict treatment response have not taken into account interactions between these factors (e.g., Braga et al., 2016; D'Alcante et al., 2012). If for instance the interaction between response inhibition and selective attention would predict poor response to CBT, a next step would be to investigate whether a training of these factors could increase treatment response. Furthermore, meta-analyses have implicated other information processing factors in OCD, such as set shifting and visuospatial working memory (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014). A large scale investigation of the effect of multiple information processing factors implicated in OCD and their interactions on OCD symptoms would be crucial in order to expand the investigation of the combined cognitive biases hypothesis (Hirsch et al., 2006) in OCD. Here it could also be interesting to see whether there are any effects of an interaction between obsessive beliefs and information processing factors on OCD symptoms. For instance, the belief that it is important to control one's thoughts could be particularly harmful for someone with low cognitive control. The effect of beliefs about the need to control thoughts and response inhibition on the frequency and distress of intrusions during a thought suppression task in OCD patients was investigated by Grisham and Williams (2013). They found that beliefs about the need to control thoughts uniquely predicted intrusions, yet they did not investigate the effects of an interaction between response inhibition and beliefs about the need to control thoughts. In line with the findings of chapter 5, response inhibition did not uniquely predict intrusions.

Relatedly, as discussed in the clinical implications, since the interaction between OCD-related response inhibition and selective attention predicted symptoms during a stressful time period, it would be interesting to see whether a training of response inhibition and selective attention in the context of OCD-related stimuli could protect against the negative influence of stress and enhance or accelerate treatment results in OCD.

Final Conclusion

Due to mixed findings in the literature the role of response inhibition and selective attention in the context of OCD has been unclear. The current doctoral dissertation further examined the role of these factors in OCD with three research aims.

The first aim was to investigate whether the effects of response inhibition and selective attention in the context of OCD were stable or dependent on the experience of current OCD symptoms. In contrast to ACT (Eysenck et al., 2007), the executive overload model (Abramovitch et al., 2012) and Van Bockstaele et al. (2014), we found no evidence for an influence of current OCD symptoms on response inhibition capacity or selective attention. Moreover, in contrast to the endophenotype view in response inhibition (e.g., Chamberlain et al., 2005) and the integrative model (Bar-Haim et al., 2007), there was also no association between stable selective attention and response inhibition on the one hand and OCD symptomatology on the other hand. Therefore, based on the current results, it is uncertain whether response inhibition and selective attention in isolation play a role in subclinical OCD.

The second aim was to investigate whether the effects of response inhibition and selective attention were general or specific to OCD-related content. Although there was more attentional interference of OCD-related stimuli compared to generally negative stimuli, this effect was only temporary and was not applicable to other measures of selective attention. Also in response inhibition there was no effect of OCD-related stimuli compared to generally negative stimuli. Therefore, based on the current results, there might not be an effect of content-specificity in the context of OCD. However, further research is necessary to draw definitive conclusions regarding content-specificity in OCD.

Finally, in accordance with Muller and Roberts (2005) and the combined cognitive biases hypothesis of Hirsch et al. (2006), in the last research aim we investigated whether OCD symptoms were best predicted by single or multiple information processing factors. Here we found no predictive effect of response inhibition or selective attention on OCD symptoms in isolation. There was also no effect of an interaction between response inhibition and selective attention on OCD symptoms after an OCD symptom induction in the lab. However, in line with the combined cognitive biases hypothesis (Hirsch et al., 2006) and ACT (Eysenck et al.,

2007), there was an effect of the interaction between response inhibition and selective attention in the context of contamination related stimuli on the prediction of contamination OCD symptoms experienced during a naturalistic stressor. Attentional bias variability was only able to predict contamination OCD symptoms during the naturalistic stressor when response inhibition was poor. This finding highlights the importance of looking at interactions between information processing factors in order to improve our understanding of OCD.

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References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163-1171. doi: 10.1016/j.cpr.2013.09.004

Abramovitch, A., Dar, R., Hermesh, H., & Schweiger, A. (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD. *Journal of Neuropsychology, 6*(2), 161-191. doi: 10.1111/j.1748-6653.2011.02021.x

Abramovitch, A., Shaham, N., Levin, L., Bar-Hen, M., & Schweiger, A. (2015). Response inhibition in a subclinical obsessive-compulsive sample. *Journal of Behavior Therapy and Experimental Psychiatry, 46*, 66-71. doi: 10.1016/j.jbtep.2014.09.001

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

Amir, N., Kuckertz, J. M., Najmi, S., & Conley, S. L. (2015). Preliminary evidence for the enhancement of self-conducted exposures for OCD using cognitive bias modification. *Cognitive Therapy and Research, 39*(4), 424-440. doi: 10.1007/s10608-015-9675-7

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive–compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157. doi: 10.1016/j.brat.2008.10.020

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1-24. doi: 10.1037/0033-2909.133.1.1

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive–compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141-150. doi: 10.1016/j.jpsychires.2006.11.008

Braga, D. T., Abramovitch, A., Fontenelle, L. F., Ferrão, Y. A., Gomes, J. B., Vivan, A. S., . . . Cordioli, A. V. (2016). Neuropsychological predictors of treatment response to cognitive behavioral group therapy in obesssive-compulsive disorder. *Depression and Anxiety, 33*(9), 848-861. doi: 10.1002/da.22509

Calkins, A. W., & Otto, M. W. (2013). Testing the boundaries of computerized cognitive control training on symptoms of obsessive compulsive disorder. *Cognitive Therapy and Research, 37*(3), 587-594. doi: 10.1007/s10608-012-9496-x

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience & Biobehavioral Reviews, 29*(3), 399-419. doi: 10.1016/j.neubiorev.2004.11.006

Cohen, Y., Lachenmeyer, J. R., & Springer, C. (2003). Anxiety and selective attention in obsessive–compulsive disorder. *Behaviour Research and Therapy, 41*(11), 1311-1323. doi: 10.1016/S0005-7967(03)00037-8

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201-215.

D'Alcante, C. C., Diniz, J. B., Fossaluza, V., Batistuzzo, M. C., Lopes, A. C., Shavitt, R. G., . . . Hoexter, M. Q. (2012). Neuropsychological predictors of response to randomized treatment in obsessive–compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 39*(2), 310-317. doi: 10.1016/j.pnpbp.2012.07.002

De Putter, L. M. S., Van Yper, L., & Koster, E. H. W. (2017). Obsessions and compulsions in the lab: A meta-analysis of procedures to induce symptoms of obsessive-compulsive disorder. *Clinical Psychology Review, 52*, 137-147. doi: 10.1016/j.cpr.2017.01.001

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353. doi: 10.1037/1528-3542.7.2.336

Grisham, J. R., Becker, L., Williams, A. D., Whitton, A. E., & Makkar, S. R. (2014). Using cognitive bias modification to deflate responsibility in compulsive checkers.

Cognitive Therapy and Research, 38(5), 505-517. doi: 10.1007/s10608-014-9621-0

Grisham, J. R., & Williams, A. D. (2013). Responding to intrusions in obsessive-compulsive disorder: The roles of neuropsychological functioning and beliefs about thoughts. *Journal of Behavior Therapy and Experimental Psychiatry, 44*(3), 343-350. doi: 10.1016/j.jbtep.2013.01.005

Harkness, E. L., Harris, L. M., Jones, M. K., & Vaccaro, L. (2009). No evidence of attentional bias in obsessive compulsive checking on the dot probe paradigm. *Behaviour Research and Therapy, 47*(5), 437-443. doi: 10.1016/j.brat.2009.02.004

Hermans, D., Martens, K., De Cort, K., Pieters, G., & Eelen, P. (2003). Reality monitoring and metacognitive beliefs related to cognitive confidence in obsessive–compulsive disorder. *Behaviour Research and Therapy, 41*(4), 383-401. doi: 10.1016/S0005-7967(02)00015-3

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223-236. doi: 10.1016/j.beth.2006.02.001

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of Anxiety Disorders, 30*, 48-58. doi: 10.1016/j.janxdis.2014.12.016

Linkovski, O., Kalanthroff, E., Henik, A., & Anholt, G. E. (2016). Stop checking: Repeated checking and its effects on response inhibition and doubt. *Journal of Behavior Therapy and Experimental Psychiatry, 53*, 84-91. doi: 10.1016/j.jbtep.2014.12.007

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Moritz, S., Fischer, B. K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., & Jelinek, L. (2008). Words may not be enough! No increased emotional Stroop effect in

obsessive–compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104. doi: 10.1016/j.brat.2008.05.005

Moritz, S., Von Muehlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive–compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371. doi: 10.1017/S1355617709090511

Moritz, S., & von Mühlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229. doi: 10.1002/da.20294

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive–Compulsive Disorder: A review. *Journal of Anxiety Disorders, 19*(1), 1-28. doi: 10.1016/j.janxdis.2003.12.001

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive-compulsive symptoms. *Journal of Abnormal Psychology, 119*(1), 136-142. doi: 10.1037/a0017549

OCCWG. (1997). Cognitive assessment of obsessive-compulsive disorder. *Behaviour Research and Therapy, 35*(7), 667-681. doi: 10.1016/S0005-7967(97)00017-X

Ougrin, D. (2011). Efficacy of exposure versus cognitive therapy in anxiety disorders: Systematic review and meta-analysis. *BMC Psychiatry, 11*(1), 200. doi: 10.1186/1471-244x-11-200

Pergamin-Hight, L., Naim, R., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., & Bar-Haim, Y. (2015). Content specificity of attention bias to threat in anxiety disorders: A meta-analysis. *Clinical Psychology Review, 35*, 10-18. doi: 10.1016/j.cpr.2014.10.005

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences, 13*(4), 160-166. doi: 10.1016/j.tics.2009.01.006

Pessoa, L., Padmala, S., Kenzer, A., & Bauer, A. (2012). Interactions between cognition and emotion during response inhibition. *Emotion, 12*(1), 192-197. doi: 10.1037/a0024109

Rasmussen, J., Siev, J., Abramovitch, A., & Wilhelm, S. (2016). Scrupulosity and contamination OCD are not associated with deficits in response inhibition.

Journal of Behavior Therapy and Experimental Psychiatry, 50, 120-126. doi: 10.1016/j.jbtep.2015.06.004

Rodebaugh, T. L., Scullin, R. B., Langer, J. K., Dixon, D. J., Huppert, J. D., Bernstein, A., . . . Lenze, E. J. (2016). Unreliability as a threat to understanding psychopathology: The cautionary tale of attentional bias. *Journal of Abnormal Psychology, 125*(6), 840-851. doi: 10.1037/abn0000184

Rosa-Alcazar, A. I., Sanchez-Meca, J., Gomez-Conesa, A., & Marin-Martinez, F. (2008). Psychological treatment of obsessive-compulsive disorder: A meta-analysis. *Clinical Psychology Review, 28*(8), 1310-1325. doi: 10.1016/j.cpr.2008.07.001

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: A meta-analysis. *Psychological Medicine, 44*(6), 1121-1130. doi: 10.1017/S0033291713001803

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2014). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science*. doi: 10.1177/2167702614534210

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in obsessional compulsive disorder. *Behaviour Research and Therapy, 34*(1), 53-60. doi: 10.1016/0005-7967(95)00041-U

Van Bockstaele, B., Verschuere, B., Tibboel, H., De Houwer, J., Crombez, G., & Koster, E. H. W. (2014). A review of current evidence for the causal impact of attentional bias on fear and anxiety. *Psychological Bulletin, 140*(3), 682-721. doi: 10.1037/a0034834

Verbruggen, F., & De Houwer, J. (2007). Do emotional stimuli interfere with response inhibition? Evidence from the stop signal paradigm. *Cognition and Emotion, 21*(2), 391-403. doi: 10.1080/02699930600625081

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2015). Temporal dynamics of attentional bias. *Clinical Psychological Science, 3*(5), 772–788. doi: 10.1177/2167702614551572

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% Author: Laura de Putter

% Date: 30/03/2017

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% Date: 30/03/2017

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% Author: Laura de Putter

% Date: 30/03/2017

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% Author: Laura de Putter

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NEDERLANDSTALIGE SAMENVATTING

Mensen met een obsessief-compulsieve stoornis (OCS) hebben last van obsessies en/of compulsies. Obsessies zijn intrusieve en recidiverende gedachten, impulsen of voorstellingen. Ze worden vaak geneutraliseerd door middel van compulsies. Compulsies zijn geritualiseerde, repetitieve gedragingen (bijv. controleren of handen wassen) of psychische activiteiten (bijv. tellen of bidden) bedoeld om angst te verminderen en/of een gevreesde gebeurtenis te voorkomen. Deze obsessies en compulsies nemen minstens een uur per dag in beslag, veroorzaken significant lijden en problemen in het sociaal functioneren en/of op het werk (American Psychiatric Association, 2013; Ruscio, Stein, Chiu, & Kessler, 2010). Een kwart van de populatie ervaart tijdens zijn leven obsessies of compulsies, terwijl tussen de 2.3% tot 3.5% van de populatie tijdens zijn leven voldoet aan een klinische obsessief-compulsieve stoornis (Angst et al., 2004; Ruscio et al., 2010). Rachman en de Silva (1978) toonden aan dat gezonde proefpersonen gelijkaardige intrusieve gedachten ervaren als klinische OCS patiënten. OCS ontstaat vaak voor de leeftijd van 20 jaar en komt meer voor bij vrouwen dan bij mannen (Angst et al., 2004; Ruscio et al., 2010). Er bestaan verschillende effectieve behandelingen voor OCS (zie Skapinakis et al., 2016). Er is echter maar 41.7% die na behandeling in remissie is (Farris, McLean, Van Meter, Simpson, & Foa, 2013). Om bestaande behandelingen te kunnen verbeteren is er meer kennis nodig over de mechanismen onderliggend aan OCS.

Men onderscheidt vaak de volgende symptoomdimensies in OCS (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008): (1) Obsessies over symmetrie welke geneutraliseerd worden a.d.h.v. herhalen, ordenen en tellen. Compulsies bij deze symptoomdimensie worden vaak gedreven vanuit een *not-just-right* gevoel, het gevoel dat iets niet helemaal klopt, in plaats van angst (McKay et al., 2004). (2) Agressieve, seksuele, religieuze of somatische obsessies welke geneutraliseerd worden a.d.h.v. controleren. Salkovskis (1985) en Rachman (1997, 1998) stellen dat de drang om te controleren voortkomt uit de overtuiging dat patiënten verantwoordelijk zijn voor hun gedachten en diens mogelijke consequenties. Deze consequenties kunnen zowel betrekking hebben op de realiteit (bijv. als iemand

een beeld heeft dat hij iemand neersteekt, is hij bang dat hij dit enkel door die gedachte echt zal doen) of op hun morele karakter (bijv. het feit dat iemand een beeld heeft dat hij iemand neersteekt, betekent dat hij moreel een slecht persoon is). (3) Obsessies over besmetting welke geneutraliseerd worden a.d.h.v. schoonmaken of wassen. Het kan hierbij zowel gaan over de angst om zelf besmet te worden als om iemand anders te besmetten (Markarian et al., 2010). Deze symptoomdimensie is één van de meest voorkomende symptoomdimensies bij OCS (Ball, Baer, & Otto, 1996). De symptoomdimensies worden gekenmerkt door verschillende neurale substraten, comorbiditeit, genetische transmissie, behandeluitkomst, en neuropsychologisch functioneren (Mataix-Cols, do Rosario-Campos, & Leckman, 2005). Leopold en Backenstrass (2015) toonden bijvoorbeeld aan dat de patiënten met smetvrees beter presteren op de meeste cognitieve taken dan patiënten met controledwang. In dit proefschrift zal de focus liggen op de symptoomdimensie van smetvrees.

Executief Functioneren bij OCS

Er zijn veel tegenstrijdige resultaten in de literatuur over executief functioneren bij OCS (Abramovitch, Abramowitz, & Mittelman, 2013). Verschillende meta-analyses rapporteren significante verschillen tussen OCS patiënten en gezonde proefpersonen in o.a. inhibitie, verbaal werkgeheugen, visuospatiaal werkgeheugen, planning, verwerkingssnelheid en aandacht (Abramovitch et al., 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2014). Tot nu toe heeft onderzoek nauwelijks moderatoren gevonden om deze heterogeniteit in de literatuur te verklaren (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014).

Vanwege het repetitief karakter van obsessies en compulsies is inhibitie van belang bij OCS (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). Er zijn vier verschillende types inhibitie (Nigg, 2000): (1) Interferentiecontrole, deze voorkomt afleiding door andere stimuli. (2) Cognitieve inhibitie, deze onderdrukt irrelevante informatie uit het werkgeheugen. (3) Responsinhibitie, het vermogen om een reeds voorbereide respons alsnog te onderdrukken (Logan, 1994). (4) Oculomotorische inhibitie, het vermogen om reflexieve oogbewegingen te onderdrukken. Friedman en Miyake (2004) toonden aan dat interferentiecontrole, responsinhibitie en oculomotorische inhibitie deel zijn van hetzelfde latente construct.

In dit proefschrift zal de focus liggen op responsinhibitie. Responsinhibitie wordt vaak gemeten a.d.h.v. een stopsignaaltaak. Bij een stopsignaaltaak dienen proefpersonen te reageren op verschillende targets (bijv. een @ teken). Bij 25% tot 30% van de trials hoort men echter een stop signaal. Bij het horen van dit stopsignaal dient men de reactie op de target te onderdrukken. Er is reeds veel onderzoek verricht naar het verband tussen responsinhibitie en OCS. Hierbij zijn er zowel studies die zwakkere responsinhibitie vinden bij patiënten met OCS (bijv. Abramovitch, Dar, Schweiger, & Hermesh, 2011; Menzies et al., 2007) als studies die gelijkaardige prestaties vinden op taken met responsinhibitie (Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Krishna et al., 2011). Daarnaast bestaan er verschillende visies op het verband tussen OCS en inhibitie. Chamberlain et al. (2005) gaan er van uit dat OCS symptomen voortkomen uit een beperking in inhibitie. Beperkte inhibitie zou dus een *endofenotype* van OCS zou zijn. Een endofenotype is een waarneembare component die een ziektebeeld verbindt aan zijn distale genotype (Gottesman & Gould, 2003). Een endofenotype is dus een marker voor een genetisch risico voor een ziektebeeld en wordt niet beïnvloed door het ervaren van symptomen. Daarentegen stellen Abramovitch, Dar, Hermesh, en Schweiger (2012) problemen met inhibitie voor als een epifenomeen van OCS symptomen. In het executive overload model stelt men dat OCS patiënten de neiging hebben om continu automatische processen te controleren (bijv. besluiten wanneer te stoppen met handen wassen). Deze controle veroorzaakt een toename aan obsessieve gedachten en hyperactiviteit in de fronto-striatale regio in het brein. Deze obsessieve gedachten nemen cognitieve capaciteit in beslag, wat vervolgens leidt tot een overbelasting van de executieve functies. Dit leidt vervolgens tot zwakkere prestaties op taken waarvoor executieve functies nodig zijn (bijv. inhibitie). Wanneer de patiënt zich bewust wordt van deze beperkingen (bijv. door te laat op afspraken te komen), leidt dit tot angst voor impulsief te zijn en vervolgens tot verdere pogingen om automatische processen te controleren. Op deze manier ontstaat er een vicieuze cirkel waarbij deze verdere pogingen tot controle leiden tot meer obsessieve gedachten, een hogere overbelasting van executieve functies en dus meer beperkingen in executieve functies.

Een ander probleem binnen de literatuur over OCS en inhibitie is dat er zelden rekening werd gehouden met een mogelijk effect van OCS-gerelateerde stimuli in

problemen met inhibitie. Patiënten met OCS hebben bijvoorbeeld moeite met het inhiberen van compulsies (bijv. handen wassen) bij specifieke stimuli (bijv. in het bijzijn van een familielid dat ze zouden kunnen besmetten), maar geen moeite om deze handelingen te inhiberen in andere situaties (bijv. in het bijzijn van een hond; Linkovski, Kalanthroff, Henik, & Anholt, 2016).

Selectieve aandacht bij OCS

Een tweede factor die regelmatig genoemd wordt als een van de mechanismen die bijdraagt tot het ontstaan en in stand houden van OCS is selectieve aandacht (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Muller & Roberts, 2005). Bij selectieve aandacht richt men vooral de aandacht op bedreigende stimuli in plaats van op neutrale stimuli. De rol van selectieve aandacht bij OCS is enkel uitgewerkt in bredere theorieën over selectieve aandacht en angst. Bar-Haim et al. (2007) voerden een meta-analyse uit en ontwikkelden het integratieve model over selectieve aandacht bij angst. In dit model zijn er vier stadia van verwerking van bedreigende informatie. In het eerste stadium wordt een stimulus in de omgeving automatisch geëvalueerd. Indien een stimulus beoordeeld worden als potentieel gevaarlijk, worden cognitieve voorzieningen toegewezen aan deze stimulus in het tweede stadium van verwerking. Dit leidt tot een interruptie van waar men op dat moment mee bezig is en een toestand van bewuste angst. In het derde stadium worden de context van de bedreiging en de beschikbare voorzieningen om met de bedreiging om te gaan beoordeeld. Ook wordt de bedreiging vergeleken met vorige leerervaringen in het geheugen. Ten slotte, als de stimulus nog steeds gezien wordt als een bedreiging, worden de huidige doelen onderbroken en zal in het laatste stadium de aandacht bewust gericht worden op de bedreiging. Bar-Haim et al. (2007) stellen dat angststoornissen ontstaan door afwijkingen in de verwerking van deze verschillende stadia. In hun meta-analyse vonden ze geen significante verschillen tussen de effecten van selectieve aandacht bij OCS en bij angststoornissen, wat impliceert dat deze theorie ook toegepast kan worden op OCS.

Van Bockstaele et al. (2014) onderzochten de empirische evidentie voor een causaal verband van selectieve aandacht naar angst. Net zoals Bar-Haim et al. (2007), concludeerden zij dat selectieve aandacht een kwetsbaarheidsfactor is voor angst.

Studies tonen immers aan dat een verandering in selectieve aandacht een invloed heeft op kwetsbaarheid voor stress (bijv. Verhaak, Smeenk, van Minnen, & Kraaimaat, 2004). Zij vonden echter ook evidentie voor het omgekeerde verband, namelijk dat symptomen een invloed kunnen hebben op selectieve aandacht. Zo vonden Foa en McNally (1986) dat OCS patiënten minder aandachtsbias voor bedreiging vertoonden na succesvolle behandeling met exposure.

Hoewel er inderdaad studies zijn die een aandachtsbias vinden voor OCS-gerelateerde stimuli in subklinische en klinische OCS personen (bijv. Amir, Najmi, & Morrison, 2009; Lavy, Van Oppen, & Van Den Hout, 1994; Moritz, Von Muehlenen, Randjbar, Fricke, & Jelinek, 2009; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996), zijn er ook studies die dit verband niet vinden (bijv. Harkness, Harris, Jones, & Vaccaro, 2009; Morein-Zamir et al., 2013; Moritz et al., 2008; Moritz & von Mühlenen, 2008). Op basis van de contrasterende bevindingen bij OCS besloten Summerfeldt en Endler (1998) dat er, in tegenstelling tot angststoornissen, geen evidentie is voor een aandachtsbias bij OCS. Bar-Haim et al. (2007) vonden echter geen significant verschil tussen OCS en angststoornissen in selectieve aandacht. Op basis van het huidige onderzoek is de aard van het verband tussen OCS en selectieve aandacht dus nog onduidelijk. Najmi en Amir (2010) vonden dat het verminderen van een aandachtsbias in subklinische OCS proefpersonen met smetvrees, leidde tot meer toenaderingsgedrag naar besmette stimuli. Dit impliceert dat een aandachtsbias wel degelijk een effect kan hebben op OCS symptomen.

Een andere vraag binnen de literatuur over selectieve aandacht bij OCS is in welke mate een aandachtsbias specifiek is voor OCS-gerelateerde stimuli of negatieve stimuli in het algemeen. Pergamin-Hight, Naim, Bakermans-Kranenburg, van IJzendoorn, en Bar-Haim (2015) voerden een meta-analyse uit om de valentiespecificiteit van een aandachtsbias bij angst te onderzoeken. Ze vonden dat een aandachtsbias specifiek was voor stoornis-gerelateerde stimuli. Dit impliceert dat selectieve aandacht beïnvloed wordt door leerervaringen. Ze vonden geen significant verschil in het type angststoornis in dit verband (post-traumatische stress stoornis, paniekstoornis, sociale angst stoornis en OCS). Deze meta-analyse bevatte echter maar vier studies met OCS en van deze studies was er maar één die een significant

valentiespecifiek effect vond. Er is dus meer onderzoek nodig naar valentiespecificiteit van selectieve aandacht bij OCS.

Selectieve aandacht wordt vaak gemeten a.d.h.v. een dot probe taak. Bij een dot probe taak worden twee foto's gepresenteerd, een boven en een onder een fixatiekruis. Deze foto's kunnen bestaan uit twee neutrale foto's of een bedreigende foto en een neutrale foto. Na de foto's verschijnt er een stip op de locatie van één van de twee plaatsen waar voorheen de foto's gepresenteerd werden. Selectieve aandacht wordt vervolgens geoperationaliseerd met verschillende maten. De meest gebruikte maat is de aandachtsbias score, de mate waarin aandacht meer wordt gefocust op bedreigende foto's dan op neutrale foto's. Een andere maat is aandachtsverstoring, deze peilt naar de mate waarin bedreigende foto's de aandacht verstoren. Bij de ontwikkeling van deze maten ging men ervan uit dat selectieve aandacht relatief stabiel was over de tijd heen. Sindsdien hebben echter verschillende onderzoeken aangetoond dat er problemen zijn met de betrouwbaarheid van deze maten. Dit impliceert dat selectieve aandacht eerder een dynamisch proces is in plaats van een stabiele trek (Rodebaugh et al., 2016). Op basis van deze conceptualisatie zijn maten ontwikkeld op trialniveau voor selectieve aandacht. Een van deze maten is de variabiliteit in aandachtsbias. Bradley et al. (2016) vonden bijvoorbeeld dat er geen effect was van OCS symptomen op stabiele maten van selectieve aandacht, maar OCS symptomen voorspelden wel dynamische selectieve aandacht, namelijk de neiging om herhaald opnieuw de aandacht te richten op OCS-gerelateerde stimuli. Op dit moment is er nog weinig onderzoek naar selectieve aandacht als een dynamisch proces bij OCS.

Interacties tussen Informatieverwerkingsfactoren

Tot op heden ging onderzoek vooral uit van individuele informatieverwerkings-factoren. Informatieverwerkingsfactoren zouden echter ook kunnen interageren bij het ontstaan en in stand houden van OCS symptomen (Hirsch, Clark, & Mathews, 2006; Muller & Roberts, 2005). De aandachtscontroletheorie is een theorie die interacties tussen informatieverwerkingsfactoren in rekening brengt (Eysenck, Derakshan, Santos, & Calvo, 2007). In deze theorie onderscheidt men bottom-up en top-down controle over aandacht. Bottom-up controle wordt gedreven door saillante of bedreigende interne (bijv. intrusieve gedachten) of externe (bijv. beangstigende foto's) stimuli. Top-

down controle wordt gestuurd door doelen, verwachtingen en kennis (Corbetta & Shulman, 2002). Een van de belangrijkste functies voor top-down controle is inhibitie. Bottom-up controle en top-down controle concurreren met elkaar voor werkgeheugencapaciteit. Bij verhoogde bottom-up controle daalt dus de top-down controle, welke nodig is om cognitieve taken goed uit te voeren. Aan de andere kant zijn mensen met betere inhibitiecapaciteit minder vatbaar voor bottom-up controle.

Verder maakt de aandachtscontroletheorie een onderscheid tussen de effectiviteit en de efficiëntie van prestatie. De effectiviteit verwijst naar de kwaliteit van de prestatie (bijv. aantal fouten), terwijl efficiëntie verwijst naar de inspanning die nodig was om te komen tot die effectiviteit (bijv. reactietijden). Effecten van verminderde top-down controle zullen eerder een impact hebben op de efficiëntie van de prestatie.

Bij OCS wordt bottom-up controle beïnvloed door obsessieve gedachten en selectieve aandacht naar OCS-gerelateerde stimuli. Dit systeem zou kunnen interageren met inhibitie in het verklaren van het ontstaan en in stand houden van OCS symptomen. Het zou bijvoorbeeld kunnen dat de neiging om selectief de aandacht te richten op obsessieve gedachten en OCS-gerelateerde stimuli samen met een probleem met inhibitie in de context van OCS-gerelateerde stimuli, bijdraagt tot verdere OCS symptomen. Dit kan leiden tot een vicieuze cirkel waarbij OCS symptomen vervolgens leiden tot meer selectieve aandacht naar OCS-gerelateerde stimuli en meer problemen met inhibitie.

Onderzoeksdoelen van het Proefschrift

Op basis van de hiaten in het huidige onderzoek, werden drie onderzoeksdoelen opgesteld in dit proefschrift die belangrijk zijn om te komen tot een beter begrip van het verband tussen selectieve aandacht, inhibitie en OCS symptomen.

(1) Zijn afwijkingen in informatieverwerking stabiel of afhankelijk van de ervaring van OCS symptomen? Op dit moment is het onduidelijk of een probleem met inhibitie fungeert als een kwetsbaarheidsfactor voor OCS of eerder een gevolg is van de ervaring van OCS symptomen. Bij het verband tussen selectieve aandacht en OCS is er een gelijkaardig debat, waarbij het onduidelijk is of selectieve aandacht bijdraagt aan de

ontwikkeling van OCS of dat OCS symptomen ook een effect kunnen hebben op selectieve aandacht.

(2) Zijn afwijkingen algemeen of valentiespecifiek? Tot nu toe is het bij OCS onduidelijk of er sprake is van een algemeen probleem met inhibitie en selectieve aandacht voor alle negatieve stimuli. Het zou ook kunnen dat deze afwijkingen enkel voorkomen in de context van OCS-gerelateerde stimuli. De aandachtscontroletheorie (Eysenck et al., 2007) voorspelt dat stoornis-gerelateerde stimuli een sterker effect zouden hebben dan neutrale stimuli.

(3) Worden OCS symptomen het beste voorspeld door aparte informatie-verwerkingsfactoren of is er sprake van een interactie tussen informatieverwerkings-factoren? Zowel de aandachtscontroletheorie (Eysenck et al., 2007), als Muller en Roberts (2005), en Hirsch et al. (2006) stelden dat informatieverwerkingsfactoren interageren in het ontstaan en in stand houden van symptomen. Het is mogelijk dat selectieve aandacht naar OCS-gerelateerde stimuli vooral problematisch is bij personen die moeite hebben met inhibitie.

Bevindingen

Stabiele Informatieverwerkingsfactoren of Afhankelijk van OCS Symptomen?

Om de invloed van de ervaring van OCS symptomen op selectieve aandacht en inhibitie te onderzoeken, begonnen we in hoofdstuk 2 met een meta-analyse naar bestaande methoden om OCS symptomen op te wekken. Uit deze meta-analyse bleek dat mentale contaminatie, een intern gevoel van vuil zijn met een morele component (bijv. bij zich schuldig voelen), een van de meest effectieve methoden was om OCS symptomen op te wekken in gezonde proefpersonen. Om deze reden zijn de inducties voor OCS symptomen in de volgende studies steeds gebaseerd op mentale contaminatie.

In hoofdstuk 3 gingen we de invloed van OCS symptomen na op responsinhibitie. In deze studie werd de prestatie op een stopsignaaltaak vergeleken tussen proefpersonen die hoog en laag scoorden op smetvrees. De stopsignaaltaak werd afgenomen voor en na ofwel een neutrale stemmingsinductie ofwel een OCS symptomen inductie. Hoewel bleek dat de OCS symptomen inductie effectief was in het opwekken van symptomen, presteerde men niet slechter op de stopsignaaltaak na een

OCS symptomen inductie. Dit is in tegenstelling tot de visie dat problemen met inhibitie afhankelijk zouden zijn van de ervaring van OCS symptomen. Indien problemen met inhibitie een stabiel kenmerk zouden zijn van OCS, zouden we verwachten dat (1) personen die laag scoren op smetvrees beter presteren dan personen die hoog scoren op smetvrees, (2) zouden we geen effect van een OCS symptomen inductie verwachten en (3) zouden we verwachten dat inhibitiecapaciteit een toename in symptomen zou kunnen voorspellen na een OCS symptomen inductie. Hoewel er inderdaad geen effect was van een OCS symptomen inductie, kon inhibitiecapaciteit geen toename in symptomen voorspellen na de inductie. Verder presteerden personen die hoog scoorden op smetvrees zelfs wat beter dan personen die laag scoorden op smetvrees. Deze bevinding zou kunnen liggen aan de focus op de smetvrees symptoomdimensie. Leopold en Backenstrass (2015) toonden namelijk aan dat prestatie op informatieverwerkingsfactoren kunnen verschillen tussen symptoomdimensies, waarbij de smetvrees symptoomdimensie over het algemeen beter presteert.

In hoofdstuk 4 gingen we de invloed van OCS symptomen na op selectieve aandacht met twee studies. In de eerste studie onderzochten we selectieve aandacht naar OCS-gerelateerde stimuli en stimuli die veiligheidssignalen representeerden. Indien selectieve aandacht naar OCS-gerelateerde stimuli een kenmerk is van OCS, zouden we meer selectieve aandacht verwachten in personen die hoog scoren op smetvrees vergeleken met personen die laag scoren op smetvrees. We vonden echter geen verschillen tussen hoog- en laagscoorders. Beide groepen vertoonden selectieve aandacht naar OCS-gerelateerde stimuli. In de tweede studie gingen we het effect van OCS symptomen op selectieve aandacht na door een dot probe taak af te nemen voor en na ofwel een neutrale stemmingsinductie ofwel een OCS symptomen inductie. Hoewel de OCS symptomen inductie effectief was in het opwekken van symptomen, vonden we geen effect van deze inductie op selectieve aandacht. Deze bevinding gaat in tegen de visie dat selectieve aandacht beïnvloed zou worden door de ervaring van OCS symptomen. Selectieve aandacht kon echter ook niet de toename in OCS symptomen voorspellen na een OCS symptomen inductie. Dit gaat in tegen de visie van selectieve aandacht als kwetsbaarheidsfactor.

Conclusie. Deze resultaten stellen de rol van responsinhibitie en selectieve aandacht bij OCS in vraag.

Algemene of Valentiespecifieke Afwijkingen in Informatieverwerking

Bij de tweede onderzoeksvraag gingen we na of het effect van responsinhibitie en selectieve aandacht valentiespecifiek is (i.e., enkel voor OCS-gerelateerde stimuli) of algemeen. In hoofdstuk 3 werd dit onderzocht bij responsinhibitie door een aangepaste stopsignaaltaak aan te bieden met neutrale, negatieve en OCS-gerelateerde stimuli. Er was echter geen effect van OCS-gerelateerde stimuli. Proefpersonen presteerden wel wat beter na negatieve stimuli vergeleken met neutrale stimuli.

In hoofdstuk 4 werd valentiespecificiteit onderzocht in twee studies. In de eerste studie werd selectieve aandacht naar OCS-gerelateerde stimuli vergeleken met selectieve aandacht naar veiligheidssignalen (bijv. schoonmaakproducten). Zoals verwacht, was er meer selectieve aandacht naar OCS-gerelateerde stimuli dan naar veiligheidssignalen, maar dit effect verschilde niet tussen personen die hoog of laag scoorden op OCS. In de tweede studie vergeleken we selectieve aandacht naar OCS-gerelateerde stimuli met selectieve aandacht naar algemeen negatieve stimuli. Hierbij was er enkel significant meer selectieve aandacht naar OCS-gerelateerde stimuli bij aandachtsverstoring. Dit was echter maar een tijdelijk effect.

Conclusie. Op basis van de resultaten is het dus twijfelachtig of er sprake is van valentiespecificiteit in inhibitie en selectieve aandacht in de context van OCS.

Het Effect van Gecombineerde Responsinhibitie en Selectieve Aandacht

In hoofdstuk 5 werd onderzocht in welke mate de interactie tussen selectieve aandacht en responsinhibitie OCS symptomen kon voorspellen. In een eerste sessie werden bij studenten aan het begin van het semester selectieve aandacht, responsinhibitie en OCS symptomen gemeten. In deze sessie werd ook een OCS symptomen inductie toegepast om na te gaan in welke mate responsinhibitie en selectieve aandacht deze symptomen konden voorspellen. Als een naturalistische stressor werden OCS symptomen opnieuw gemeten tijdens de examenperiode (68 tot 80 dagen later). Responsinhibitie na OCS-gerelateerde stimuli en selectieve aandacht naar OCS-gerelateerde stimuli konden zowel apart als in interactie geen OCS symptomen na de inductie in het lab voorspellen, nadat gecorrigeerd was voor de OCS symptomen gemeten in de eerste sessie en typische denkfouten bij OCS. Er was ook

geen effect van deze factoren op algemene OCS symptomen tijdens de examenperiode. Er was echter wel een effect van de interactie tussen responsinhibitie na OCS-gerelateerde stimuli en selectieve aandacht naar OCS-gerelateerde stimuli in het voorspellen van smetvreessymptomen tijdens de examenperiode, nadat gecorrigeerd was voor de OCS symptomen gemeten in de eerste sessie, typische denkfouten bij OCS en de informatieverwerkingsfactoren apart. Dit effect was specifiek voor de interactie tussen variabiliteit in aandachtsbias en responsinhibitie in de context van OCS-gerelateerde stimuli. Enkel wanneer personen zwakker presteerden op inhibitie kon meer variabiliteit in aandachtsbias toekomstige smetvreessymptomen voorspellen.

Conclusie. Deze bevindingen impliceren dat interagerende informatie-verwerkingsfactoren een rol kunnen hebben in het ontstaan van OCS symptomen, zoals voorgesteld door Muller en Roberts (2005) en Hirsch et al. (2006).

Implicaties

Theoretische Implicaties

De bovenstaande bevindingen hebben verschillende theoretische implicaties. Binnen theorieën over het verband tussen inhibitie en OCS vonden we geen evidentie voor het executive overload model (Abramovitch et al., 2012). We vonden namelijk geen effecten van een OCS symptomen inductie op inhibitie. Dit zou kunnen betekenen dat het executive overload model enkel van toepassing is op klinische OCS of andere informatieverwerkingsfactoren dan inhibitie. Abramovitch, Shaham, Levin, Bar-Hen, en Schweiger (2015) vonden echter dat subklinische personen slechter presteerden dan gezonde personen die laag scoorden op OCS. Dit impliceert dat het effect van responsinhibitie niet enkel voorkomt bij klinische OCS patiënten. Chamberlain et al. (2005) gingen uit van inhibitie als endofenotype van OCS. Voor deze theorie vinden we gemengde evidentie: aan de ene kant voorspelde de interactie tussen responsinhibitie en selectieve aandacht prospectief smetvreessymptomen tijdens een naturalistische stressor en vonden we geen effect van OCS symptomen op responsinhibitie. Aan de andere kant presteerden subklinische smetvrees OCS proefpersonen beter op inhibitie dan proefpersonen die laag scoorden op smetvrees en kon responsinhibitie apart geen OCS symptomen voorspellen. Op basis van de huidige bevindingen kunnen dus geen sluitende conclusies gemaakt worden omtrent deze theorie.

Binnen theorieën over het verband tussen selectieve aandacht en OCS stelden Bar-Haim et al. (2007) het integratieve model voor waarbij selectieve aandacht gezien wordt als kwetsbaarheidsfactor voor angststoornissen. De bevinding dat aandachtsverstoring groter was voor individueel geselecteerde OCS-gerelateerde stimuli vergeleken met algemeen negatieve stimuli wijst op een rol van geheugen in selectieve aandacht, zoals in het integratieve model wordt verondersteld. Dit effect was echter tijdelijk en veralgemeende zich niet naar andere maten van selectieve aandacht. Verder was er gemengde evidentie voor selectieve aandacht als kwetsbaarheidsfactor voor OCS. Selectieve aandacht apart kon namelijk in geen enkele studie OCS symptomen voorspellen en er was geen verschil tussen proefpersonen die hoog of laag op smetvrees scoorden. Variabiliteit in aandachtsbias kon in interactie met responsinhibitie echter wel smetvreessymptomen voorspellen tijdens een naturalistische stressor. Dit impliceert dat selectieve aandacht in OCS beter begrepen kan worden als een dynamisch proces in plaats van een stabiel kenmerk. Als dynamisch proces is het dus mogelijk dat selectieve aandacht fungeert als kwetsbaarheidsfactor voor OCS in interactie met andere informatieverwerkingsfactoren. Van Bockstaele et al. (2014) stelden selectieve aandacht bij angst voor als een bidirectioneel model waarbij selectieve aandacht een invloed heeft op angst, maar angst ook een invloed heeft op selectieve aandacht. Op basis van bovenstaande resultaten vonden we echter geen effect van OCS symptomen op selectieve aandacht. Het is dus mogelijk dat selectieve aandacht op een kwalitatief andere manier werkt in de context van OCS dan bij angst.

De aandachtscontroletheorie (Eysenck et al., 2007) houdt rekening met de interactie tussen selectieve aandacht en inhibitie, waarbij men ervan uitgaat dat selectieve aandacht naar bedreigende stimuli vooral schadelijk is in de context van problematische inhibitie. We vonden gemengde evidentie voor de toepassing van de aandachtscontroletheorie op OCS. Aan de ene kant hadden OCS symptomen geen effect op selectieve aandacht of inhibitie. Verder was er ook geen effect van OCS-gerelateerde stimuli op inhibitie. Aan de andere kant kon de interactie tussen variabiliteit in aandachtsbias en responsinhibitie in de context van OCS-gerelateerde stimuli wel smetvreessymptomen voorspellen tijdens een naturalistische stressor. Er kunnen dus geen sluitende conclusies getrokken worden over de toepasbaarheid van de aandachtscontroletheorie op OCS.

Klinische Implicaties

De bevinding dat enkel de interactie tussen inhibitie en variabiliteit in aandachtsbias OCS symptomen kan voorspellen sluit aan bij observaties van de klinische praktijk: Veel OCS patiënten proberen immers wel hun aandacht te verplaatsen van hun triggers, maar als er weinig controle is over aandacht en deze steeds opnieuw gefocust wordt op triggers, biedt dit veel mogelijkheden voor problemen met het inhiberen van compulsies. Deze vicieuze cirkel zou verbroken worden indien patiënten controle zouden hebben over hun aandacht of controle over inhibitie. Om deze reden zou het dan enkel een probleem zijn indien inhibitie en selectieve aandacht tegelijk aangetast zijn.

Dit interactie-effect was echter significant maar klein: Maar 3.4% aanvullende variantie kon verklaard worden met dit effect. Een belangrijke en kritische vraag is dan ook: Is dit effect klinisch significant? Dit is een ingewikkelde vraag. Aan de ene kant vonden we dat de interactie tussen responsinhibitie en variabiliteit in aandachtsbias een sterker effect had dan typische denkfouten bij OCS in het voorspellen van OCS symptomen. Denkfouten typisch bij OCS (bijv. overschatting verantwoordelijkheid) waren zelfs geen significante predictor voor OCS symptomen tijdens een naturalistische stressor. Het aanpakken van deze denkfouten is echter effectief in behandeling van OCS (bijv. Ougrin, 2011; Rosa-Alcazar, Sanchez-Meca, Gomez-Conesa, & Marin-Martinez, 2008). Dit toont aan dat het gegeven dat een factor al dan niet een predictor is voor OCS, niet noodzakelijk iets zegt over het bestaan en de grootte van het omgekeerde verband: het effect van het tegelijkertijd verbeteren van variabiliteit in aandachtsbias en responsinhibitie op OCS symptomen. Om dit na te gaan is het nodig om te onderzoeken of een training die simultaan variabiliteit in aandachtsbias en responsinhibitie aanpakt een invloed heeft op OCS symptomen. Zelfs als een dergelijke training een invloed heeft op OCS symptomen, dient nog onderzocht te worden of en hoe dit de huidige behandelingen zou kunnen verbeteren. Wat is bijvoorbeeld het effect van enkel een computertraining van deze factoren vergeleken met wachtlijstcondities of de huidige behandelingen? Verder is ook onderzoek belangrijk naar de mogelijke meerwaarde van deze training toe te voegen aan de huidige behandelingen. Het zou bijvoorbeeld kunnen dat cognitieve gedragstherapie en

computertraining hierbij zouden interageren waardoor er sneller resultaten geboekt kunnen worden met de behandeling.

Tot nu toe is de wetenschappelijke evidentie voor het trainen van cognitieve functies in OCS gemengd. Najmi en Amir (2010) toonden bijvoorbeeld aan dat training voor het verminderen van aandachtsbias leidde tot meer toenaderingsgedrag naar gevreesde stimuli in subklinische smetvrees OCS. Dit impliceert dat computertraining behandelresultaten zou kunnen versnellen in de context van exposure therapie. Andere trainingen waren echter minder succesvol. Calkins en Otto (2013) vonden bijvoorbeeld geen effecten van een cognitieve controle training van drie sessies op OCS symptomen. Ook Grisham, Becker, Williams, Whitton, en Makkar (2014) vonden geen effecten van een sessie interpretatiebias training op OCS symptomen bij subklinische OCS personen. Verder onderzochten Amir, Kuckertz, Najmi, en Conley (2015) de bruikbaarheid van exposure en responspreventie in combinatie met drie trainingssessies van aandachtsbias, aandachtscontrole, interpretatiebias, en werkgeheugen. Hierbij vonden ze dat enkel een training van interpretatiebias en aandachtscontrole leidde tot een significante vermindering van OCS symptomen. Aangezien uit hoofdstuk 5 bleek dat informatieverwerkingsfactoren kunnen interageren, zou het interessant zijn voor toekomstig onderzoek om na te gaan of het effect van cognitieve training sterker zou kunnen zijn door verschillende informatieverwerkingsfactoren simultaan te trainen.

Concluderend kunnen we stellen dat de huidige resultaten een beter begrip van de mechanismen onderliggend aan OCS bieden, maar er is meer onderzoek nodig om uitspraken te kunnen maken over het potentieel van trainingen van responsinhibitie en selectieve aandacht als mogelijke (aanvullende) behandeling.

Sterktes en Beperkingen

In de studies in dit proefschrift zijn verschillende sterktes en beperkingen aan te merken. De huidige studies waren een van de eerste om systematisch het effect van de ervaring van OCS symptomen te onderzoeken op responsinhibitie en selectieve aandacht. Ook was het een van de eerste studies naar het effect van interagerende informatieverwerkingsfactoren op OCS symptomen. Daarnaast werd selectieve aandacht zowel onderzocht als stabiel kenmerk (i.e., aandachtsbias en aandachtsverstoring) en als een dynamisch proces (variabiliteit in aandachtsbias).

Maten van aandachtsbias en aandachtsverstoring worden immers vaak bekritiseerd om hun lage betrouwbaarheid, terwijl variabiliteit in aandachtsbias een betrouwbare maat is (Rodebaugh et al., 2016; Zvielli, Bernstein, & Koster, 2015). Ten slotte is een sterkte van de huidige studies dat in hoofdstukken 4 en 5 een procedure is toegepast om de persoonlijke relevantie van OCS-gerelateerde stimuli voor proefpersonen te verhogen (Muller & Roberts, 2005). Aangezien OCS symptomen eerder dimensioneel zijn dan categorisch (Abramowitz et al., 2014), is het plausibel dat voor subklinische en gezonde proefpersonen specifieke OCS-gerelateerde stimuli relevant zijn.

Een van de beperkingen van de studies in dit proefschrift is dat alle studies gebaseerd waren op subklinische of gezonde proefpersonen en de resultaten dus niet noodzakelijk te generaliseren zijn naar klinische OCS patiënten. Subklinische en gezonde proefpersonen zijn echter zeer geschikt voor onderzoek naar de mechanismen onderliggend aan OCS. OCS symptomen zijn immers dimensioneel in plaats van categorisch, fenomenologisch gelijkaardig in niet-klinische en klinische populaties (het merendeel van de populatie ervaart bijvoorbeeld wel eens een intrusieve gedachte), en gelijkaardige mechanismen spelen mee in het ontstaan en in stand houden van OCS symptomen bij klinische en niet-klinische populaties (Abramowitz et al., 2014). Daarnaast is het een beperking dat in de huidige studies enkel OCS symptomen inducties zijn gebruikt die gebaseerd zijn op mentale contaminatie. Het zou echter kunnen dat OCS symptomen opgewekt a.d.h.v. mentale contaminatie kwalitatief anders zijn dan andere OCS symptomen en andere effecten zouden hebben op informatieverwerking. Informatieverwerking kan immers verschillen tussen symptoomdimensies in OCS (Leopold & Backenstrass, 2015).

Besluit

Dit proefschrift onderzocht de rol van responsinhibitie en selectieve aandacht in OCS symptomen aan de hand van drie onderzoeksdoelen. Het eerste doel was om te onderzoeken of responsinhibitie en selectieve aandacht stabiele kenmerken van OCS zijn of afhankelijk zijn van de ervaring van OCS symptomen. In tegenstelling tot de aandachtscontroletheorie (Eysenck et al., 2007), het executive overload model (Abramovitch et al., 2012) en Van Bockstaele et al. (2014), vonden we geen effect van de ervaring van OCS symptomen op responsinhibitie of selectieve aandacht. Verder

vonden we geen evidentie voor een verband tussen selectieve aandacht en responsinhibitie als kenmerk en OCS symptomatologie. Dit gaat in tegen de visie van inhibitie als een endofenotype (bijv. Chamberlain et al., 2005) en het integratieve model over selectieve aandacht (Bar-Haim et al., 2007). Op basis van de huidige resultaten is het dus onduidelijk of en hoe responsinhibitie en selectieve aandacht apart een rol spelen bij OCS symptomen.

Het tweede doel van dit proefschrift was het onderzoeken of afwijkingen in responsinhibitie en selectieve aandacht algemeen waren of specifiek voor OCS-gerelateerde stimuli. We vonden meer aandachtsverstoring van OCS-gerelateerde stimuli vergeleken met negatieve stimuli. Dit effect was echter tijdelijk en vonden we niet terug bij andere maten van selectieve aandacht. Ook bij responsinhibitie was er geen effect van OCS-gerelateerde stimuli vergeleken met negatieve stimuli. Op basis van de huidige resultaten is het dus mogelijk dat het effect van inhibitie en selectieve aandacht bij OCS niet valentiespecifiek is.

Het laatste doel was om te onderzoeken of OCS symptomen het best voorspeld konden worden door aparte of interagerende informatieverwerkingsfactoren. Muller en Roberts (2005) en Hirsch et al. (2006) stelden voor dat informatieverwerkings-factoren zouden kunnen interageren in het verklaren van stoornissen. Responsinhibitie en selectieve aandacht konden apart geen OCS symptomen voorspellen. Er was ook geen interactie-effect tussen responsinhibitie en selectieve aandacht in het voorspellen van OCS symptomen na een OCS symptomen inductie in het lab. Echter, overeenkomstig met de aandachtscontroletheorie (Eysenck et al., 2007), voorspelde de interactie tussen responsinhibitie en selectieve aandacht in de context van contaminatie-gerelateerde stimuli smetvrees OCS symptomen tijdens een naturalistische stressor. Variabiliteit in aandachtsbias voorspelde hierbij enkel smetvrees OCS symptomen tijdens een naturalistische stressor bij zwakke responsinhibitie. Deze bevinding wijst op het belang van het in rekening brengen van interacties tussen informatieverwerkingsfactoren bij verklaringsmodellen van OCS.

Referenties

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical Psychology Review, 33*(8), 1163-1171. doi: 10.1016/j.cpr.2013.09.004

Abramovitch, A., Dar, R., Hermesh, H., & Schweiger, A. (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD. *Journal of Neuropsychology, 6*(2), 161-191. doi: 10.1111/j.1748-6653.2011.02021.x

Abramovitch, A., Dar, R., Schweiger, A., & Hermesh, H. (2011). Neuropsychological impairments and their association with obsessive-compulsive symptom severity in obsessive-compulsive disorder. *Archives of Clinical Neuropsychology*. doi: 10.1093/arclin/acr022

Abramovitch, A., Shaham, N., Levin, L., Bar-Hen, M., & Schweiger, A. (2015). Response inhibition in a subclinical obsessive-compulsive sample. *Journal of Behavior Therapy and Experimental Psychiatry, 46*, 66-71. doi: 10.1016/j.jbtep.2014.09.001

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206-217. doi: 10.1016/j.cpr.2014.01.004

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC.

Amir, N., Kuckertz, J. M., Najmi, S., & Conley, S. L. (2015). Preliminary evidence for the enhancement of self-conducted exposures for OCD using cognitive bias modification. *Cognitive Therapy and Research, 39*(4), 424-440. doi: 10.1007/s10608-015-9675-7

Amir, N., Najmi, S., & Morrison, A. S. (2009). Attenuation of attention bias in obsessive–compulsive disorder. *Behaviour Research and Therapy, 47*(2), 153-157. doi: 10.1016/j.brat.2008.10.020

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rossler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence,

comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164. doi: 10.1007/s00406-004-0459-4

Ball, S. G., Baer, L., & Otto, M. W. (1996). Symptom subtypes of obsessive-compulsive disorder in behavioral treatment studies: A quantitative review. *Behaviour Research and Therapy, 34*(1), 47-51. doi: 10.1016/0005-7967(95)00047-2

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1-24. doi: 10.1037/0033-2909.133.1.1

Bloch, M. H., Landeros-Weisenberger, A., Rosario, M. C., Pittenger, C., & Leckman, J. F. (2008). Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry, 165*(12), 1532-1542. doi: 10.1176/appi.ajp.2008.08020320

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive–compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141-150. doi: 10.1016/j.jpsychires.2006.11.008

Bradley, M. C., Hanna, D., Wilson, P., Scott, G., Quinn, P., & Dyer, K. F. W. (2016). Obsessive–compulsive symptoms and attentional bias: An eye-tracking methodology. *Journal of Behavior Therapy and Experimental Psychiatry, 50*, 303-308. doi: 10.1016/j.jbtep.2015.10.007

Calkins, A. W., & Otto, M. W. (2013). Testing the boundaries of computerized cognitive control training on symptoms of obsessive compulsive disorder. *Cognitive Therapy and Research, 37*(3), 587-594. doi: 10.1007/s10608-012-9496-x

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience & Biobehavioral Reviews, 29*(3), 399-419. doi: 10.1016/j.neubiorev.2004.11.006

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201-215.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336-353. doi: 10.1037/1528-3542.7.2.336

Farris, S. G., McLean, C. P., Van Meter, P. E., Simpson, H. B., & Foa, E. B. (2013). Treatment response, symptom remission and wellness in obsessive-compulsive disorder. *The Journal of clinical psychiatry, 74*(7), 685-690. doi: 10.4088/JCP.12m07789

Foa, E. B., & McNally, R. J. (1986). Sensitivity to feared stimuli in obsessive-compulsives: A dichotic listening analysis. *Cognitive Therapy and Research, 10*(4), 477-485. doi: 10.1007/bf01173299

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology: General, 133*(1), 101-135.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160*(4), 636-645. doi: 10.1176/appi.ajp.160.4.636

Grisham, J. R., Becker, L., Williams, A. D., Whitton, A. E., & Makkar, S. R. (2014). Using cognitive bias modification to deflate responsibility in compulsive checkers. *Cognitive Therapy and Research, 38*(5), 505-517. doi: 10.1007/s10608-014-9621-0

Harkness, E. L., Harris, L. M., Jones, M. K., & Vaccaro, L. (2009). No evidence of attentional bias in obsessive compulsive checking on the dot probe paradigm. *Behaviour Research and Therapy, 47*(5), 437-443. doi: 10.1016/j.brat.2009.02.004

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and interpretations in social phobia: Support for the combined cognitive biases hypothesis. *Behavior Therapy, 37*(3), 223-236. doi: 10.1016/j.beth.2006.02.001

Krishna, R., Udupa, S., George, C. M., Kumar, K. J., Viswanath, B., Kandavel, T., . . . Reddy, Y. C. J. (2011). Neuropsychological performance in OCD: A study in medication-naïve patients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 35*(8), 1969-1976. doi: 10.1016/j.pnpbp.2011.09.009

Lavy, E., Van Oppen, P., & Van Den Hout, M. (1994). Selective processing of emotional information in obsessive compulsive disorder. *Behaviour Research and Therapy, 32*(2), 243-246. doi: 10.1016/0005-7967(94)90118-X

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of Anxiety Disorders, 30*, 48-58. doi: 10.1016/j.janxdis.2014.12.016

Linkovski, O., Kalanthroff, E., Henik, A., & Anholt, G. E. (2016). Stop checking: Repeated checking and its effects on response inhibition and doubt. *Journal of Behavior Therapy and Experimental Psychiatry, 53*, 84-91. doi: 10.1016/j.jbtep.2014.12.007

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory and language*. San Diego, CA: Academic Press.

Markarian, Y., Larson, M. J., Aldea, M. A., Baldwin, S. A., Good, D., Berkeljon, A., . . . McKay, D. (2010). Multiple pathways to functional impairment in obsessive–compulsive disorder. *Clinical Psychology Review, 30*(1), 78-88. doi: 10.1016/j.cpr.2009.09.005

Mataix-Cols, D., do Rosario-Campos, M. C., & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *American Journal of Psychiatry, 162*(2), 228-238. doi: 10.1176/appi.ajp.162.2.228

McKay, D., Abramowitz, J. S., Calamari, J. E., Kyrios, M., Radomsky, A., Sookman, D., . . . Wilhelm, S. (2004). A critical evaluation of obsessive-compulsive disorder subtypes: Symptoms versus mechanisms. *Clinical Psychology Review, 24*(3), 283-313. doi: 10.1016/j.cpr.2004.04.003

Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C.-H., del Campo, N., . . . Bullmore, E. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*(12), 3223-3236. doi: 10.1093/brain/awm205

Morein-Zamir, S., Papmeyer, M., Durieux, A., Fineberg, N. A., Sahakian, B. J., & Robbins, T. W. (2013). Investigation of attentional bias in obsessive compulsive disorder with and without depression in visual search. *PLoS ONE, 8*(11), e80118. doi: 10.1371/journal.pone.0080118

Moritz, S., Fischer, B. K., Hottenrott, B., Kellner, M., Fricke, S., Randjbar, S., & Jelinek, L. (2008). Words may not be enough! No increased emotional Stroop effect in obsessive–compulsive disorder. *Behaviour Research and Therapy, 46*(9), 1101-1104. doi: 10.1016/j.brat.2008.05.005

Moritz, S., Von Muehlenen, A., Randjbar, S., Fricke, S., & Jelinek, L. (2009). Evidence for an attentional bias for washing- and checking-relevant stimuli in obsessive–compulsive disorder. *Journal of the International Neuropsychological Society, 15*(3), 365-371. doi: 10.1017/S1355617709090511

Moritz, S., & von Mühlenen, A. (2008). Investigation of an attentional bias for fear-related material in obsessive-compulsive checkers. *Depression and Anxiety, 25*(3), 225-229. doi: 10.1002/da.20294

Muller, J., & Roberts, J. E. (2005). Memory and attention in Obsessive–Compulsive Disorder: A review. *Journal of Anxiety Disorders, 19*(1), 1-28. doi: 10.1016/j.janxdis.2003.12.001

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive-compulsive symptoms. *Journal of Abnormal Psychology, 119*(1), 136-142. doi: 10.1037/a0017549

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126*(2), 220-246. doi: 10.1037//0033-2909.126.2.220

Ougrin, D. (2011). Efficacy of exposure versus cognitive therapy in anxiety disorders: Systematic review and meta-analysis. *BMC Psychiatry, 11*(1), 200. doi: 10.1186/1471-244x-11-200

Pergamin-Hight, L., Naim, R., Bakermans-Kranenburg, M. J., van Ijzendoorn, M. H., & Bar-Haim, Y. (2015). Content specificity of attention bias to threat in anxiety disorders: A meta-analysis. *Clinical Psychology Review, 35*, 10-18. doi: 10.1016/j.cpr.2014.10.005

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy, 35*(9), 793-802. doi: 10.1016/S0005-7967(97)00040-5

Rachman, S. (1998). A cognitive theory of obsessions: Elaborations. *Behaviour Research and Therapy, 36*(4), 385-401. doi: 10.1016/S0005-7967(97)10041-9

Rachman, S., & de Silva, P. (1978). Abnormal and normal obsessions. *Behaviour Research and Therapy, 16*(4), 233-248. doi: 10.1016/0005-7967(78)90022-0

Rodebaugh, T. L., Scullin, R. B., Langer, J. K., Dixon, D. J., Huppert, J. D., Bernstein, A., . . . Lenze, E. J. (2016). Unreliability as a threat to understanding psychopathology: The cautionary tale of attentional bias. *Journal of Abnormal Psychology, 125*(6), 840-851. doi: 10.1037/abn0000184

Rosa-Alcazar, A. I., Sanchez-Meca, J., Gomez-Conesa, A., & Marin-Martinez, F. (2008). Psychological treatment of obsessive-compulsive disorder: A meta-analysis. *Clinical Psychology Review, 28*(8), 1310-1325. doi: 10.1016/j.cpr.2008.07.001

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. doi: 10.1038/mp.2008.94

Salkovskis, P. M. (1985). Obsessional-compulsive problems: A cognitive-behavioural analysis. *Behaviour Research and Therapy, 23*(5), 571-583. doi: 10.1016/0005-7967(85)90105-6

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: A meta-analysis. *Psychological Medicine, 44*(6), 1121-1130. doi: 10.1017/S0033291713001803

Skapinakis, P., Caldwell, D., Hollingworth, W., Bryden, P., Fineberg, N., Salkovskis, P., . . . Lewis, G. (2016). A systematic review of the clinical effectiveness and cost-effectiveness of pharmacological and psychological interventions for the management of obsessive-compulsive disorder in children/adolescents and adults. *Health Technology Assessment, 20*(43), 1-392. doi: 10.3310/hta20430

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2014). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science*. doi: 10.1177/2167702614534210

Summerfeldt, L. J., & Endler, N. S. (1998). Examining the evidence for anxiety-related cognitive biases in obsessive-compulsive disorder. *Journal of Anxiety Disorders, 12*(6), 579-598. doi: 10.1016/S0887-6185(98)00035-8

Tata, P. R., Leibowitz, J. A., Prunty, M. J., Cameron, M., & Pickering, A. D. (1996). Attentional bias in obsessional compulsive disorder. *Behaviour Research and Therapy, 34*(1), 53-60. doi: 10.1016/0005-7967(95)00041-U

Van Bockstaele, B., Verschuere, B., Tibboel, H., De Houwer, J., Crombez, G., & Koster, E. H. W. (2014). A review of current evidence for the causal impact of attentional bias on fear and anxiety. *Psychological Bulletin, 140*(3), 682-721. doi: 10.1037/a0034834

Verhaak, C. M., Smeenk, J. M. J., van Minnen, A., & Kraaimaat, F. W. (2004). Neuroticism, preattentive and attentional biases towards threat, and anxiety before and after a severe stressor: A prospective study. *Personality and Individual Differences, 36*(4), 767-778. doi: 10.1016/S0191-8869(03)00131-4

Zvielli, A., Bernstein, A., & Koster, E. H. W. (2015). Temporal dynamics of attentional bias. *Clinical Psychological Science, 3*(5), 772–788. doi: 10.1177/2167702614551572