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Do obsessive-compulsive symptoms and contamination-related stimuli affect inhibition capacity?

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ABSTRACT

The current study set out to investigate trait versus state views regarding inhibitory deficits in participants scoring high and low on contamination fear. Furthermore, it was investigated whether inhibitory deficits are specific for contamination-related 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 obsessive-compulsive (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 contamination-related picture valence was found. Moreover, in contrast to the trait view, baseline inhibition capacity did not predict an increase in symptoms after an OCD symptom induction. Finally, contrary to expectations, participants high in contamination fear showed better inhibition than 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. 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 a common psychiatric disorder, with a lifetime prevalence of 2–3.5% and is characterized by high levels of individual suffering and 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 circuits in OCD has been established as the main neural model for OCD (Saxena & Rauch, 2000). These neural circuits underlie 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, and Mittelman (2013), Shin, Lee, Kim, and Kwon (2014), Snyder, Kaiser, Warren, and 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 related to elevated genetic risk for developing OCD. This implies that a deficit in inhibition is largely 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 precompared to post-treatment (Bannon, Gonsalvez, Croft, & Boyce, 2006). Moreover, several studies have shown that good inhibitory control can protect from negative effects of repeated checking (Linkovski, Kalanthroff, Henik, & Anholt, 2013) and priming response inhibition

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affects behavioral responses to uncertainty, which is an important aspect in OCD (Kalanthroff, Linkovski, Henik, Wheaton, & Anholt, 2016). 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 these results are mixed (see Kuelz, Hohagen, and 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. 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). Moreover, Kalanthroff, Aslan, and Dar (2017) showed that inducing mental contamination through threatened morality negatively impacted response inhibition capacity if the effects of the induction were not nullified by washing hands. Currently most research that examines the nature of inhibitory impairments 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 hypotheses of trait versus state models of inhibitory control in OCD in the context of contamination fear. 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). In order to test the effect of a contamination fear induction on inhibition, we chose to select participants scoring high on contamination fear (HCF) and participants scoring low on contamination fear (LCF). Abramowitz et al. (2014) showed that OCD symptoms are dimensional rather than categorical in frequency and severity and that similar causal and maintenance factors occur in clinical and nonclinical samples. Since response inhibition has been suggested as an endophenotype of OCD (Chamberlain et al., 2005), we would expect to observe decreased inhibition capacity in participants scoring high in contamination fear. 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. One of the methods that is used to elicit contamination fear symptoms in the lab is mental contamination (De Putter & Van Yper, 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). Furthermore, we examined whether a deficit in inhibition is specific for contamination-related stimuli. This was investigated by using negative, contamination-related, and neutral pictures in the Stop-Signal Task (SST). Finally, if inhibition capacity is indeed an endophenotype, we expected that baseline capacity to inhibit contamination-related stimuli would predict the magnitude of the increase of symptoms after an OCD symptom induction.

2. Methods

2.1. Participants

According to an a priori power analysis based on a medium effect size ($f = .25$), with $\alpha = .05$ and a power of .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 preselection 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.

2.2. Measures

2.2.1. Impulsiveness–Venturesomeness–Empathy questionnaire (I_7)

Since impulsivity can have an effect on inhibition, group differences in impulsivity were checked with the Impulsiveness subscale of the I_7 (Eysenck, Pearson, Easting, & Allsopp, 1985; Lijffijt, Caci, & Kenemans, 2005). The impulsiveness subscale of the I_7 consists of 19 dichotomous (yes/no) items.

2.2.2. 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 & Weber, 1995; Watson, Clark 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).

2.2.3. 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).