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Emotional Interference of Response Inhibition in Obsessive-Compulsive Disorder

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by

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ABSTRACT

Researchers have hypothesized that failures of inhibition are partially responsible for habitual and perseverative symptoms that are unique to Obsessive-Compulsive Disorder (OCD). It is also well known that sequelae of emotional processes are also implicated in the etiology and maintenance of obsessions and compulsions. However, little research has tested how emotional processes moderate inhibitory functions in OCD. In the present study, high contamination phobic (HCP, n = 17) and low contamination phobic (LCP, n = 30) participants completed an emotional go/no-go task, which measured the interfering effects contamination-threat processing on action restraint. The present study had a two level between-subjects-quasi-independent factor (Group: LCP vs. HCP), and a two level within-subjects-experimental-factor (Threat: Contamination vs. Neutral). The proportion of errors of commission (failures of action restraint) was the primary dependent variable. There were three predictions: 1) for the main effect of Threat, it was predicted that the visual processing of contamination images would significantly interfere with action restraint (Contamination errors of commission > Neutral errors of commission); 2) for the main effect of Group, it was predicted that HCP participants would show poorer action restraint when compared to LCP participants (HCP errors of commission > LCP errors of commission); 3) for the Group x Threat interaction, it was predicted that the visual processing of contamination images would interfere more with action restraint among HCP than LCP participants (Contamination errors of commission among HCP participants > Neutral errors of commission among HCP participants, Neutral errors of commission among LCP participants, and Contamination errors of commission among LCP participants). Predictions 1 and 3 were supported by results while results failed to support the second prediction. These data suggest that the processing of emotionally arousing imagery interferes with action restraint and the magnitude

of this effect is greater among an analogue OCD sample reporting contamination symptoms. These findings are clinically relevant and significantly extend etiological models of OCD by integrating basic neurocognitive and affective mechanisms. The unique and complimentary roles of emotional, attentional, and inhibitory processes in the etiology and maintenance of obsessions and compulsions are explored and updates to models of OCD are discussed.

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I. INTRODUCTION

A. Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD) is a chronic and intractable condition characterized by severe obsessions and/or compulsions that cause an individual significant impairment (American Psychiatric Association, 2000; Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999). Once thought to be relatively rare, recent research has estimated the lifetime prevalence of OCD to be between 1% and 3.3% (Kessler, Chiu, Demler, & Walters, 2005). According to some estimates, this would make OCD the second most common anxiety disorder and the fourth most common psychiatric illness (Karno, Golding, Sorenson, & Burnam, 1988; Kessler et al., 2005). The most commonly accepted operational definition of OCD is provided by the Diagnostic and Statistical Manual of Mental Disorders [DSM-IV-TR; American Psychiatric Association, 2000 (APA), pp. 462-463]. According to the DSM-IV-TR, an individual can meet diagnostic criteria for OCD if the following five criteria (items A-E) are satisfied:

A. Either obsessions or compulsions:

Obsessions as defined by (1), (2), (3), and (4):

(1) recurrent and persistent thoughts, impulses, or images that are experienced at some time during the disturbance as intrusive and inappropriate and that cause marked anxiety or distress

(2) the thoughts, impulses, or images are not simply excessive worries about real-life problems

(3) the person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action

(4) the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)

Compulsions as defined by (1) and (2):

(1) repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly

(2) the behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive

B. At some point during the course of the disorder, the person has recognized that the obsessions or compulsions are excessive or unreasonable. ***Note:*** *This does not apply to children.*

C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.

D. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorder; hair pulling in the presence of Trichotillomania; concern

with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges or fantasies in the presence of a Paraphilia; or guilty ruminations in the presence of Major Depressive Disorder).

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

B. Subtypes and Dimensions in OCD

The quality of obsessions and compulsions can span the range of human experience, but they tend to cluster within reliable factor dimensions (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008; McKay et al., 2004). Although the number of dimensions varies within the published literature, recent qualitative analyses of the adult Yale-Brown Obsessive-Compulsive Symptoms Checklist [Y-BOCS; (Goodman, Price, Rasmussen, & Mazure, 1989)] suggested that there are four reliable factor dimensions of obsessions and compulsions among adults diagnosed with OCD (Bloch et al., 2008, pp. 1532)

1) "Symmetry"; symmetry obsessions and repeating, ordering, and counting compulsions; 2) "Forbidden Thoughts"; aggression, sexual, religious, and somatic obsessions and checking compulsions; 3) "Cleaning"; cleaning and contamination obsessions and compulsions; and 4) "Hoarding"; hoarding obsessions and compulsions.

In general, the findings from the meta-analyses of Bloch and colleagues (2004) were consistent with a majority of studies that attempted to create factors based on symptoms. There are two important limitations to much of this research (McKay et al., 2004). First, most factor-analytic studies on obsessions and compulsions neglected the "others" categories of the Y-BOCS

symptom checklist, which contains a number of symptoms that are common among those diagnosed with OCD, particularly mental compulsions. Second, factor analyses are used to create latent dimensions which do not assign cases to a particular subgroup (e.g., washers vs. checkers). These limitations have has been addressed with cluster analyses (Abramowitz, Franklin, Schwartz, & Furr, 2003; Calamari, Wiegartz, & Janeck, 1999; Calamari et al., 2004).

A cluster analysis of the Y-BOCS symptom checklist, excluding the "other" symptoms, found five clusters that closely paralleled the factors reported by Bloch and colleagues, including: harming, hoarding, contamination, certainty, and obsessionals (Calamari et al., 1999). In a replication study that included "other" symptoms from the Y-BOCS symptom checklist, Calamari and colleagues (2004) found seven clusters, including: contamination, harming, hoarding, obsessionals, symmetry, certainty, and contamination/harming. The novel contamination/harming cluster was mostly characterized by contamination and washing symptoms in relation to moral or spiritual impurity. Finally, Abramowitz and colleagues (2003) conducted a cluster analysis using a modified Y-BOCS symptom checklist that appropriately measured mental rituals. In partial keeping with Calamari and colleagues (2004), Abramowitz and colleagues (2003) identified five factors, including: harming, contamination, hoarding, unacceptable thoughts, and symmetry.

C. Emotion in OCD

The most recent edition of the DSM (DSM-IV-TR; APA, 2000) categorized OCD as an anxiety disorder, which is consistent with over a century of research and theory (Bartz & Hollander, 2006; Tynes, White, & Steketee, 1990)[1]. At a fundamental level, cognitive-behavioral models of the manifestation and maintenance of OCD differ very little from models of other anxiety disorders (Rachman, 1997; Rachman, 2002; Rachman, 2004; Salkovskis, 1985). Most

cognitive-behavioral theorists assert that obsessions and compulsions arise and are maintained by pathological levels of negative affect, unique interpretations of intrusions, classical, and operant conditioning. Those diagnosed with OCD report higher levels of trait anxiety than non-clinical samples, but they report lower levels of trait anxiety compared to participants diagnosed other forms of disordered anxiety (Antony, Bieling, Cox, Enns, & Swinson, 1998; Kennedy, Schwab, Morris, & Beldia, 2001). The affective correlates of obsessions and compulsions are not, however, restricted to fear and anxiety. Research has reliably shown that one of the most common dimension of obsessions and compulsions (contamination and washing, respectively) are also motivated by disgust (Olatunji & McKay, 2007; Olatunji & Sawchuk, 2005). While most cognitive-behavioral models of the manifestation and maintenance of OCD differ very little from models of other anxiety disorders, there are a several unique factors that distinguish OCD from other anxiety disorders. Perhaps most important are differences in neuropsychological functions and the sequelae of related neurobiological factors (see Cox, 1997; Greisberg & McKay, 2003; Otto, 1992; and Schultz, Evans, & Wolff, 1999 for reviews).

D. Attention in OCD

Attentional functions are carried out by three distinct subsystems that are separate from perceptual and motor functions (Posner & Boies, 1971). These subsystems include: 1) the alerting network (activation of the attentional system); 2) the orienting network (orienting of attention to a source) and; 3) the executive attentional control network (conflict resolution and attention inhibition; (Posner & Petersen, 1990). Research has provided direct and indirect evidence to suggest that the executive attentional control network is impaired among individuals diagnosed with OCD (Armstrong, Zald, & Olatunji, 2011). This is an important proposition given that contemporary theoretical accounts of OCD have proposed that impaired inhibition of

attentional resources fosters symptoms of OCD (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). Examples of executive attentional control deficits in OCD include impaired ability to sustain attention (Gambini, Abbruzzese, & Scarone, 1993; Kim, Park, Shin, & Kwon, 2002; Morein-Zamir et al., 2010), over-focused attention to irrelevant stimuli [distractibility; (Nelson, Early, & Haller, 1993)], and delayed attention disengagement (Cisler & Olatunji, 2010; Schmidtke, Schorb, Winkelmann, & Hohagen, 1998).

Research has shown that analogue samples and samples of individuals diagnosed with OCD evidence attentional biases toward affectively arousing information, particularly symptom specific information (Foa, Ilai, McCarthy, Shoyer, & Murdock, 1993; Foa & McNally, 1986; Lavy, Van Oppen, & Van Den Hout, 1994; Tata, Leibowitz, Prunty, Cameron, & Pickering, 1996). More specifically, recent research suggests that attentional biases in OCD are likely due to prolonged maintenance of attention on and/or difficulty disengaging attention from emotionally arousing stimuli, as evidenced by: 1) maintenance of attention toward symptom specific pictorial stimuli (Armstrong, Sarawgi, & Olatunji, 2012); 2) difficulty disengaging attention from symptom specific pictorial stimuli (Cisler & Olatunji, 2010); 3) prolonged maintenance of attention toward highly-arousing/negatively-valenced pictorial stimuli that are not symptom specific [e.g., fearful faces (Armstrong, Olatunji, Sarawgi, & Simmons, 2010)]; 4) difficulty disengaging attention from highly-arousing/negatively-valenced pictorial stimuli that are not symptom specific (Cisler & Olatunji, 2010); and more recently, 5) difficulty disengaging attention from highly-arousing/*positively*-valenced pictorial stimuli (i.e., erotica) that are not symptom specific (Olatunji, Ciesielski, & Zald, 2011).

It should be noted that the evidence for any form of attentional bias in OCD is controversial and notoriously difficult to replicate across laboratories, assessment paradigms, or

samples (Summerfeldt & Endler, 1998). There are several theoretical explanations for this unreliability. The most common explanation hinges on the heterogeneity of OCD, with greater evidence of attentional biases among participants who report primary symptoms of contamination obsessions and washing compulsions relative to all other symptom subtypes [e.g., primary checking or symmetry types (Summerfeldt & Endler, 1998)]. There is also evidence that detection of attentional biases in OCD may be dependent on the procedures used. For example, task complexity, stimulus presentation times, stimulus types, and latency between affective distractor offset and target onset can significantly affect results (Cisler & Olatunji, 2010; Kyrios & Iob, 1998; Moritz, Wendt, Jelinek, Ruhe, & Arzola, 2008). Also, one study has shown that attentional biases in OCD may attenuate over the course of experimental trials and blocks (Amir, Najmi, & Morrison, 2009). Therefore, researchers may be more likely to detect attentional biases when using briefer assessment tools or, similarly, biases may be more evident within earlier assessment blocks than later blocks.

E. Inhibition in OCD

A vast majority of theoretical and empirical literatures suggest that failures of inhibition may underlie the etiology and expression of OCD (Chamberlain et al., 2007; Fineberg et al., 2009; Graybiel & Rauch, 2000; Lipszyc & Schachar, 2010; Schultz, Evans, & Wolff, 1999). For the present manuscript, the term inhibition is constrained to refer to forms of intentional executive control (Aron, 2007). Executive inhibition is not a unitary construct (Friedman & Miyake, 2004; Nigg, 2000). Nigg (2000, p. 237) proposed that the higher order construct of executive inhibition be split into four separate components, including (words in italics added for clarity): “1) Interference control: prevent interference due to resource or stimulus competition; 2) Cognitive inhibition: suppress nonpertinent ideation to protect working memory/attention; 3)

Behavioral (*response*) inhibition: suppress prepotent [automatic/prepared/cued] response; and 4) Oculomotor (*inhibition*): suppress reflexive saccade."

Friedman and Miyake (2004) provided convincing data that simplified and challenged Nigg's heuristic. In keeping with previous literature, Friedman and Miyake abandoned the construct of cognitive inhibition as it lacks basic psychometric reliability and validity. This is in keeping with MacLeod and colleague's (2003; 2007) criticisms of cognitive inhibition. Namely, there is simply insufficient evidence to support the proposition that negative priming and directed forgetting tasks – the two tests most often associated with cognitive inhibition – are actually dependent on any inhibitory processes (MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003; MacLeod, 2007). Miyake and Friedman also proposed two components of interference inhibition (referred to herein as resistance to interference[2]): 1) *resistance to distractor interference* ("ability to resist or resolve interference from information in the external environment that is irrelevant to the task at hand"); and 2) *resistance to proactive interference* ("ability to resist memory intrusions from information that was previously relevant to the task but has since become irrelevant") (pp. 104-105). It is worth noting that Friedman and Miyake's resistance to proactive inhibition is akin to Nigg's cognitive inhibition. Finally, Friedman and Miyake provided data to support the merging of Nigg's oculomotor and response inhibition into what they described as prepotent response inhibition.

More recent research has shown that there are even multiple forms of response inhibition (Aron, 2007; Eagle, Bari, & Robbins, 2008; Schachar et al., 2007). Instead of Nigg's behavioral and oculomotor inhibition, recent data suggest that response inhibition can be separated by the temporal relations between the initiation and suppression of actions. As such, response inhibition can be separated into two factors, *action restraint* (i.e., inhibition before initiation) and *action*

cancellation (i.e., inhibition following initiation) (Aron, 2007; Eagle, Bari, & Robbins, 2008; Schachar et al., 2007). In summary, a modern conceptualization of executive inhibition would likely include two higher-order executive inhibitory factors, each with two lower order factors that are unique in their temporal qualities (see Figure 2).

It has been hypothesized that deficits in resistance to interference (often coined "cognitive inhibition") may partially account for the ease with which obsessions can enter into and take over the consciousness of those diagnosed with OCD (Chamberlain et al., 2005). Experimental and neurobiological research have shown that the classic Stroop effect [difficulty naming ink color of printed words [e.g., "blue"] that are written in incongruent colored ink (Stroop, 1935)] is a measure of resistance to interference and prepotent response inhibition (Friedman & Miyake, 2004; Vendrell et al., 1995). Several studies have shown that adults diagnosed with OCD evidence greater Stroop interference when compared to a healthy control sample (Hartston & Swerdlow, 1999; Martinot et al., 1990; Penades et al., 2007). This effect not only remained but appeared to be larger when samples diagnosed with OCD were compared to samples diagnosed with panic disorder (Bannon, Gonsalvez, Croft, & Boyce, 2002; 2006; Bannon, Gonsalvez, & Croft, 2008). One study that failed to detect significant differences in Stroop interference among a sample diagnosed with OCD, nevertheless did report abnormal patterns of neural activation during the Stroop procedures [e.g., anterior cingulate cortex (ACC) and right caudate nucleus hypoactivity (Nakao et al., 2005)]. This is an important finding as the ACC is believed to be largely responsible for error monitoring, resistance to interference, and the processing of affective information, all of which have clear implications for obsessions and compulsions (Bush et al., 1998; Bush, Luu, & Posner, 2000; Saxena, O'Neill, & Rauch, 2009; Whalen et al., 1998).

There is strong evidence that individuals diagnosed with OCD perform more poorly on tests designed to measure response inhibition than healthy controls and anxious controls. Two early studies by Rosenberg and colleagues (Rosenberg, Dick, O'Hearn, & Sweeney, 1997; Rosenberg et al., 1997) showed that OCD-diagnosed children performed more poorly than healthy controls on a task that requires simple inhibition of eye movements (antisaccade). This finding has been supported by more recent research with adult samples (Lennertz et al., 2012). Similarly, a recent meta-analysis (Lipszyc & Schachar, 2010) found that, collapsed across 4 studies, individuals diagnosed with OCD performed more poorly on the stop-signal task – a measure of action cancellation – than healthy controls, and the magnitude of this effect was medium-large and reliable (g = .77, p < .01). Moreover, this same meta-analysis found no reliable differences in stop-signal performance between healthy controls and those diagnosed with an anxiety disorder other than OCD (g = .09).

A host of research has also shown that participants diagnosed with OCD perform more poorly on the go/no-go task – a measure of action restraint (go/no-go errors of commission) – when compared to healthy controls and anxious controls (Aycicegi, Dinn, Harris, & Erkmen, 2003; Bannon, Gonsalvez, Croft, & Boyce, 2002; 2006; Penades et al., 2007; Watkins et al., 2005). Two studies failed to detect differences in action restraint between healthy controls and participants diagnosed with OCD (Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Herrmann, Jacob, Unterecker, & Fallgatter, 2003).

There is convincing evidence that deficits in response inhibition in OCD are trait-like and heritable (Bannon et al., 2006; Lennertz et al., 2012; Menzies et al., 2007). This has led several theorists to propose that deficient response inhibition may serve as a developmental endophenotype of OCD (Chamberlain et al., 2008; Chamberlain & Menzies, 2009; Chamberlain

et al., 2005; Fineberg et al., 2009; Menzies et al., 2008; Rosenberg & Keshavan, 1998; Taylor, 2012). Two studies have shown that unaffected (symptom free) first-degree relatives of participants diagnosed with OCD performed more poorly on tests of response inhibition when compared to healthy controls (Lennertz et al., 2012; Menzies et al., 2007). Neither study detected significant differences in response inhibition between participants diagnosed with OCD and their first-degree relatives. These findings and the inhibitory endophenotype theory of OCD are consistent with genetic research which has shown that individual differences in response inhibition abilities (as measured by a latent combination of the Stroop, stop-signal, and antisaccade tasks) are approximately 99% heritable (Friedman et al., 2008)].

F. Emotion and Inhibition

Research has shown that emotions and executive inhibitory functions play an important role in OCD. However, very little published research has attempted to integrate affective and inhibitory processes to further the understanding of OCD. A limited body of research has also shown that, among obsessive-compulsive (OC) samples, the processing of emotionally arousing stimuli interferes with resistance to interference[3]. As noted by Morein-Zamir and colleagues (2010), findings have been inconsistent across studies that have tested affective interference of inhibitory processes in OCD. Inconsistent findings may be an artifact of task complexity (Bannon et al., 2008; Morein-Zamir et al., 2010) or the use of interference stimuli that are difficult to process rapidly and may be nominally arousing [i.e., emotional words (Bannon et al., 2008; Tolin, Hamlin, & Foa, 2002; Wilhelm, McNally, Baer, & Florin, 1996). Moreover, all but one (Bannon et al., 2008) of the aforementioned studies attempted to assesses cognitive inhibition with directed forgetting and negative priming paradigms, which may be tasks of

limited validity (MacLeod, 2007). As noted by Krikorian and colleagues (2004), no study has directly tested how affective factors might interfere with response inhibition in OCD.

A recent publication by Morein-Zamir and colleagues (2013) showed that adults diagnosed with OCD performed more poorly than healthy controls on a go/no-go task that incorporated punitive feedback. An increase in commission errors following punishment was also highly correlated with self-reported symptom severity. Although not a direct test of affective interference, this study suggests that arousal (in anticipation of punishment) may cause greater response inhibition interference among those diagnosed with OCD than healthy controls. This interpretation is consistent with several studies that used non-selected samples to test how affective factors interfere with response inhibition. In a rather ingenious study, Pessoa and colleagues (2012) first conditioned participants to fear one auditory stimulus (e.g., "one") and not the other (e.g., "two") by pairing one stimulus (CS+) with shock and not the other (CS-). Participants then completed a stop-signal task that utilized both auditory stimuli as the stop-signal. Pessoa and colleagues were then able to test the specific effects of early threat activation on action cancellation. Consistent with their hypotheses, they found that action cancellation was poorer on threat stop-signal trials (CS+) than no-threat stop-signal trials (CS-). Moreover, this effect was largely explained by physiological indices of affective arousal following the CS+ stop-signal (galvanic skin conductance).

Several studies have also investigated how the processing of emotional stimuli interferes with response inhibition (De Houwer & Tibboel, 2010; Pessoa et al., 2012; Verbruggen & De Houwer, 2007). Separate from the previously mentioned study, Pessoa and colleagues (2012) used emotional faces (happy, fearful, and neutral) as visual stop signals. Counter to their hypotheses, they found that the emotional faces facilitated action cancellation and this effect was

invariant across face valence. Verbruggen and DeHouwer (2007) showed that the 250 ms presentation of emotionally-arousing pictures immediately prior to target stimuli in a classic stop-signal paradigm resulted in delays in action cancellation. Although Verbruggen and DeHouwer found that pictures that were high in emotional arousal caused delays in action cancellation, they did not detect a significant effect of valence. Similarly, De Houwer and Tibboel (2010) presented emotional pictorial stimuli for 250 ms prior to target and no-go stimuli during a classic go/no-go task. Pictures that were high in emotional arousal interfered with no-go trials (more errors of commission) and delayed go trials. De Houwer and Tibboel reported almost identical interference effects among high arousing pictures that were positively valenced (e.g., nude model) and high arousing pictures that were negatively valenced (e.g., mutilated hand). Taken as a whole, these studies show that the processing of emotional information can modulate response inhibition, but timing and arousal play important roles in the direction of influence.

Accoding to De Houwer and Tibboel (2010), there were two competing hypotheses that might explain the interfering effects of emotion on response inhibition and base reaction time (i.e., go-signal trials): 1) the freezing account, and 2) the attentional account. According to the freezing account, emotionally arousing stimuli can cause brief and incomplete tonic immobility, which in should slow prepotent responses and thus increase the probability of an action being inhibited. According to the attentional account, emotionally arousing stimuli capture and hold attention. Given that attention is required for response inhibition, the decrease in attentional resources caused by the processing of emotional information results in a diminished capacity to exercise motor control (e.g., delayed response inhibition). If the freezing account were accurate, then participants would have evidenced slower go-signal reaction times but fewer rates of commission errors on arousing relative to non-arousing trials. If the attentional account were

accurate, then participants would have evidenced faster go-signal reaction times and higher rates of commission errors on arousing relative to non-arousing trials. Attention is a limited resource (Huang & Pashler, 2005; Kane & Engle, 2002) and emotionally arousing information captures and holds attention more so than emotionally neutral or personally irrelevant information (Fox, Russo, Bowles, & Dutton, 2001; Koster, Crombez, Verschuere, & De Houwer, 2004; Koster, Verschuere, Crombez, & Van Damme, 2005). This attentional capture affects other cognitive processes that are also dependent on attentional resources. Response inhibition procedures, and particularly, the go/no-go paradigm, require attentional resources. When stimuli capture, hold, and residually affect attention, then response inhibition should be impaired (Verbruggen & Logan, 2008b). This is precisely what was found by De Houwer and Tibboel (2010) and Verbruggen and De Houwer (2007). As such, the most tenable interpretation of the findings reported by Verbruggen and colleagues and De Houwer and Tibboel is that emotionally arousing information interfered with response inhibition via attention. Said otherwise, attention was the mediating mechanism between emotional processing and response inhibition.

G. Present Study

If the attentional account of emotional interference of response inhibition is correct, then interfering effects of emotionally arousing stimuli on response inhibition should be greater among populations with attentional biases toward emotionally arousing information. Participants diagnosed with OCD and analogue obsessive-compulsive (OC) samples have reliably shown biases toward emotionally arousing information, particularly disorder relevant information. Therefore, the interfering effects of disorder relevant stimuli on response inhibition should be intensified among participants diagnosed with OCD and those reporting elevated OC symptoms. This is particularly germane to OCD as clinical examples of failed response inhibition occur

during moments that are brimming with emotional information. Take, for example, compulsive hand washing. Compulsive hand washing, as opposed to non-pathological hand washing, can be explained by a strong motivation to wash and a failure to stop washing (i.e., failures of inhibition). The internal and contextual features that motivate washing are emotional in nature (e.g., disgust and anxiety) and can, therefore, be assumed to potentially interfere with inhibition.

The present study addresses critical gaps in the extant literature by testing affective interference of action restraint in OCD. The proposed study utilized two participant groups, high contamination phobic (HCP) and low contamination phobic (LCP). All participants will complete a symptom-specific version of the emotional go/no-go task (DeHouwer & Tibboel, 2010), which was designed to test the interfering effects of contamination-threat stimuli on action restraint. The primary dependent variable of the go/no-go task is proportion of errors of commission (going when required to restrain action). The contamination-threat go/no-go has one within-subjects main effect with two levels [Threat (contamination vs. neutral)]. The present study will, therefore, utilize a 2 (Group: HCP vs. LCP) x 2 (Threat: contamination vs. neutral] factorial design with errors of commission as the primary dependent variable. Additional dependent variables will also be analyzed for exploratory purposes, including errors of omission (not going on go trials), no-go RT [NGRT (latency of key press on no-go trials)], and go RT [GRT (latency of key press on go trials)]; each of which will be analyzed at both levels of the within subjects factor.

II. STUDY HYPOTHESES

1) For the main effect of Threat:

   H_o: The visual processing of contamination imagery will not interfere with action restraint (Contamination- Errors of Commission = Neutral- Errors of Commission)

H_1: The visual processing of contamination imagery will interfere with action restraint (Contamination- Errors of Commission > Neutral- Errors of Commission)

2) For the main effect of Group:

H_o: Collapsed across both levels of threat, HCP participants will show no difference in action restraint when compared to LCP participants (Errors of Commission among HCP participants = Errors of Commission among LCP participants)

H_1: Collapsed across both levels of threat, HCP participants will show poorer action restraint when compared to LCP participants (Errors of Commission among HCP participants > Errors of Commission among LCP participants)

3) For the Group x Threat interaction:

H_o: The visual processing of contamination imagery will not differentially interfere with action restraint among HCP and LCP participants (Contamination errors of commission among HCP participants = Neutral errors of commission among HCP participants, Neutral errors of commission among LCP participants, and Contamination errors of commission among LCP participants).

H_1: The visual processing of contamination imagery will interfere with action restraint more among HCP than LCP participants (Contamination errors of commission among HCP participants > Neutral errors of commission among HCP participants, Neutral errors of commission among LCP participants, and Contamination errors of commission among LCP participants).

III. METHODS

A. Materials

1. Affective Interference Stimuli. De Houwer and Tibboel (2010) used pictorial interference stimuli in their emotional go/no-go task. Furthermore, affective pictorial stimuli are processed more rapidly (Gläscher & Adolphs, 2003), achieve greater amygdala activation (Kensinger & Schacter, 2006; Markowitsch, 1998) and are exert a greater effect on attention (Moritz et al., 2008) than words. Therefore, pictorial stimuli, rather than words, were considered more appropriate interference stimuli for the emotional go-no/go task.

The contamination and neutral pictures from Armstrong and colleagues (2012) were utilized as interference stimuli for the present study. These are 24 pictures (12 contamination, 12 neutral), most of which were selected from the International Affective Picture Set (IAPS) (Lang, Bradley, & Cuthbert, 1997) and several of which were found on the internet. Armstrong and colleagues validated this picture set and showed that the contamination pictures were rated as more unpleasant, arousing, fearful, and disgusting than the neutral picture set. Six separate affectively neutral (low arousal, neutral valence) images were selected from the IAPS to be used in practice trials. All pictures were 12cm wide x 11 cm high.

2. Emotional Go/No-Go (Figure 3). The emotional go/no-go in the present study was identical to that used by De Houwer and Tibboel (2010) except different pictures and emotion categories were used in the present study. Participants first received written instructions comparable to those outlined by De Houwer and Tibboel. The experimenter also described the procedures in a casual fashion to the participant and questions were answered prior to beginning practice blocks. In brief, participants were instructed that they would be completing a series of test trials that would begin with the presentation of a picture followed by either a go symbol or no-go symbol. They were told that their task was to press the spacebar as fast as possible every time they saw the go-symbol and to do nothing when they saw no-go symbol. They were also

told that speed was very important on go-trials and, because of this, some errors were okay as long as they were doing their best to go as fast as they could while still doing their best to resist pushing the spacebar following the presentation of the no-go target.

Practice and test trials started with the presentation of a 12cm x 11cm white rectangle in the center of the screen. After 500 ms, one of the IAPS pictures appeared in the center of the square for 250 ms. A go symbol (character A) or no-go symbol (character B) then appeared in the middle of the screen until the participant responded or until 400 ms elapsed. When participants took longer than 400 ms to respond on go trials, the software recorded a reaction time of 400ms. Characters A and B were either *§* or #, counterbalanced as go or no-go symbols across participants. If the participant did not respond within 400 ms on go trials, then "TOO SLOW" appeared on the screen for 200 ms. Each new trial began 600 ms after a response or feedback.

The emotional go/no-go began with a block of 24 practice trials in which neutral IAPS pictures were followed by the go symbol 12 times and the no-go symbol 12 times. If a participant received more than 6 "TOO SLOW" warning messages during practice, then the experimenter had the participant complete another practice block. This was done to stress the importance of speed to participants who appeared to strategically slow their responding to prevent errors of commission. No participant was required to complete more than 2 practice blocks. Participants then completed 2 test blocks, each starting with 6 warm-up trials followed by 96 test trials. The warm-up trials were randomly drawn from the practice block. During the test trials, each of the 24 test-trial pictures was presented 8 times and was followed by the go symbol the no-go symbol and equal number of times. This resulted in 24 neutral go, 24 neutral no-go, 24 contamination go, and 24 contamination no-go trials per block. As such, each participant completed a total of 192

test trials (48 test trials per trial type). The order of test trials was determined randomly within each test block for each participant. The primary dependent variable, errors of commission, was measured via proportion of response errors (commission) relative to total no-go trials. Secondary variables included errors of omission (proportion of response errors on go trials), go RT (GRT; average RT on go trials), and no-go RT (NGRT; average RT on no-go trials). All 4 of the emotional go/no-go variables were calculated across each of the two threat categories (contamination vs. neutral) for a total of eight measurement variables.

3. *Dimensional Obsessive Compulsive Scale* (DOCS; Appendix A) (Abramowitz et al., 2010) is a 20-item, self-report measure that assesses severity of four symptom dimensions of OCD [5-items each: contamination (DOCS-C), responsibility (DOCS-R), unacceptable thoughts (DOCS-O), and symmetry (DOCS-S)]. The DOCS uses a 5-point Likert-type scale ranging from 0 to 4. The total score of the DOCS ranges from zero to 80 and the subscale scores each range from 0 to 20. A DOCS-Total cut-off score of 18 has strong sensitivity (78%) and specificity (78%) when used to differentiate between individuals diagnosed with OCD and nonclinical adults (Abramowitz et al., 2010). The DOCS is a valid and reliable measure of OC symptoms, with previous research reporting Cronbach's alpha coefficients ranging from .83 to .96 for each of the subscale scores. Cronbach's alpha was high for the DOCS-Total score ($\alpha = .96$) and moderate to high for all DOCS subscales ($\alpha_{contamination} = .91$, $\alpha_{responsibility} = .92$, $\alpha_{obsessions} = .87$, $\alpha_{symmetry} = .93$) within the present sample.

4. Generalized Anxiety Disorder – 7 (GAD-7; Appendix B) (Spitzer, Kroenke, Williams, & Lowe, 2006) is a 7-item, self-report measure of anxiety. While originally designed to assess the severity of Generalized Anxiety Disorder (GAD), the GAD-7 can also be used to assess levels of trait anxiety and can be useful in discriminating between individuals diagnosed with an

anxiety disorder [i.e., GAD, Panic Disorder, Social Anxiety Disorder, and Posttraumatic Stress Disorder (PTSD)] and those with no anxiety disorder (Kroenke, Spitzer, Williams, Monahan, & Lo□we, 2007). The GAD-7 uses a 4-point Likert-type scale ranging from 0 'not at all' to 3 'nearly every day' to assess symptoms of anxiety (e.g., "Feeling nervous, anxious, or on edge") over the last 2 weeks. The GAD-7 total score ranges from zero to 21. A GAD-7 cut-off score of 8 has strong sensitivity (77%) and specificity (82%) when used to differentiate between adults diagnosed with an anxiety disorder and those with no anxiety disorder diagnosis (Kroenke et al., 2007). Lo□we and colleagues (2008) have shown that the GAD-7 is a valid and reliable measure of symptoms of anxiety, with a Cronbach's alpha coefficient of .89. Cronbach's alpha was moderate, $\alpha = .81$, within the present sample.

B. Participants

Inclusion in the HCP group required DOCS-C and DOCS-Total scores greater than or equal to 7 and 18, respectively, during laboratory assessment. Inclusion in the LCP group required DOCS-C and DOCS-Total scores less than or equal to 3 and less than 18, respectively, during laboratory assessment. Abramowitz and colleagues (2010) reported a mean DOCS-C score of 6.53 (*SD* = 6.4) among a large sample diagnosed with OCD and a mean DOCS-C score of 3.07 (*SD* = 2.76) and 2.03 (*SD* = 2.89) among samples diagnosed with other anxiety disorders and student samples, respectively. As such, the selected cut-off scores ensured that HCP participants had clinically significant symptoms of OCD, particularly contamination obsessions and washing compulsions, while also ensuring that these symptoms were minimal among LCP participants.

Seven hundred eighty six participants completed the DOCS-C scale during a mass screening of Introductory Psychology students at a large southern university. Only the DOCS-C

subscale was administered due to page and cost restrictions during screening procedures. Participants who scored ≥ 7 or ≤ 3 on the DOCS-C were contacted by the primary author to schedule a testing session, which resulted in the scheduling of 86 participants. From those scheduled, 18 HCP and 31 LCP participants scored above or below their respective DOCS-C and DOCS-Total cut-off scores during the testing session. Two participants (one from each group) were not included in the final analyses due to problematic data (see *Data Preparation*). As such, the final sample size was 47.

A majority of participants were female (63.8%) and Caucasian (83%). The average age of participants was 19.28 years (*SD* = 1.58, range = 18-28). HCP and LCP groups did not significantly differ in age, gender, or race, all *ps* > .10, (Table 1). Only two participants reported that they were taking psychotropic medication at the time of the experiment. One participant in the LCP group reported that she was prescribed Adderall and one participant in the HCP group reported that she was prescribed Prozac. HCP participants scored higher than LCP participants on all DOCS subscales and the GAD-7, all *ps* < .01 (Table 1). All participants were offered and provided either class credit toward a course requirement or $20 cash as compensation for their participation in the present study. A sizeable majority of participants (91.5%) received class credit.

C. Procedures

All participants were tested individually in a dark 6x8 room. All procedures were completed on a Dell Optiplex 745 PC with an Intel Duo Core processor. Pictorial stimuli were presented on a 36cm by 29cm flat screen Dell monitor set at a 1280x1024 resolution and 60 hz refresh rate. All participants were seated approximately 18cm from the screen. The emotional go/no-go task was programmed and administered using Inquisit software (version 3.0.6.0,

Milliscond Software) and participant responses were registered via a standard USB keyboard. Each experimental session began with the completion of an IRB approved informed consent. Participants then completed the emotional go/no-go task followed by a questionnaire battery. All participants were then fully debriefed and offered appropriate referral information.

IV. RESULTS

A. Data Analytic Approach

Multilevel modeling (MLM) – also known as hierarchical linear modeling or mixed modeling – was utilized to test all hypotheses and most secondary analyses[4]. MLM was chosen due to unequal sample sizes between groups and the fact that observations across the within subjects factor were not independent, both of which would have violated major assumptions of mixed factor ANOVA but are not required for MLM (Tabachnick & Fidell, 2008). Moreover, MLM improves reliability of parameter estimates and, thus, improves Type I errors rates when compared to mixed factor ANOVA.

Models were specified in an iterative, additive fashion, whereby fixed and random factors were added to the baseline (null) model one by one to ensure that the final model evidenced greater goodness of fit relative to the baseline model. For each hypothesis test, models were specified and compared in the following order: 1) baseline model; 2) level 1 (Threat), 3) level 2 (Threat and Group), and 4) full model (Threat, Group, and Threat by Group) (Field & Wright, 2011; Tabachnick & Fidell, 2008). Despite small sample sizes, full maximum likelihood (ML) – as opposed to restricted maximum likelihood (REML) – was used to calculate parameter estimates. This was largely to allow for comparisons between non-nested models. The default covariance structures of the repeated effect (diagonal covariance) were utilized for all analyses as the repeated measurement (Threat) only had two levels, which were not heterogeneous and the

random effects (intercept and subjects) only had one level. Type III sum of squares were used to test all omnibus fixed effects. Finally, correlational and hierarchical regression analyses were used for exploratory analyses aimed at testing mechanisms of action. All analyses were carried out with SPSS 20 (IBM Corp.). The SPSS syntax and data matrix can be found in Appendices C and D, respectively.

B. Data Preparation and Assumption Testing

Missing values analyses revealed that less than 1% of data were missing from both the DOCS and GAD-7. Little's missing completely at random (MCAR) test was utilized to ensure that data were MCAR. Results indicated that data were MCAR, X^2 (26) = 21.10, p = .74. Missing data were imputed using estimation maximization. No data were missing from the emotional go/no-go.

To enhance interpretability of parameter estimates, errors of commission and omission were transformed from decimal percentages to full percentages [e.g., from .21 to 21 (21%)]. Data from the emotional go/no-go were inspected and cleaned according to the standards outlined by De Houwer and Tibboel (2010). Reaction times below 150 ms were removed from the data matrix. This resulted in the removal of 21 trials, less than 1% of all data. As previously mentioned, two participants were not included in any analyses as their data were indicative of poor effort or strategic performance. Despite appropriately completing 24 trials of go/no-go practice, these two participants performed below chance on go trials (i.e., errors of omission > 50%) and, as such, their performance on no-go trials was nearly perfect.

Data from the emotional go/no-go were inspected for normality. Visual inspection of the data (i.e., histograms and P-P plots) and test statistics suggested that the proportion of neutral errors of commission was significantly positively skewed (skewness = 1.16, *SE* = .35), and

neutral NGRT was significantly negatively skewed (skewness = -1.28, *SE* = .36) and leptokurtic (kurtosis = 3.82, *SE* = .70). When collapsed across the within subjects factor, none of the emotional go/no-go variables evidenced significant skewness or kurtosis; although NGRT was still somewhat leptokurtic (kurtosis = 1.38, *SE* = .50). The severity of skewness of neutral errors of commission was, therefore, deemed acceptable and errors of commission were analyzed in their raw form (not transformed). The non-normality of NGRT and particularly neutral-NGRT was deemed problematic. Given that this variable was only included in secondary analyses and the fact that there are no empirically supported methods for transforming leptokurtic data, these data were also analyzed in their raw form. However, interpretations of results from analyses using NGRT as a DV were treated as less reliable and interpreted more cautiously.

C. Primary Analyses

The first series of MLM models were carried out to test all primary hypotheses (the effects of Threat and Group on errors of commission). Akaike's Information Criterion (AIC) and Schwarz's Bayesian Criterion (BIC) of the baseline model were 696.59 and 704.22, respectively. The interclass correlation (ICC) of the baseline model was .41, suggesting adequate variance within errors of commission. The intercept of the baseline model suggested that, when collapsed across Threat and Group, participants committed, on average, 13.76% errors of commission (β = 13.76, *SE* = 1.22). The level 1 model was used to estimate the main effect of the within subjects variable (Threat). Threat was modeled as a fixed and repeated effect while the intercept was modeled as a random and fixed effect. AIC of the within subjects model was 679.52 while Schwarz's Bayesian Criterion BIC was 692.24, suggesting model improvement relative to the baseline model. The level 2 model was used to estimate the main effects of the within (Threat) and between (Group) subjects variables. Threat and Group were modeled as fixed effects, Threat

was modeled as a repeated effect, and the intercept was modeled as a fixed and random effect. AIC for the within subjects model was 680.32 while BIC was 695.58, suggesting that the addition of the Group factor did not improve model fit. However, the Group factor was retained in subsequent models because of the importance of the interaction term to hypotheses tests. Finally, the full model was identical to the level 2 model, save for the addition of the Threat by Group interaction as a fixed effect. AIC for the full model was 678.28 and BIC was 696.09. While BIC suggests a slight increase in model fit relative to the baseline model, it also suggests a poorer fit relative to the level 1 and level 2 models. Conversely, AIC suggests superior fit relative to all models with fewer parameter estimates. Given AIC estimates and the fact that BIC can over penalize when estimating with small samples (Tabachnick & Fidell, 2008), parameter estimates of the full model are considered reliable.

The main effect of Threat was significant, F (1, 47) = 31.13, $p < .01$, suggesting that, when collapsed across Group, participants committed more errors of commission on contamination no-go trials than neutral no-go trials. The main effect of Group was not significant F (1, 47) = 1.82, $p = .18$, suggesting that, when collapsed across Threat, HCP participants did not commit more errors of commission on no-go trials than LCP participants. Finally, the Threat by Group interaction effect was significant F (1, 47) = 4.21, $p < .05$, suggesting that the effects of Threat were greater for one group relative to the other. The effects of Threat are clearly greater among HCP compared to LCP participants (Figure 4), a series of probing analyses are nonetheless outlined below.

SPSS treats the group with the highest value as the reference group. Therefore, for the first set of contrast analyses, Threat was coded so that neutral trials were the reference group (1 = Neutral and 0 = Contamination) and Group was coded so that LCP was the reference group (1 =

LCP and 0 = HCP). This ensured that all estimates of the fixed effect were compared to the proper reference groups (Neutral, LCP, and Neutral by LCP). The estimate of the fixed effect of the intercept suggested that, on neutral no-go trials, the proportion of errors of commission committed by LCP participants was 10.30% ($\beta_{intercept}$ = 10.30, *SE* = 1.61). The estimate of the fixed effect of Threat suggested that LCP participants committed 4.50% more errors of commission on contamination no-go trials (β_{threat} = 4.50, *SE* = 1.53, $p < .01$, d = 0.86). The estimate of the fixed effect of Group suggested that LCP participants committed only 0.74% more errors of commission than HCP participants on neutral no-go trials (β_{group} = 0.74, *SE* = 2.68, p = .78, d = 0.08). Finally, the estimate of the fixed effect of the Threat by Group interaction term suggested that, compared to the percentage of errors of commission committed by LCP participants on neutral no-go trials (10.30%), an average of 5.24% more errors of commission were committed across the other three experimental cells (LCP/Contamination, HCP/Neutral, HCP/Contamination) ($\beta_{threat*group}$ = 5.24, *SE* = 2.55, $p < .05$, d = 0.60).

To target the magnitude of the effect of the cell of interest (HCP/Contamination), Threat was recoded so that contamination trials were the reference group (0 = Neutral and 1 = Contamination) and Group was recoded so that HCP was the reference group (0 = LCP and 1 = HCP) and one additional MLM model was carried out. The estimate of the fixed effect of the intercept suggested that the proportion of errors of commission committed by HCP participants on contamination no-go trials was 20.78% ($\beta_{intercept}$ = 20.78, *SE* = 2.32). The estimate of the fixed effect of Threat suggested that HCP participants committed 9.74% more errors of commission on contamination no-go trials (β_{threat} = 9.73, *SE* = 2.04, $p < .01$, d = 1.39). The estimate of the fixed effect of Group suggested that HCP participants committed 5.98% more errors of commission than LCP participants on contamination no-go trials (β_{group} = 5.98, *SE* = 2.91, $p < .05$, d = 0.60).

Finally, the estimate of the fixed effect of the Threat by Group interaction term suggested that, compared to the percentage of errors of commission committed by HCP participants on contamination no-go trials (20.78%), an average of 5.24% fewer errors of commission were committed across the other three experimental cells (LCP/Contamination, HCP/Neutral, HCP/Contamination) ($\beta_{threat*group}$ = 5.24, *SE* = 2.55, $p < .05$, d = 0.60). Descriptive statistics are summarized in Table 2.

D. Secondary Analyses

The second series of MLM analyses were conducted to test the effects of Threat and Group on errors of omission. Model building and estimation procedures were identical to the previously described series of MLMs. AIC and BIC of the baseline model were 700.01 and 707.64, respectively. The ICC of the baseline model was .66, suggesting adequate variance within errors of omission. The intercept of the baseline model suggested that, when collapsed across Threat and Group, participants committed, on average, 24.32% errors of omission (β = 24.32, *SE* = 1.48). AIC of the level 1, or within subjects (Threat), model was 693.70 while BIC was 706.42, suggesting model improvement relative to the baseline model. AIC of the level 2, or between subjects (Group), model was 695.39 and BIC was 710.65, suggesting that the addition of the Group factor did not improve model fit. Finally, AIC for the full model was 697.23 and BIC was 715.03, suggesting that the addition of the Threat by Group interaction term did not improve model fit. Inspection of cell means clearly show no effects of Group or interaction between Threat and Group on errors of omission (Table 3). As a consequence of these analyses, only the effects from the level 1, or within subjects (Threat), model are reported. The fixed effect estimate of the intercept of the Threat model suggested that, collapsed across the level of Group, participants committed an average of 22.36% errors of omission on neutral go trials (β = 22.36,

SE = 1.52). The fixed effect estimate of Threat suggested that the rate of errors of omission increased 3.92% on contamination go trials (β = 3.92, *SE* = 1.22, $p < .01$, d = 0.93).

The third series of MLM analyses were conducted to test the effects of Threat and Group on NGRT. Because some participants had 0% errors of commission, they had no NGRT to include in analyses. Model building and estimation procedures were identical to the previously described series of MLMs. AIC and BIC of the baseline model were 851.82 and 859.35, respectively. The ICC of the baseline model was .29, suggesting adequate variance within NGRT. The intercept of the baseline model suggested that, when collapsed across Threat and Group, NGRT was 298.14 ms (β = 298.14, *SE* = 3.06). AIC of the level 1, or within subjects (Threat), model was 846.88 while BIC was 859.44, suggesting model improvement relative to the baseline model. AIC of the level 2, or between subjects (Group), model was 848.76 and BIC was 869.82, suggesting that the addition of the Group factor did not improve model fit. Finally, AIC for the full model was 848.35 and BIC was 865.92, suggesting that the addition of the Threat by Group interaction term did not improve model fit relative to the level 1 model. Inspection of cell means clearly show no effects of Group or interaction between Threat and Group on NGRT (Table 4). As a result of these findings, only the effects of the level 1, within subjects (Threat) model are reported. The fixed effect estimate of the intercept of the Threat model suggested that, collapsed across the level of Group, the average NGRT on neutral no-go trails was 291.40 ms (β = 291.40, *SE* = 3.63). The fixed effect estimate of Threat suggested that the NGRT increased 12.78 ms on contamination no-go trials (β = 12.78, *SE* = 4.08, $p < .01$, d = 0.91).

The final series of MLM analyses were conducted to test the effects of Threat and Group on GRT. Model building and estimation procedures were similar to the previously described

series of MLMs. AIC and BIC of the baseline model were 745.17 and 752.80, respectively. The ICC of the baseline model was .64, suggesting adequate variance within GRT. The intercept of the baseline model suggested that, when collapsed across Threat and Group, GRT was 339.91 ms (β = 339.91, *SE* = 1.87). AIC of the level 1, or within subjects (Threat), model was 747.69 and BIC was 760.41, suggesting that the addition of the Threat factor did not improve model fit relative to the baseline model. Given this, the level 2, or between subjects (Group), model was estimated without the Threat factor. The addition of the fixed effect of Group failed to improve model fit relative to the baseline model (AIC = 746.74, BIC = 756.92). To ensure no effects were missed, a full model was tested and compared to the baseline model. The full model also failed to outperform the baseline model (AIC = 750.46, BIC = 768.26). Inspection of cell means clearly show no main effects or interaction effects (Table 5). None of the estimates of fixed effects are, therefore, reported or interpreted.

E. Exploratory Analyses

Correlations between each of the dependent variables (errors of commission, errors or omission, NGRT, and GRT), each of the DOCS subscales, DOCS-Total, and GAD-7 total were inspected within the entire sample (Table 6) and within each of the quasi-experimental groups (Table 7). Correlations among the entire sample showed that neutral and contamination errors of commission were highly correlated (r = .57, $p \leq .01$). DOCS-Contamination was only marginally correlated with the proportion of errors of commission (r = .25, $p \leq .10$). None of the other DOCS subscales and severity of anxiety (GAD-7 Total) correlated with neutral or contamination errors of commission. The pattern of correlations was considerably different and more informative when separately inspected within each phobic group.

Severity of obsessions, compulsions, and anxiety were largely unrelated to any go/no-go variables among LCP participants. This was likely due to restricted variance in DOCS and GAD-7 scores among LCP participants, which was possibly a consequence of sampling. For example, DOCS-Total and DOCS-C variance were 15.50 and 1.02 among LCP participants and 75.62 and 5.40 among HCP participants. Correlations between go/no-go variables and DOCS scores should be, therefore, interpreted minimally and cautiously among LCP participants. Among HCP participants DOCS-C scores were not related to neutral or contamination errors of commission but were marginally related to neutral errors of omission (r = .44, p = .07), suggesting that the probability to fail to respond on go-trials increased as severity of contamination-washing symptoms increased. DOCS-Total was positively correlated with neutral (r = .49, $p \leq .05$) and contamination (r = .59, $p \leq .01$) NGRT. This suggests that, as severity of obsessions and compulsions increased, HCP participants reacted more quickly on no-go trials. Similarly, GRT increased as severity of obsessions and compulsions increased. This effect was relatively constant for both neutral GRT (r = .41, $p \leq .10$) and contamination GRT (r = .52, $p \leq .05$). Finally, there were weak, negative correlations between DOCS-Total and neutral (r = -.37, p = .14) and contamination errors of commission (r = -.30, p = .24) among HCP participants. This suggests that, among HCP participants, action restraint marginally improved (fewer errors of commission) as severity of OC symptoms increased.

Among HCP participants, there were strong negative relations between errors of commission and NGRT. As NGRT decreased, the proportion of errors of commission increased (all rs > -.46, Table 7). More specifically, as contamination NGRT decreased both neutral (r = -.53, $p \leq .05$) and contamination (r = -.54, $p \leq .05$) errors of commission increased. Among HCP participants, there were also strong negative relations between errors of commission and

contamination GRT. These relations were not present between neutral GRT and errors of commission. As contamination GRT decreased, the proportion of errors of commission increased (all *r*s > -.46, Table 7). More specifically, as contamination GRT decreased both neutral (r = -.71, $p \leq .01$) and contamination (r = -.67, $p \leq .01$) errors of commission increased.

Threat, Group, the Threat by Group interaction term, errors of omission, GRT, and NGRT were all simultaneously regressed onto errors of omission (see Table 8 for summary). The overall model predicted 41.3% variance in errors of commission [F (5, 88) = 12.40, $p < .01$, R^2 = 41.30%]. All predictors were significant except NGRT (β = -.09, t = -0.821, p = .41, R^2 = 0.50%) and the Threat by Group interaction term (β = .21, t = 1.27, p = .21, R^2 = 1.17%). GRT was the strongest predictor of errors of commission (β = -.50, t = -4.98, $p < .01$, R^2 = 17.72%), suggesting that errors of commission increased as GRT decreased. Errors of omission was also a robust predictor of errors of commission (β = .29, t = 3.13, $p < .01$, R^2 = 7.02%), suggesting that errors of commission increased as errors of omission increased. Next, Group, neutral errors of omission, and neutral GRT were regressed onto contamination errors of commission. Consistent with previous analyses, Group was not a significant predictor but neutral errors of omission and GRT both remained significant predictors of neutral errors of commission, with neutral GRT explaining 19.54% variance in neutral errors of commission (see Table 9). Finally, Group, contamination errors of omission, and contamination GRT were regressed onto contamination errors of commission. Also consistent with previous results, Group, contamination errors of omission, and contamination GRT were all significant predictors of contamination errors of commission. Contamination GRT explained 33.30% variance in contamination errors of commission, making it the strongest predictor. Taken as a whole, regression analyses show a strong and clear relation between GRT and errors of commission.

V. DISCUSSION

A. Summary and Conclusions

Basic experimental research has shown that emotional arousal and the processing of emotional information can significantly interfere with response inhibition. To the author's knowledge, the present study was the first to test how the processing of emotional information interferes with response inhibition within a clinically relevant sample. The present study tested how the processing of emotionally arousing and symptom relevant pictorial information – contamination-threat images – interfered with action restraint among an analogue sample of obsessive-compulsive participants with elevated contamination fears. This sample was particularly relevant to the questions at hand given the large body of experimental psychopathology research that suggests obsessions and compulsions are caused and maintained by failures of response inhibition.

In concert with the findings of De Houwer and Tibboel (2010), the present study clearly showed that the presentation of emotionally arousing information prior to no-go-signals resulted in a large attenuation of action restraint. The first null hypothesis stated that there would be no effect of Threat on action restraint (Contamination errors of commission = Neutral errors of commission). The present findings, therefore, allow for the rejection of the first null hypothesis. The second null hypothesis states that there would be no effect of Group on action restraint (LCP errors of commission = HCP errors of commission). In contrast to a majority of the published literature, the present study failed to detect a statistically significant difference in action restraint between HCP and LCP participants. This was evidenced by no meaningful between group difference in neutral errors of commission and no main effect of group on errors of commission.

The third null hypothesis stated that the effect of Threat on action restraint would not differ between LCP and HCP participants. The proportion of errors of commission almost doubled for HCP participants but only increased by approximately 50% for LCP participants. These findings, therefore, allow for the rejection of the third null hypothesis.

Secondary analyses were only partially in accord with previous research (De Houwer & Tibboel, 2010). There was an effect of Threat on NGRT and errors of omission but not GRT. Despite this, exploratory analyses repeatedly showed that GRT was the most robust predictor of errors of commission. The final regression analysis provided the strongest exemplar of this effect, with contamination GRT predicting over 30% variance in contamination errors of commission. These results strongly support the attentional account of the interference effect of Threat on action restraint but also suggest that attention plays a role in basic action restraint (i.e., neutral errors of commission). As GRT increased, proportion of errors of commission markedly decreased. Whether by strategy or individual differences in abilities, longer latencies on go trials were highly related to lower rates of errors of commission on no-go trials.

B. Implications

Inhibition does not occur in a vacuum (Logan & Cowan, 1984; Pessoa et al., 2012; Verbruggen & De Houwer, 2007). This is an incredibly important point when considering clinical implications of the present study. In the case of OCD, inhibition is needed and often fails during moments that are fraught with emotional information. Obsessions and compulsions occur in phobic contexts. Interoceptive and exteroceptive emotional factors arise in such contexts and, as such, can negatively affect inhibitory performance. Intrusive thoughts beget more intrusive thoughts. The emotional salience motivates further intrusions but also hampers attempts to suppress. Intrusive thoughts may even enter into consciousness more easily or occupy more

attention as a consequence of emotional factors, which attenuate inhibitory functions and may thus increase the probability that intrusions can intrude. Compulsions, much the same, are motivated by emotionally arousing information in the environment and resultant interoceptive emotional factors. Motivations for compulsion increase the potency of the prepotent response but may also interfere with the ability to restrain from engaging in habitual acts or stop them prematurely. These ideas can be best described with a case example and simple metaphor.

Lisa (not the real name of a patient) evinces what many would consider classic contamination obsessions and washing compulsions. She has strong aversions toward dirt, germs, and illness. Whenever she comes into contact with real or perceived contaminants, she plays back mental imagery of the contaminated stimuli and scenery, much like a movie reel. This leads to catastrophic thoughts about the implications of her contact with said contaminant. These obsessions, in concert with affective arousal, then motivate the initiation of washing compulsions. Lisa's washing compulsions are not simply brief one time acts, like a key press; they recur both within and between individual acts. Lisa's compulsions are provoked with relative ease, occur frequently, are more powerful than "normal" but comparable behaviors, and are protracted once initiated. Lisa's initiates washing behaviors dozens of times per day and she vigorously washes, sometimes for hours at a time.

From a purely motivational perspective, Lisa's obsessions and compulsions are caused and maintained by environmental and individual "go" factors. Contaminants in the environment motivate thoughts and emotions, which in turn motivate multiple response systems. This is, essentially, a basic summary of widely accepted cognitive-behavioral perspectives on obsessions and compulsions (Rachman, 1997; Salkovskis, 1985) and, more specifically, contamination obsessions and washing compulsions (Rachman, 2006). This perspective is accurate but

incomplete. It is akin to claiming that the acceleration system is the only system of importance on an automobile.

Automobiles do not just go, they *stop* and go. Even the most powerful automobile can be restrained and stopped. Functional emergency brakes can keep high power automobiles from moving and, with well-maintained brakes, an automobile can be stopped on a dime. The motivationalist perspective is focused on mechanisms that motivate going (e.g., emotions, beliefs, environmental factors, etc.). Much like the acceleration system of an automobile, there are dozens of psychological mechanisms working in concert to make obsessions and compulsions go. There are a host of additional psychological mechanisms that govern thoughts and behaviors. Inhibitory mechanisms (resistance to interference, action restraint, action cancellation, etc.) are the braking system of the human machine and, in the case of OCD, they may be faulty. To take this metaphor toward its limit, Lisa might be analogous to a Mustang with bicycle brakes, brakes that are hampered by the very same factors that improve acceleration. Said otherwise, Lisa's faulty or inadequate inhibitory capacities are even less effective at restraining or stopping obsessions and compulsions when greased by emotional factors.

C. Limitations and Future Directions

The present study highlights the importance of inhibition within the emotional context. The present study was, however, cross sectional. Some inhibitory models of OCD focus not just on the importance of inhibition in the maintenance of obsession and compulsions but also on the role of inhibition in the pathogenesis of obsessions and compulsions (Rosenberg & Keshavan, 1998). Compulsive tendencies are relatively common among children and inhibition is thought to be crucial in the extinction of maladaptive, repetitive habits. Given that inhibitory functions are highly heritable in the general population (Friedman et al., 2008) and within OCD probands

(Lennertz et al., 2012; Menzies et al., 2007), it is plausible that inadequate inhibitory functions play a role in the development of compulsions, and perhaps – although less tenable – obsessions. As outlined by Roseberg and Keshavan (1998), repetitive behaviors may persist for longer periods of time due to delayed or incomplete development of inhibitory functions among children at risk of developing OCD. The present study cannot speak to this developmental psychopathological model. Perhaps children at risk of developing OCD are also anxious-neurotic or evidence attentional biases toward threatening information. As seen in the present study and previous research, these factors could further impair inhibitory functioning, thus potentiating risk of repetitive habits developing into clinically significant compulsions. This idea could be tested by administering a similar protocol as the present one – or better yet, a more generalist paradigm such as the white-noise based emotional stop-signal employed by Pessoa and colleagues (2012) – to children at risk for developing OCD (e.g., children with chronic tics or children whose parents are diagnosed with OCD). Simple between-group differences in emotional interference of response inhibition would suggest a more precise vulnerability than basic inhibitory deficits.

The interference effect of contamination images and, for that matter, neutral images, may have tapped into another neurocognitive function other than attention and response inhibition; namely, resistance to distractor interference. This is not to say that attention was not implicated, but rather attention was mediated by other executive function(s). This interpretation of the data is in keeping with the operational definition of the construct of resistance to distractor interference (Friedman & Miyake, 2004) and previous research showing that response inhibition is related to resistance to interference. Previous research has already shown that symptom-specific distractor stimuli interfere with basic interference tasks – such as the emotional Stroop – among participants diagnosed with OCD and analogue OC samples (Moritz et al., 2008; Rao, Arasappa,

Reddy, Venkatasubramanian, & Reddy, 2010; Tobon, Ouimet, & Dozois, 2011; Unoki, Kasuga, Matsushima, Ohta, & Doi, 2000; Wyble, Sharma, & Bowman, 2008). If the present findings are tapping resistance to distractor interference, then the interpretation of the data would be quite different. The lateral orbitofrontal loop is thought to be integral to the pathogenesis and maintenance of OCD (Chamberlain et al., 2005; Graybiel & Rauch, 2000). Within this loop, the anterior cingulate cortex (ACC) and orbital frontal cortex (OFC) are both broadly implicated in the processing of valenced information and inhibitory functions (Bush, Luu, & Posner, 2000), particularly in OCD. The ACC, however, is likely the primary mediator of resistance to distractor interference (Bush et al., 1998; Bush, Luu, & Posner, 2000; Whalen et al., 1998) whereas the orbital frontal cortex (OFC) is largely responsible for prepotent response inhibition (Eagle et al., 2008; Eagle, Bari, & Robbins, 2008; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Rubia et al., 2005). Although there are no data to directly speak to this, the emotional go/no-go task is likely mediated by multiple neural mechanisms. More specifically, it is possible that response inhibition is mediated by OFC activity and resistant to emotional interference is mediated by ACC activity.

The absence of the Group effect was unexpected and is somewhat odd given the strength of the Threat by Group interaction effect. The simplest explanation for this finding is that an analogue OCD sample was used instead of a diagnosed sample. HCP participants all scored above the clinical cut-off score identified by Abramowitz and colleagues (2010) and, as a group, HCP participants actually scored higher than the clinical sample reported by Abramowitz and colleagues. At first blush, this would imply that a majority of participants (approximately 75% given specificity of the DOCS) would likely meet DSM diagnostic criteria for OCD. However, Abramowitz and colleagues did not publish false positive rates when using a DOCS-Total cut-off

score of 18. Given the author's experience with previous research using similar cut-off scores and high rates of false-positive with other self-report assessment tools (Flament et al., 1988) it is possible, if not likely, that a large portion of HCP participants in the present study would not meet diagnostic criteria for OCD if properly assessed. It would be, therefore, advisable for the present study to be replicated with a sample diagnosed with OCD. A clinical or diagnosed community sample may evidence based deficits in response inhibition – which were not observed in the present data – which would allow for a better test of the interaction of inhibition and emotion. Another possible explanation for the absence of a Group effect may lie in the assessment tool that was used in the present study. The emotional go/no-go used in the present study had a time pressure (responses were required within 400 ms), which may have artificially cut off errors of commission that escaped restraint after 400 ms. This is, however, an unlikely explanation as average GRT and NGRT were well below 400 ms. The go/no-go is also an easier task than other measures of response inhibition (Verbruggen & Logan, 2008a). Perhaps Group effects would have been more evident if a more difficult task were used, such as the emotional stop-signal tasks used by Verbruggen and De Houwer (2007) or Pessoa and colleagues (2012).

The sample size used in the present study is too small to draw decisive conclusions. There was also a lack of a non-OCD anxious control group. It is possible that the present findings are due to general anxiety and not OC symptoms. HCP participants scored much higher on the GAD-7 than LCP participants. However, GAD-7 scores showed almost no relations to errors of commission or omission. This is in keeping with previous response inhibition research that showed no meaningful relation between a measure of trait anxiety and neutral and emotional response inhibition (Derakshan, Ansari, Hansard, Shoker, & Eysenck, 2009). Nonetheless, larger

samples that include anxious controls should be recruited for any future replications and extensions.

Previous literature has suggested that inhibitory deficits in OCD may be due in part to comorbid symptoms of depression (Aycicegi et al., 2003; Moritz et al., 2001). Unfortunately, neither continuous nor categorical assessment tools for depression were used in the present study. It is, therefore, difficult to say what, if any, role depression played in the present findings. There are several other psychiatric disorders that are also thought to be affected by failures of inhibition. Due to the lack of diagnostic assessments and lack of overly restrictive study inclusion criteria (e.g., only allow participants diagnosed with OCD and no other psychiatric condition), the available data cannot comprehensively speak to the degree to which symptoms of other disorders did or did not affect the present findings. The absence of any meaningful correlations between GAD-7 scores and errors of commission does, nonetheless, suggest that the observed findings are not a simple artifact of anxiety. Given the present data, it is therefore unlikely that the primary findings could be explained by any possible anxious comorbidity.

As has been mentioned throughout this manuscript, response inhibition is implicated in both the pathogenesis and maintenance of OCD. Very little research has directly focused on the importance of response inhibition in the treatment of OCD. However, there is a convincing literature that highlights the importance of response prevention during exposure therapy for OCD [known as exposure and response prevention (ERP)] (Abramowitz, 1996; Foa & Goldstein, 1978). In most cases, response prevention involves action restraint. For example, contact is made with a contaminant during exposure exercises and then the patient is implored to resist engaging in any compensatory compulsive behaviors. The present study suggests that failures of response prevention may be exacerbated by emotionally salient stimulus characteristics. Given this

possible link between response prevention and emotional action restraint, future research might focus on how response inhibition and, more specifically, emotional response inhibition, predicts treatment compliance and treatment outcome.

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Footnotes

[1] While DSM-IV-TR and all previous versions of the DSM categorized OCD as an anxiety disorder, OCD will soon be removed from the "Anxiety Disorders" category and placed within a new category labeled "Obsessions Compulsive and Related Disorders" (OCRD; APA, 2013). The OCRD category of mental disorders will include OCD, hoarding disorder, body dysmorphic disorder, trichotillomania, and the new excoriation (skin picking) disorder. Despite efforts to increase transparency during preparation of DSM-V, the final rationale behind the decision to create OCRD still remains unclear and is controversial. There will, therefore, be no further treatment of this issue within the present manuscript.

[2] Inhibition is defined as a mechanism or process, while interference is an effect. Therefore, the term resistance to interference is used to clarify that a process (resistance) and not an affect (interference) is being discussed (MacLeod, 2003; Friedman & Miyake, 2004).

[3] The emotional Stroop task has been administered to samples diagnosed with OCD in a large number of published studies. However, the findings from these studies are usually interpreted as evidence (or lack thereof) of an attentional bias. This interpretation of the emotional Stroop effect is controversial and quite possibly inaccurate (Bush, Luu, & Posner, 2000; Whalen et al., 1998). Therefore, published papers with attentional interpretations of emotional Stroop paradigms will not be reviewed herein.

[4] Primary hypotheses were also tested using mixed factor ANOVA. Effects were similar to those derived from MLM. The only differences being a slightly lesser Threat by Group effect and slightly larger main effects of Threat and Group.

Table 1. Descriptive data for low contamination phobic (LCP) and high contamination phobic (HCP) participants.

	LCP (*M*, *SD*)	HCP (*M*, *SD*)	*F* or X^2
DOCS-Total	6.47 (.72)	30.65 (8.70)	172.06[b]
DOCS-C	1.53 (.18)	9.47 (2.32)	2.65.88[b]
DOCS-R	1.97 (.31)	8.12 (2.87)	85.24[b]
DOCS-O	1.74 (.32)	7.12 (2.62)	71.63[b]
DOCS-S	1.41 (.26)	5.94 (4.41)	29.38[b]
GAD-7	2.77 (1.83)	6.47 (3.62)	21.78[b]
Age	19.07 (.94)	19.65 (2.32)	.23
Gender	67% Female	59% Female	.29
Race	83% Caucasian	82% Caucasian	1.99

Note. Superscript “a” denotes $p \leq .05$ and superscript “b” denotes $p \leq .01$

Table 2. Descriptive statistics [*M* (*SD*)] from main effects and interaction of Threat (neutral and contamination) and Group (LCP and HCP) on errors of commission. All values are percentages of errors of commission relative to total number of commission trials ($n_{errors}/n_{no\text{-}go\ trials}$)

Threat	Group	
	LCP	HCP
Neutral	10.30 (1.62)	11.04 (2.15)
Contamination	14.80 (1.75)	20.78 (2.32)

Table 3. Descriptive statistics [*M* (*SD*)] from main effects and interaction of Threat (neutral and contamination) and Group (LCP and HCP) on errors of omission. All values are percentages of errors of omission relative to total number of omission trials ($n_{errors}/n_{go\ trials}$)

Threat	Group	
	LCP	HCP
Neutral	23.09 (1.89)	21.08 (2.51)
Contamination	26.64 (2.11)	25.66 (2.80)

Table 4. Descriptive statistics [*M* (*SD*)] from main effects and interaction of Threat (neutral and contamination) and Group (LCP and HCP) on no-go reaction time (NGRT). All values are in milliseconds.

Threat	Group	
	LCP	HCP
Neutral	288.14 (4.73)	296.26 (5.83)
Contamination	305.91 (4.73)	301.13 (6.29)

Note. NGRT values were available for all 17 HCP participants for both neutral and contamination no-go trials. Contamination no-go NGRT values were available for all 30 LCP participants but only 27 neutral no-go NGRT values were available for LCP participants due to 3 participants.

Table 5. Descriptive statistics [*M* (*SD*)] from main effects and interaction of Threat (neutral and contamination) and Group (LCP and HCP) on correct reaction time (GRT). All values are in milliseconds.

Threat	Group	
	LCP	HCP
Neutral	338.18 (2.38)	342.10 (3.16)
Contamination	339.18 (2.74)	340.98 (3.63)

Table 6. Pearson's correlation coefficients within entire sample

	DOCS Total	DOCS Cont.	DOCS Resp.	DOCS Obsess	DOCS Symm.	GAD7 Total	Neu. Comm.	Cont. Comm.	Neu. Omm.	Cont. Omm.	Neu. NGRT	Cont. NGRT	Neu. GRT
DOCS Cont.	.94[b]												
DOCS Resp.	.91[b]	.88[b]											
DOCS Obsess	.89[b]	.83[b]	.73[b]										
DOCS Symm.	.80[b]	.62[b]	.63[b]	.62[b]									
GAD7 Total	.69[b]	.64[b]	.71[b]	.58[b]	.49[b]								
Neu. Comm.	-.08	-.00	-.10	.03	-.19	.01							
Cont. Comm.	.16	.25[*]	.11	.23	-.05	.18	.57[b]						
Neu. Omm.	-.00	-.01	.09	-.01	-.08	-.00	.31[a]	.04					
Cont. Omm.	.01	-.03	.11	-.03	-.03	.13	.28[a]	-.04	.71[b]				
Neu. NGRT	.21	.15	.23	.05	.30[a]	.11	-.11	-.25	.32[a]	.42[b]			
Cont. NGRT	.06	-.05	.06	.06	.17	.18	-.16	-.36[b]	.16	.37[b]	.39[b]		
Neu. GRT	.23	.15	.15	.18	.35[a]	.21	-.38[b]	-.36[b]	.15	.12	.26[*]	.39[b]	
Cont. GRT	.20	.07	.23	.08	.33[a]	.15	-.45[b]	-.53[b]	.21	.40[b]	.47[b]	.68[b]	.65[b]

Note. Superscript "a" denotes $p \leq .05$, superscript "b" denotes $p \leq .01$, and "*" denotes $p \leq .10$.

Table 7. Pearson's correlation coefficients split by LCP (upper diagonal) and HCP (lower diagonal)

	DOCS Total	DOCS Cont.	DOCS Resp.	DOCS Obsess	DOCS Symm.	GAD7 Total	Neu. Comm.	Cont. Comm.	Neu. Omm.	Cont. Omm.	Neu. NGRT	Cont. NGRT	Neu. GRT	Cont. GRT
DOCS Total	-	.71[b]	.68[b]	.63[b]	.68[b]	.49[b]	-.17	-.18	-.01	-.11	-.11	.04	.10	.19
DOCS Cont.	.67[b]	-	.35[*]	.40[a]	.35[*]	.22	-.11	-.14	-.03	-.12	-.05	-.02	.06	.14
DOCS Resp.	.65[b]	.67[b]	-	.06	.38[a]	.56[b]	-.10	-.09	.11	.06	-.03	.02	.02	.17
DOCS Obsess	.73[b]	.48[b]	.41	-	.17	.24	.03	.04	-.20	-.21	-.38[*]	.08	-.09	-.05
DOCS Symm.	.70[b]	.07	.23	.33	-	.25	-.30	-.35[*]	.13	-.04	.21	-.01	.33[*]	.28
GAD7 Total	.46[*]	.42[*]	.49[a]	.29	.21	-	.03	.01	.06	.10	-.31	.26	.11	.22
Neu. Comm.	-.37	-.13	-.41	-.06	-.36	-.06	-	.62[b]	.42[a]	.43[a]	.07	.02	-.42[a]	-.32[*]
Cont. Comm.	-.31	.05	.37	-.02	-.37	.04	.51[b]	-	.15	.08	-.01	-.26	-.54[b]	-.50[b]
Neu. Omm.	.39	.44[*]	.53[a]	.53[a]	-.13	.07	.06	-.10	-	.75[b]	.39[a]	.22	.26	.28
Cont. Omm.	.37	.21	.59[a]	.38	.01	.37	-.12	-.31	.61[b]	-	.37[*]	.35[*]	.19	.37
Neu. NGRT	.49[a]	.19	.49[a]	.26	.40	.37	-.47[*]	-.46[*]	.23	.61[b]	-	.30	.31	.38[a]
Cont. NGRT	.59[a]	.16	.47[*]	.38	.54[a]	.34	-.53[a]	-.54[a]	.02	.42[*]	.64[b]	-	.42[a]	.66[b]
Neu. GRT	.41[*]	.06	.10	.41[*]	.46[*]	.23	-.31	-.17	-.06	-.08	.12	.36	-	.74[b]
Cont. GRT	.52$_a$	.06	.52[a]	.22	.52[a]	.12	-.71[b]	-.67[b]	.12	.51[a]	.63[b]	.75[b]	.51[a]	-

Note. Superscript "a" denotes $p \leq .05$, superscript "b" denotes $p \leq .01$, and "*" denotes $p \leq .10$.

Table 8. Threat, Group, Threat by Group, errors of omission, GRT, and NGRT regressed onto errors of commission.

Predictor	Std. β	t	$R^2\Delta$
Threat	-.43	-3.04[b]	6.60
Group	-.32	-2.78[b]	5.15
Threat by Group	.21	1.27	1.16
Omission	.29	3.13	7.02
GRT	-.50	-4.98	17.72
NGRT	-.09	-0.82	0.00

Note. Superscript "b" denotes $p \leq .01$

Table 9. Group, errors of omission, and GRT, and regressed onto neutral errors of commission and contamination errors of commission

DV	Predictor	Std. β	t	$R^2\Delta$
Neutral Errors of Comission				
	Group	-.14	-1.09	01.93
	Omission	.39[b]	2.99	14.59
	GRT	-.45[b]	-3.45	19.54
Contamination Errors of Comission				
	Group	-.32[b]	-2.74	10.18
	Omission	.23[*]	1.78	4.33
	GRT	-.63[a]	-4.95	33.30

Note. Superscript “a” denotes $p \leq .05$, superscript “b” denotes $p \leq .01$, and “*” denotes $p \leq .10$.

Figure 1. Modern nested hierarchical heuristic of executive inhibition. The two higher order constructs of response inhibition and resistance to interference can be further separated based on the temporal properties of resistance or inhibition. Exemplar tasks are displayed in square boxes and reciprocal arrows are included to indicate possible shared variance among tasks.

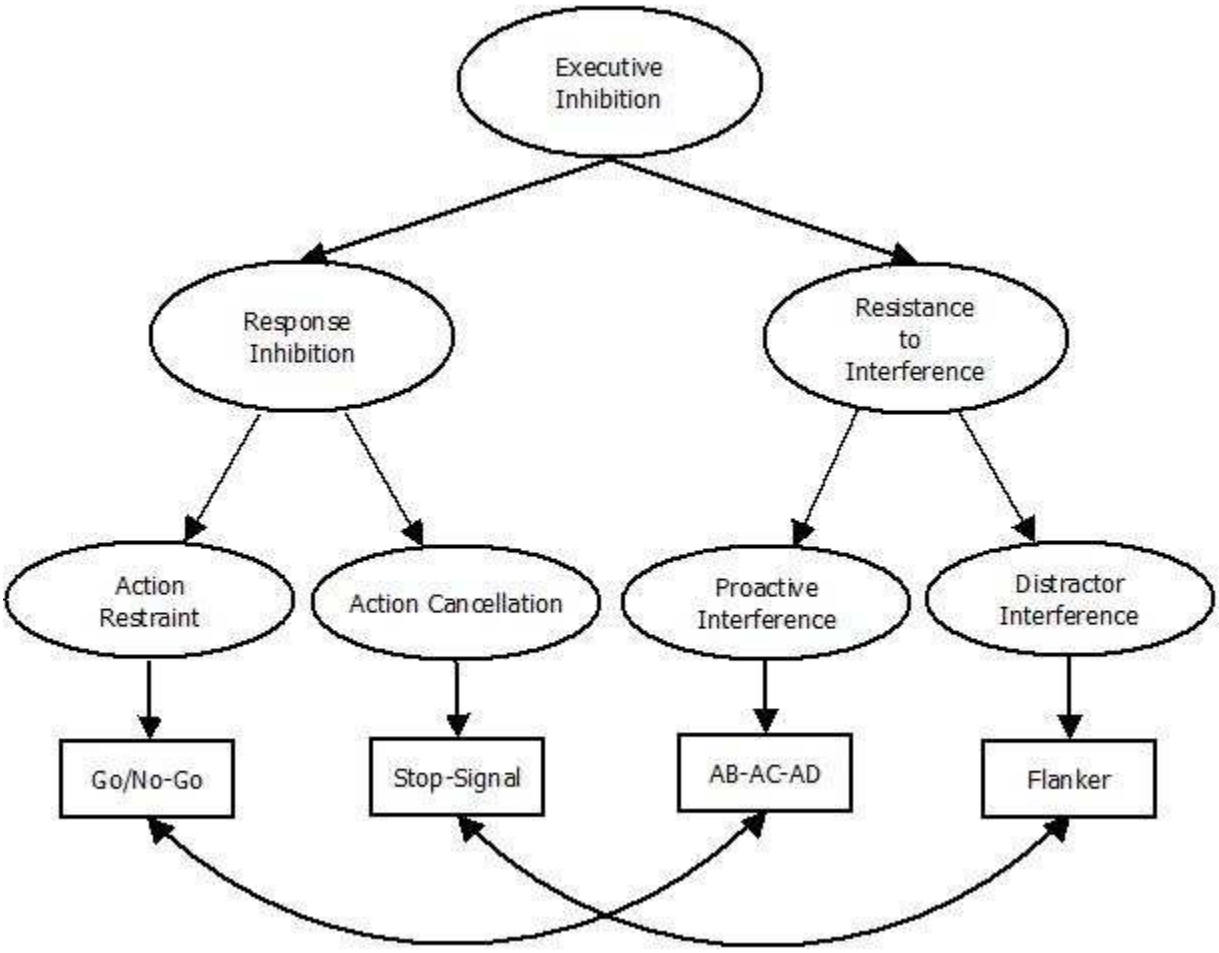
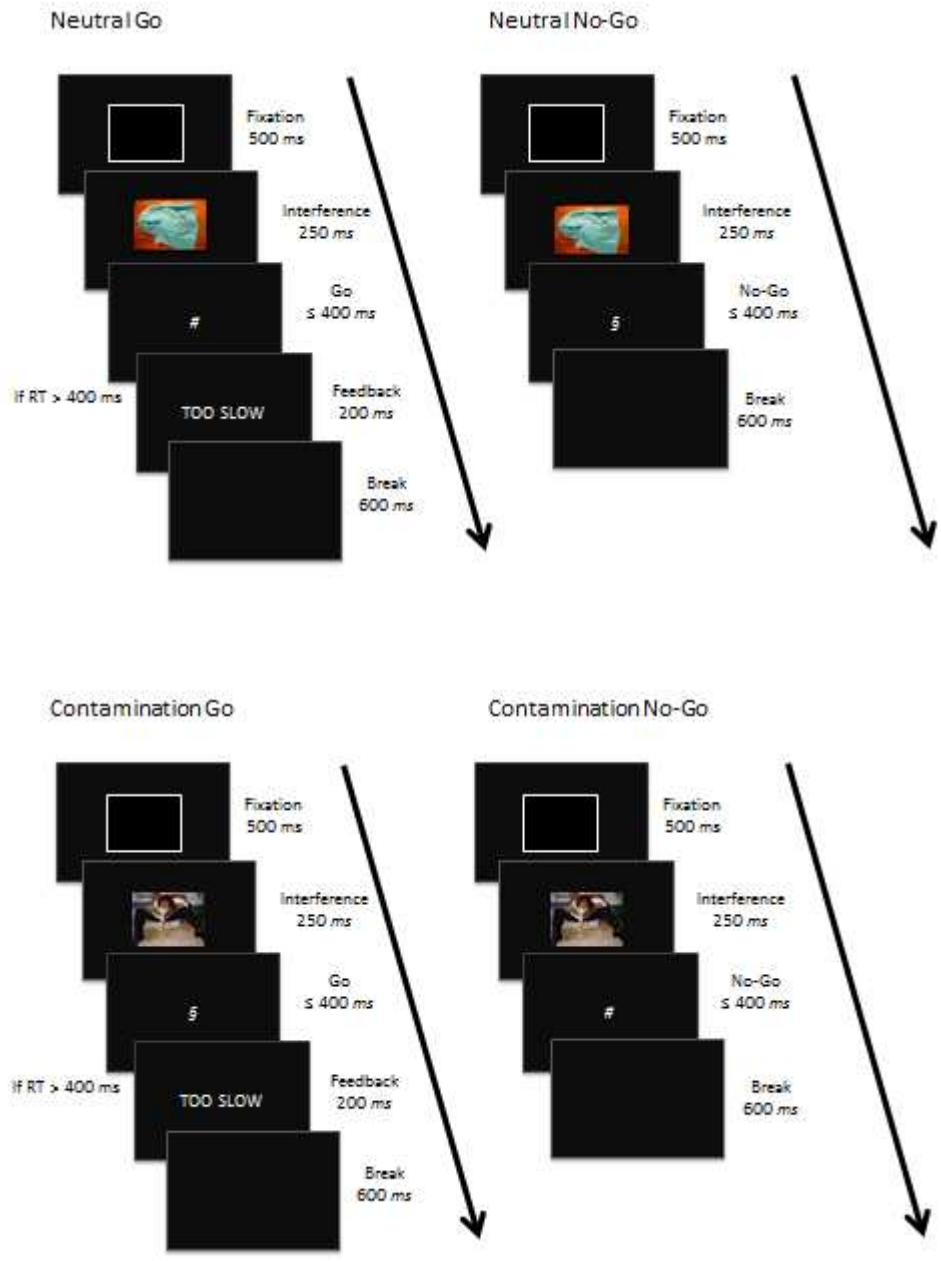
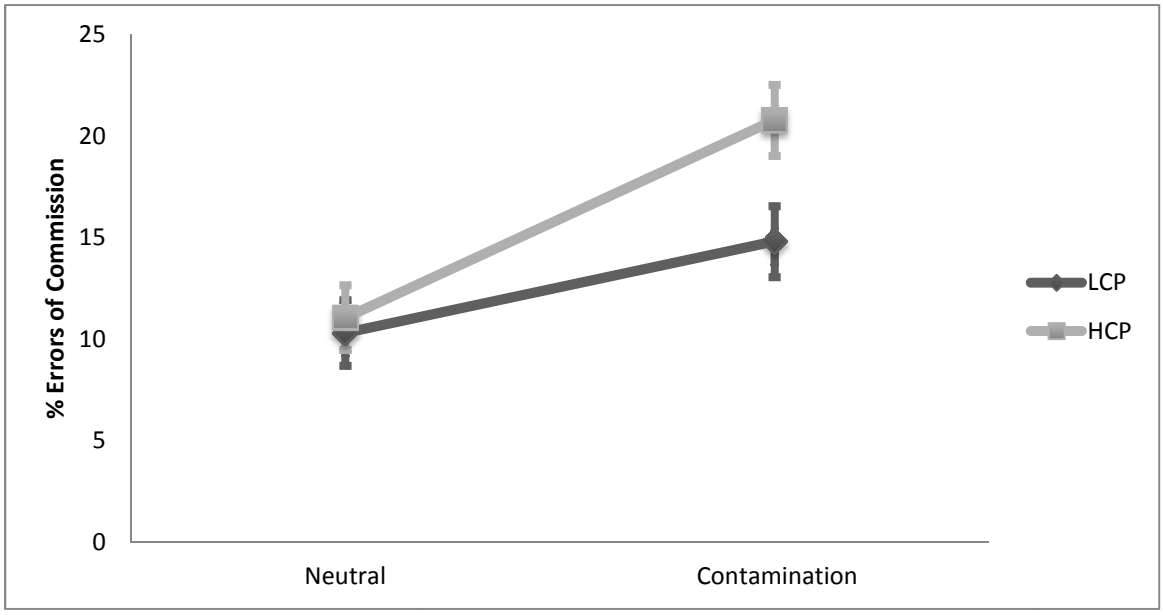


Figure 2. Pictorial display of the emotional go/no-go task used in the present study.



Note. For the sake of illustration, *§* is a stop-signal for the neutral trials and a go-signal for the contamination trials. In the actual experiment, each symbol was used as the go or stop-signal for all conditions.

Figure 3. Main effects and interaction of Threat (neutral and contamination) and Group (LCP and HCP) on errors of commission.



Note. LCP denotes “Low Contamination Phobic” and HCP denotes “High Contamination Phobic”. Errors of commission are presented as whole percentage of trials.

Appendix A

Dimensional Obsessive Compulsive Scale (DOCS)

Instructions: This questionnaire asks you about 4 different categories of concerns that you might or might not experience. For each category there is a description of the kinds of thoughts (sometimes called *obsessions*) and behaviors (sometimes called *compulsions*) that are typical of that particular concern, followed by five questions about your experiences with these thoughts and behaviors. Please read each description carefully and answer the questions for each category based on your experiences in the last month.

Category 1: Concerns about Germs and Contamination

Examples…

-Thoughts or feelings that you are contaminated because you came into contact with (or were nearby) a certain object or person.
-The feeling of being contaminated because you were in a certain place (such as a bathroom).
-Thoughts about germs, sickness, or the possibility of spreading contamination.
-Washing your hands, using hand sanitizer gels, showering, changing your clothes, or cleaning objects because of concerns about contamination.
-Following a certain routine (e.g., in the bathroom, getting dressed) because of contamination
-Avoiding certain people, objects, or places because of contamination.

The next questions ask about your experiences with thoughts and behaviors related to contamination over the last month. Keep in mind that your experiences might be different than the examples listed above. Also, if any of the items concern something that was not part of your experience in the last month, answer on the basis of how you think you might feel *if you had* such an experience. Otherwise, answer all items on the basis of your own experience. Please circle the number next to your answer:

1. About how much time have you spent each day thinking about contamination and engaging in washing or cleaning behaviors because of contamination?

   0 None at all
   1 Less than 1 hour each day
   2 Between 1 and 3 hours each day
   3 Between 3 and 8 hours each day
   4 8 hours or more each day

Continued →

2. To what extent have you avoided situations in order to prevent concerns with contamination or having to spend time washing, cleaning, or showering?

   0 None at all
   1 A little avoidance
   2 A moderate amount of avoidance
   3 A great deal of avoidance
   4 Extreme avoidance of nearly all things

3. If you had thoughts about contamination but could not wash, clean, or shower (or otherwise remove the contamination), how distressed or anxious did you become?

   0 Not at all distressed/anxious
   1 Mildly distressed/anxious
   2 Moderately distressed/anxious
   3 Severely distressed/anxious
   4 Extremely distressed/anxious

4. To what extent has your daily routine (work, school, self-care, social life) been disrupted by contamination concerns and excessive washing, showering, cleaning, or avoidance behaviors?

   0 No disruption at all.
   1 A little disruption, but I mostly function well.
   2 Many things are disrupted, but I can still manage.
   3 My life is disrupted in many ways and I have trouble managing.
   4 My life is completely disrupted and I cannot function at all.

5. How difficult is it for you to disregard thoughts about contamination and refrain from behaviors such as washing, showering, cleaning, and other decontamination routines when you try to do so?

   0 Not at all difficult
   1 A little difficult
   2 Moderately difficult
   3 Very difficult
   4 Extremely difficult

Continued →

Category 2: Concerns about being Responsible for Harm, Injury, or Bad Luck

Examples…

-A doubt that you might have made a mistake that could cause something awful or harmful to happen.
-The thought that a terrible accident, disaster, injury, or other bad luck might have occurred and you weren't careful enough to prevent it.
-The thought that you could prevent harm or bad luck by doing things in a certain way, counting to certain numbers, or by avoiding certain "bad" numbers or words.
-Thought of losing something important that you are unlikely to lose (e.g., wallet, identify theft, papers).
-Checking things such as locks, switches, your wallet, etc. more often than is necessary.
-Repeatedly asking or checking for reassurance that something bad did not (or will not) happen.
-Mentally reviewing past events to make sure you didn't do anything wrong.
-The need to follow a special routine because it will prevent harm or disasters from occurring.
-The need to count to certain numbers, or avoid certain bad numbers, due to the fear of harm.

The next questions ask about your experiences with thoughts and behaviors related to harm and disasters over the last month. Keep in mind that your experiences might be slightly different than the examples listed above. Please circle the number next to your answer:

1. About how much time have you spent each day thinking about the possibility of harm or disasters and engaging in checking or efforts to get reassurance that such things do not (or did not) occur?

   0 None at all
   1 Less than 1 hour each day
   2 Between 1 and 3 hours each day
   3 Between 3 and 8 hours each day
   4 8 hours or more each day

2. To what extent have you avoided situations so that you did not have to check for danger or worry about possible harm or disasters?

   0 None at all
   1 A little avoidance
   2 A moderate amount of avoidance
   3 A great deal of avoidance
   4 Extreme avoidance of nearly all things

Continued →

3. When you think about the possibility of harm or disasters, or if you cannot check or get reassurance about these things, how distressed or anxious did you become?

   0 Not at all distressed/anxious
   1 Mildly distressed/anxious
   2 Moderately distressed/anxious
   3 Severely distressed/anxious
   4 Extremely distressed/anxious

4. To what extent has your daily routine (work, school, self-care, social life) been disrupted by thoughts about harm or disasters and excessive checking or asking for reassurance?

   0 No disruption at all.
   1 A little disruption, but I mostly function well.
   2 Many things are disrupted, but I can still manage.
   3 My life is disrupted in many ways and I have trouble managing.
   4 My life is completely disrupted and I cannot function at all.

5. How difficult is it for you to disregard thoughts about possible harm or disasters and refrain from checking or reassurance-seeking behaviors when you try to do so?

   0 Not at all difficult
   1 A little difficult
   2 Moderately difficult
   3 Very difficult
   4 Extremely difficult

Category 3: Unacceptable Thoughts

Examples…

-Unpleasant thoughts about sex, immorality, or violence that come to mind against your will.
-Thoughts about doing awful, improper, or embarrassing things that you don't really want to do.
-Repeating an action or following a special routine because of a bad thought.
-Mentally performing an action or saying prayers to get rid of an unwanted or unpleasant thought.
-Avoidance of certain people, places, situations or other triggers of unwanted or unpleasant thoughts

Continued →

The next questions ask about your experiences with unwanted thoughts that come to mind against your will and behaviors designed to deal with these kinds of thoughts over the last month. Keep in mind that your experiences might be slightly different than the examples listed above. Please circle the number next to your answer:

1. About how much time have you spent each day with unwanted unpleasant thoughts and with behavioral or mental actions to deal with them?

   0 None at all
   1 Less than 1 hour each day
   2 Between 1 and 3 hours each day
   3 Between 3 and 8 hours each day
   4 8 hours or more each day

2. To what extent have you been avoiding situations, places, objects and other reminders (e.g., numbers, people) that trigger unwanted or unpleasant thoughts?

   0 None at all
   1 A little avoidance
   2 A moderate amount of avoidance
   3 A great deal of avoidance
   4 Extreme avoidance of nearly all things

3. When unwanted or unpleasant thoughts come to mind against your will how distressed or anxious did you become?

   0 Not at all distressed/anxious
   1 Mildly distressed/anxious
   2 Moderately distressed/anxious
   3 Severely distressed/anxious
   4 Extremely distressed/anxious

4. To what extent has your daily routine (work, school, self-care, social life) been disrupted by unwanted and unpleasant thoughts and efforts to avoid or deal with such thoughts?

   0 No disruption at all.
   1 A little disruption, but I mostly function well.
   2 Many things are disrupted, but I can still manage.
   3 My life is disrupted in many ways and I have trouble managing.
   4 My life is completely disrupted and I cannot function at all.

5. How difficult is it for you to disregard unwanted or unpleasant thoughts and refrain from using behavioral or mental acts to deal with them when you try to do so?

   0 Not at all difficult
   1 A little difficult
   2 Moderately difficult
   3 Very difficult
   4 Extremely difficult

Category 4: Concerns about Symmetry, Completeness, and the Need for Things to be "Just Right"

Examples…

-The need for symmetry, evenness, balance, or exactness.
-Feelings that something isn't "just right."
-Repeating a routine action until it feels "just right" or "balanced."
-Counting senseless things (e.g., ceiling tiles, words in a sentence).
-Unnecessarily arranging things in "order."
-Having to say something over and over in the same way until it feels "just right."

The next questions ask about your experiences with feelings that something is not "just right" and behaviors designed to achieve order, symmetry, or balance over the last month. Keep in mind that your experiences might be slightly different than the examples listed above. Please circle the number next to your answer:

1. About how much time have you spent each day with unwanted thoughts about symmetry, order, or balance and with behaviors intended to achieve symmetry, order or balance?

   0 None at all
   1 Less than 1 hour each day
   2 Between 1 and 3 hours each day
   3 Between 3 and 8 hours each day
   4 8 hours or more each day

2. To what extent have you been avoiding situations, places or objects associated with feelings that something is not symmetrical or "just right?"

   0 None at all
   1 A little avoidance
   2 A moderate amount of avoidance
   3 A great deal of avoidance
   4 Extreme avoidance of nearly all things

3. When you have the feeling of something being "not just right," how distressed or anxious did you become?

   0 Not at all distressed/anxious
   1 Mildly distressed/anxious
   2 Moderately distressed/anxious
   3 Severely distressed/anxious
   4 Extremely distressed/anxious

Continued →

4. To what extent has your daily routine (work, school, self-care, social life) been disrupted by the feeling of things being "not just right," and efforts to put things in order or make them feel right?

   0 No disruption at all.
   1 A little disruption, but I mostly function well.
   2 Many things are disrupted, but I can still manage.
   3 My life is disrupted in many ways and I have trouble managing.
   4 My life is completely disrupted and I cannot function at all.

5. How difficult is it for you to disregard thoughts about the lack of symmetry and order, and refrain from urges to arrange things in order or repeat certain behaviors when you try to do so?

   0 Not at all difficult
   1 A little difficult
   2 Moderately difficult
   3 Very difficult
   4 Extremely difficult

Appendix B

Generalized Anxiety Disorder-7 (GAD-7)

	INSTRUCTIONS: Over the last 2 weeks, how often have you been bothered by the following problems? Please use the following scale: 0 = not at all 1 = several days 2 = more than ½ the days 3 = nearly every day				
1	Feeling nervous, anxious, or on edge	0	1	2	3
2	Not being able to stop or control worrying	0	1	2	3
3	Worrying too much about different things	0	1	2	3
4	Having trouble relaxing	0	1	2	3
5	Being so restless that it is hard to sit still	0	1	2	3
6	Becoming easily annoyed or irritable	0	1	2	3
7	Feeling afraid as if something awful might happen	0	1	2	3

Appendix C

****

Errors of Commission

****

Baseline (null) MLM on commission

```
mixed commission
/fixed intercept | SSTYPE (3)
/random intercept  | subject(id)
/print solution testcov
/EMMEANS  TABLES  (OVERALL)
/method ML.
```

Threat as fixed and repeated effects

```
mixed commission by threat
/fixed intercept threat | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat | subject (id)
/print solution testcov
/method ML.
```

Threat and Group as fixed effects and Threat as repeated effect

```
mixed commission by threat group
/fixed intercept threat group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/method ML.
```

Threat, Group, and their interaction term as fixed effects with Threat as repeated effect

```
mixed commission by threat group
/fixed intercept threat group threat*group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/EMMEANS TABLES (threat)
/EMMEANS   TABLES   (group)
/EMMEANS TABLES   (threat*group)
/method ML.
```

the below syntax results in an estimate in the interaction effect that reflects the overall difference between HCP/Contamination and all other means

```
**************************************************************************
mixed commission by threat_1 group_1
/fixed intercept threat_1 group_1 threat_1*group_1 | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat_1  | subject(id)
/EMMEANS TABLES (threat_1)
/EMMEANS   TABLES   (group_1)
/EMMEANS TABLES   (threat_1*group_1)
/print solution testcov
/method ML.

**************************************************************************
```

Errors of Omission

```
**************************************************************************

***Baseline (null) model MLM on omission***

mixed omission
/fixed intercept | SSTYPE (3)
/random intercept  | subject(id)
/print solution testcov
/EMMEANS  TABLES  (OVERALL)
/method ML.

***Threat as fixed and repeated effect***

mixed omission by threat
/fixed intercept threat | SSTYPE (3)
/random intercept | subject(id)
/repeated threat | subject (id)
/print solution testcov
/method ML.

***Threat and Group as fixed effects and Threat as repeated effect***

mixed omission by threat group
/fixed intercept threat group | SSTYPE (3)
```

```
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/method ML.
```

Threat, Group, and their interaction term as fixed effects with Threat as repeated effect

```
mixed omission by threat group
/fixed intercept threat group threat*group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/EMMEANS TABLES (threat)
/EMMEANS   TABLES   (group)
/EMMEANS TABLES   (threat*group)
/method ML.
```

****

NGRT

****

Baseline (null) model MLM on NGRT

```
mixed NGRT
/fixed intercept | SSTYPE (3)
/random intercept  | subject(id)
/print solution testcov
/EMMEANS  TABLES  (OVERALL)
/method ML.
```

Threat as fixed and repeated effect

```
mixed NGRT by threat
/fixed intercept threat | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat | subject (id)
/print solution testcov
/method ML.
```

Threat and Group as fixed effects and Threat as repeated effect

```
mixed NGRT by threat group
/fixed intercept threat group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
```

```
/print solution testcov
/method ML.

***Threat, Group, and their interaction term as fixed effects with Threat as repeated effect***

mixed NGRT by threat group
/fixed intercept threat group threat*group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/EMMEANS TABLES (threat)
/EMMEANS   TABLES   (group)
/EMMEANS TABLES   (threat*group)
/method ML.

*****************************************************************************
                                        GRT
*****************************************************************************

***Baseline (null) model MLM on GRT***

mixed GRT
/fixed intercept | SSTYPE (3)
/random intercept  | subject(id)
/print solution testcov
/EMMEANS  TABLES  (OVERALL)
/method ML.

***Threat as fixed and repeated effect***

mixed GRT by threat
/fixed intercept threat | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat | subject (id)
/print solution testcov
/method ML.


***Group as fixed effects ***

mixed GRT by group
/fixed intercept group | SSTYPE (3)
/random intercept  | subject(id)
/print solution testcov
/method ML.
```

Threat and Group as fixed effects and Threat as repeated effect

```
mixed GRT by threat group
/fixed intercept threat group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/method ML.
```

Threat, Group, and their interaction term as fixed effects with Threat as repeated effect

```
mixed GRT by threat group
/fixed intercept threat group threat*group | SSTYPE (3)
/random intercept  | subject(id)
/repeated threat  | subject (id)
/print solution testcov
/EMMEANS TABLES (threat)
/EMMEANS   TABLES   (group)
/EMMEANS TABLES   (threat*group)
/method ML.
```

Appendix D

id,threat,group,commission,omission,NGRT,GRT,threat_1,group_1
1,1,1,6.25,16.67,277.75,341.36,0,0
1,0,1,31.25,20.83,315.35,335.53,1,0
2,1,1,8.33,39.58,315,324.66,0,0
2,0,1,17.39,23.4,264.74,319.67,1,0
3,1,1,10.42,25,295.5,349.13,0,0
3,0,1,14.58,41.67,339,348.12,1,0
4,1,1,6.25,31.25,286.5,340.13,0,0
4,0,1,29.17,25,277.98,335.72,1,0
5,1,1,6.25,25,302,348.16,0,0
5,0,1,10.42,16.67,342.25,356.22,1,0
6,1,1,22.92,14.58,282.29,328.6,0,0
6,0,1,37.5,14.58,281.5,313.62,1,0
7,1,1,4.17,14.58,288,355.46,0,0
7,0,1,2.08,6.25,315,347.87,1,0
9,1,1,12.5,14.58,323.13,339.93,0,0
9,0,1,6.25,25,328.5,345.85,1,0
10,1,1,8.33,10.42,297.67,328.27,0,0
10,0,1,8.33,12.5,284,322.35,1,0
11,1,1,25,43.75,295.25,346.79,0,0
11,0,1,15.22,41.67,317,338.51,1,0
12,1,1,18.75,12.5,277.75,307.55,0,0
12,0,1,22.92,27.08,293.82,331.74,1,0
13,1,1,2.08,12.5,309,336.9,0,0
13,0,1,4.17,8.33,314.5,334.08,1,0
14,1,1,2.08,29.17,314,353.71,0,0
14,0,1,8.33,47.92,349.17,360.54,1,0
15,1,1,12.5,10.42,284.75,318.18,0,0
15,0,1,16.67,12.5,303.84,334.74,1,0
16,1,1,0,16.67,,338.55,0,0
16,0,1,16.67,12.5,245.79,330.1,1,0
17,1,1,25.53,21.28,266.49,316.34,0,0
17,0,1,25,36.17,244.92,307.32,1,0
18,1,1,14.58,33.33,302.43,349.2,0,0
18,0,1,14.58,39.58,342.54,356.22,1,0
19,1,1,35.42,41.67,283.22,321.07,0,0
19,0,1,29.79,35.42,314.71,323.73,1,0
20,1,1,8.33,16.67,292.17,330.73,0,0
20,0,1,18.75,20.83,294.32,354.24,1,0
21,1,1,0,25,,358.65,0,0
21,0,1,4.17,33.33,279,350.8,1,0
22,1,1,4.17,18.75,273,350.04,0,0
22,0,1,14.58,18.75,336.75,350.17,1,0
23,1,1,6.25,22.92,307.75,328.75,0,0

23,0,1,12.5,35.42,311.5,345.67,1,0
24,1,1,2.08,18.75,242,360.33,0,0
24,0,1,2.08,16.67,294,354.58,1,0
25,1,1,0,16.67,,339.8,0,0
25,0,1,10.64,14.58,293.75,336.75,1,0
26,1,1,4.17,4.17,274.5,336.72,0,0
26,0,1,4.17,16.67,308,324.44,1,0
27,1,1,14.58,45.83,305.5,345,0,0
27,0,1,12.5,47.92,327.3,348.32,1,0
28,1,1,8.33,35.42,323,359.33,0,0
28,0,1,2.08,43.75,314,361.95,1,0
29,1,1,2.08,12.5,201,315.54,0,0
29,0,1,16.67,16.67,302.24,322.74,1,0
32,1,0,4.17,16.67,308,342.94,0,1
32,0,0,33.33,25,317.09,345.59,1,1
33,1,0,10.42,12.5,285.5,329.06,0,1
33,0,0,35.42,14.58,274.95,311.24,1,1
34,1,0,16.67,2.08,291.8,339.55,0,1
34,0,0,25.53,20.83,306.5,345.68,1,1
35,1,0,29.17,16.67,272.9,329.93,0,1
35,0,0,27.08,12.5,266.59,300.3,1,1
30,1,1,31.25,34.04,298.22,334.3,0,0
30,0,1,31.25,45.83,322.36,347.59,1,0
37,1,0,20.83,37.5,305.25,341.26,0,1
37,0,0,29.17,45.83,313.86,341.58,1,1
38,1,0,6.25,14.58,297.5,362.95,0,1
38,0,0,25,22.92,279.19,346.05,1,1
39,1,0,8.33,27.08,295.5,334.38,0,1
39,0,0,29.17,25,308.72,336.97,1,1
40,1,0,10.42,25,324.13,364.55,0,1
40,0,0,12.5,27.08,346.3,362.34,1,1
41,1,0,4.17,33.33,308,339.95,0,1
41,0,0,8.33,36.17,331.5,351.81,1,1
42,1,0,8.51,27.08,297.5,325.35,0,1
42,0,0,12.77,35.42,296,346.95,1,1
43,1,0,2.08,10.42,267,355.05,0,1
43,0,0,10.42,12.5,320.75,348.87,1,1
44,1,0,27.08,22.92,247.04,333.54,0,1
44,0,0,31.25,16.67,252.6,316.13,1,1
31,1,1,6.25,29.17,302.75,342.17,0,0
31,0,1,4.17,41.67,319.5,355.15,1,0
45,1,0,4.17,22.92,279.5,343.09,0,1
45,0,0,16.67,22.92,280.2,351.14,1,1
46,1,0,16.67,31.25,287.27,351,0,1
46,0,0,25,31.25,277.93,337.74,1,1
47,1,0,6.25,25,342.75,326.48,0,1

47,0,0,6.25,35.42,303,351.66,1,1
48,1,0,4.17,27.08,316,353.07,0,1
48,0,0,14.89,20.83,308.5,349.79,1,1
49,1,0,8.33,6.25,310.75,343.57,0,1
49,0,0,10.42,31.25,335.5,352.78,1,

Appendix E

Office of Research Compliance
Institutional Review Board

March 5, 2012

MEMORANDUM

TO:	Thomas Adams William Levine Jeffrey Lohr
FROM:	Ro Windwalker IRB Coordinator
RE:	New Protocol Approval
IRB Protocol #:	12-02-506
Protocol Title:	*Emotion, Inhibition, and Anxiety*
Review Type:	☐ EXEMPT ☒ EXPEDITED ☐ FULL IRB
Approved Project Period:	Start Date: 03/04/2012 Expiration Date: 03/03/2013

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form *Continuing Review for IRB Approved Projects*, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (http://vpred.uark.edu/210.php). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 200 participants. If you wish to make *any* modifications in the approved protocol, including enrolling more than this number, you must seek approval *prior to* implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 210 Administration Building, 5-2208, or irb@uark.edu.