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Facilitation and inhibition abnormalities in obsessive-compulsive disorder

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FACILITATION AND INHIBITION ABNORMALITIES IN OBSESSIVE-COMPULSIVE DISORDER

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THESIS CERTIFICATION

I, Christen Annette Elks, declare that this thesis, submitted in partial fulfillment of the requirements for the award of Doctor of Psychology (Clinical), in the Department of Psychology, University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other academic institution. The document was submitted for examination on 25th February 2011.

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Christen Annette Elks

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ABSTRACT

There has been a disproportionate level of attention invested on inhibitory deficits in OCD to the reciprocal neglect of facilitation processes. This thesis makes an important contribution to the OCD literature by examining changes in both facilitation and inhibition across several tasks by systematically manipulating stimulus repetition sequences and by charting the temporal courses of these processes by varying interstimulus intervals (ISIs).

Study 1 comprised 3 sequential experiments and was designed to overcome limitations of commonly used tasks. Priming and Go/Nogo tasks were modified and trialed at multiple ISIs from 600-1000ms to examine the temporal course of facilitation and inhibition in normal controls. The results supported the use of two paradigms that were employed in Study 2.

Study 2 comprised a systematic examination of facilitation and inhibition abnormalities in OCD. Three groups, OCD (n = 21), normal control (n = 21), and GAD participants (n = 18) completed the modified priming task at 1000ms ISI and the modified Go/Nogo task at two ISIs (600ms and 1000ms). OCD participants exhibited enhanced facilitation compared to anxious and normal control groups on both tasks, indicating that facilitation abnormalities in OCD was a reliable and robust finding and that these effects may be specific to OCD. Further, group differences were best detected at earlier ISIs (600ms), and early in the repetition sequence. The thesis findings contribute to a better understanding of the mechanisms that may underpin clinical symptoms in OCD and have important theoretical and treatment implications. A second valuable contribution of the thesis to both experimental and clinical research is the validation of a new and

effective paradigm that can chart facilitation effects across stimulus repetitions and temporal variations.

THESIS OVERVIEW

People with Obsessive-Compulsive Disorder (OCD) often complain that they "get stuck" on thoughts or images, that these images are "carved on their brains," and that they have difficulty ignoring or discarding them. A self-help manual for sufferers of OCD is even called "brain-lock." From an information processing perspective, these problems may be translated into abnormalities of "facilitation" and/or inhibition. Throughout the OCD literature a disproportionate amount of attention has investigated whether people with OCD have inhibitory deficits, to the reciprocal neglect of facilitation processes in OCD. However, recent findings suggest people with OCD may have both stronger facilitation and weaker inhibition (Bannon, Gonsalvez, & Croft, 2008). Given such information processing abnormalities may contribute to the aetiology of OCD, this thesis aimed to systematically examine both facilitation and inhibition independently in OCD.

Chapter 1 of this thesis provides a brief introduction to OCD by discussing clinical features, prevalence, course, comorbidity, and treatment of OCD. Despite vast research, the aetiology of the disorder remains elusive. Of specific relevance to this thesis are information processing mechanisms of selective attention, such as facilitation and inhibition, which could explain the repetitive quality of obsessions and compulsions in OCD.

Before reviewing research on selective attention in OCD, it is helpful for the reader understand how these processes operate and are measured in a normal population. Accordingly, **Chapter 2** reviews theories of selective attention and critically evaluates paradigms commonly used to measure selective attention (i.e., priming and Go/Nogo paradigms). Salient points to emerge from this review include: (i) recent models posit that selective attention requires both the facilitation of task-relevant stimuli and inhibition of task-irrelevant stimuli; (ii) paradigms

commonly used to measure selective attention confound facilitation and inhibition; (iii) limited research has examined the temporal course of facilitation and inhibition and (iv) commission errors in Go/Nogo tasks may not reflect inhibition failure.

Chapter 3 reviews research investigating selective attention in OCD. To determine whether selective attention abnormalities are specific to OCD or anxiety disorders in general, individuals with generalized anxiety disorder (GAD) were included in Study 2 as an anxious control group. Hence, the clinical features of GAD and research investigating selective attention in GAD are also reviewed in Chapter 3. The salient points to emerge from this review include: (i) behavioural data from studies examining inhibition processes in OCD are inconsistent; (ii) facilitation in OCD is an understudied area, with preliminary findings suggesting stronger facilitation processes are present in OCD; and (iii) limited research has examined facilitation or inhibition in GAD.

An important research question arises from this literature review. Are stronger facilitation and/or weaker inhibition processes present in and specific to OCD? However, because tasks measuring selective attention (e.g., Go/Nogo task) have been criticised on a number of reliability and validity counts, and for confounding facilitation and inhibition, one firstly needs to establish paradigms that disentangle facilitation and inhibition, so these could be measured independently in OCD. **Chapter 4** (Study 1) describes such an endeavour. This study was expected to comprise of one experiment which aimed to corroborate and extend on Bannon et al.'s (2008) findings using the modified priming task. However, it eventuated in a long endeavour to try and capture inhibition, involving delays in designing, programming, pilot-testing, and evaluating several tasks. Specifically, Experiment 1 and 2 evaluated a priming task and Go/Nogo task, respectively. A traditional priming task (Experiment 3) in which target and distractor stimuli were presented

simultaneously was also evaluated. All tasks were completed at multiple interstimulus intervals (ISIs) from 600ms-1000ms, to examine the temporal course of facilitation and inhibition, which is an understudied area in the selective attention literature. This series of experiments was primarily conducted to determine which paradigms would be the most appropriate when assessing facilitation and inhibition in OCD in Study 2. The results indicated that the modified priming and modified Go/Nogo tasks would provide the best information.

Chapter 5 describes Study 2 of this thesis, which investigated whether stronger facilitation and/or weaker inhibition processes are present in and specific to OCD. Accordingly, Study 2 included OCD patients (n = 21), a normal control group (n = 21), and a group of GAD patients for an anxious control group (n = 18). GAD patients were selected for the anxious control group because of the similarity between the repetitive cognitive activity of OCD (obsessions) and GAD (worry). To determine whether selective attention abnormalities in OCD were due to differences in the temporal course of and/or strength of facilitation and/or inhibition, participants completed the modified priming task at one ISI (1000ms) and the modified Go/Nogo twice at two ISIs (600ms and 1000ms).

Consistent with the study's predictions, the OCD group exhibited enhanced facilitation to a primed response compared to normal control and GAD groups indicating stronger facilitation effects may be specific to OCD. These results were robust and were demonstrated on both experiments. Facilitation effects in OCD were also more pronounced at the 600ms ISI, suggesting that facilitation may not only be stronger in OCD, but may also have a different temporal course. Excessive facilitation scores on the modified priming task were also found to correlate with higher scores on the checking subscale of a self-administered inventory. Against predictions, no group differences were found for measures of inhibition on either of the modified

tasks. The implications of these findings, limitations and suggestions for future research are discussed in Chapter 5. A manuscript regarding Study 2's findings has also been submitted to *Behaviour Research and Therapy*.

The major findings and conclusions of this thesis are presented in **Chapter 6**. Overall, this thesis is, we believe, the most comprehensive investigation of facilitation in OCD undertaken to date, because it examined facilitation in OCD across several tasks, across systematic manipulations of sequences to track the development and plateauing of the facilitation process, across ISIs and against a normal control group and anxious control group. A second major contribution is the new Go/Nogo paradigm which provides researchers with multiple dependent measures, each of which can also be analysed as a function of sequence. In summary, this thesis contributes a new paradigm and new findings to both the selective attention and OCD literature and enhances the understanding of the specific aetiological factors that contribute to the development of OCD.

CHAPTER 1

THE NATURE OF OBSESSIVE-COMPULSIVE DISORDER

Recent findings suggest people with OCD may have both stronger facilitation and weaker inhibition (Bannon et al., 2008). Given such selective attention abnormalities may contribute to the aetiology of OCD, this thesis aimed to systematically examine both facilitation and inhibition independently in OCD. The following literature review covers the nature of OCD (Chapter 1) and selective attention processes in a normal population (Chapter 2) and OCD population (Chapter 3). This literature review is presented in three chapters to enhance clarity for the reader.

1.1 Aim

The aim of this chapter is to provide an introduction to Obsessive-Compulsive Disorder (OCD) by discussing clinical features, prevalence, course, comorbidity, treatment, and aetiological theories of OCD. Coverage of the clinical issues is brief and no more than an overview. The main focus of the thesis is selective attention processes in OCD, which will be covered in detail in Chapter 3.

1.2 Clinical features of OCD

OCD is an anxiety disorder that is characterised by the presence of obsessions and/or compulsions (American Psychiatric Association, 2000). Obsessions are recurring intrusive thoughts, images or impulses that are experienced by the individual as uncontrollable and 'ego-dystonic'. That is, the content of the obsessions are unwanted and experienced as alien to one's

self-concept (American Psychiatric Association, 2000). Common obsessions include: fear of contamination, persistent doubt (e.g., wondering if left door unlocked), a need for symmetry or order, somatic fears, and aggressive or sexual images (Foa & Kozak, 1995; Rasmussen & Eisen, 1992; Rasmussen & Tsuang, 1986). The ego-dystonic quality of obsessions causes significant distress, anxiety or guilt are typical. To reduce this distress, the individual typically attempts to suppress the obsessions, or engages in compulsive or ritualistic behaviours to neutralise the obsession (American Psychiatric Association, 2000)

Compulsions may take the form of repetitive overt behaviours or covert mental rituals that are often performed according to rigid rules the individual holds (American Psychiatric Association, 2000). To illustrate, excessive handwashing or cleaning is often performed to reduce anxiety associated with a fear of contamination, while repetitive checking is common in those who experience persistent doubt. In addition to excessive washing and checking, other common compulsions include counting, ordering, and hoarding (Foa & Kozak, 1995; Rasmussen & Eisen, 1992; Rasmussen & Tsuang, 1986). Most people with OCD experience both obsessions and compulsions. A DSM-IV field trial found 96% of individuals with OCD experienced both obsessions and compulsions, while 2% had mainly obsessions and 2% mainly compulsions (Foa & Kozak, 1995). It is also typical for people with OCD to have more than one type of obsession or compulsion (Rasmussen & Tsuang, 1986).

The heterogeneity of OCD clinical presentations has prompted various researchers to identify different subtypes of OCD (e.g., Leckman et al., 1997; Summerfeldt, Richter, Antony, & Swinson, 1999). For example, Leckman et al. (1997) conducted two factor-analytic studies of OCD symptoms and identified four symptom factors: (i) obsessions and checking; (ii) symmetry and ordering; (iii) contamination and cleaning; and (iv) hoarding. This four factor model was

further supported by a confirmatory factor analysis conducted by Summerfeldt and colleagues (1999). Although other studies using factor and cluster analyses of OCD symptoms have yielded different results, one consistent finding is the tendency for 'washers' and 'checkers' to be categorised on different dimensions (e.g., Khanna, Kaliaperumal, & Channabasavanna, 1990; van Oppen, Hoekstra, & Emmelkamp, 1995)

1.3 Prevalence of OCD

Prior to research conducted in the 1980's OCD was considered to be a relatively rare disorder (Antony, Downie, & Swinson, 1998). However, an Epidemiological Catchment Area (ECA) survey conducted in the United States found OCD was the fourth most common psychiatric disorder, with a lifetime prevalence rate ranging from 1.9% to 3.3% across five sites (Karno, Golding, Sorenson, & Burnam, 1988). Other epidemiological studies using DSM-III (American Psychiatric Association, 1980) criteria for OCD indicate similar prevalence rates of OCD across different countries and cultures. For example, 6-month and lifetime prevalence rates of 1.6% and 2.9% were reported in Canada, respectively (Kolada, Bland, & Newman, 1994). In addition, Weissmann and colleagues (1994) found similar prevalence rates in community samples in the United States, Edmonton, Puerto Rico, Munich, Korea, and New Zealand. Lifetime and 1-year prevalence rates in these locations ranged from 1.9% to 2.5% and 1.1% to 1.8%, respectively.

More recently Crino, Slade, and Andrews (2005) found a lower 12-month prevalence rate (0.6%) in an Australian adult population using DSM-IV criteria (American Psychiatric Association, 1994). The authors suggested that the difference between their finding and previously reported prevalence rates may be due to changes in the diagnostic criteria for OCD

from the DSM-III to DSM-IV. That is, the DSM-III has been criticised for being overinclusive compared to the DSM-IV due to: (i) its inability to determine the frequency and distress of OCD symptoms, and (ii) its inability to differentiate between worries (associated with generalized anxiety disorder) and obsessions (Crino et al., 2005). Consistent with their argument Crino and colleagues (2005) found a higher 12-month prevalence rate (2.1%) after their data was rescored for DSM-III criteria. These findings highlight the need for further research on OCD prevalence rates using current diagnostic criteria.

In regards to gender differences in prevalence rates, several epidemiological studies have found that OCD is slightly more prevalent among females (Henderson & Pollard, 1988; Weissman et al, 1994). However, studies with clinical samples failed to demonstrate gender differences (e.g., Faravelli, Degl'lnnocenti, & Giardinelli, 1989; Rasmussen & Eisen, 1992).

1.4 Course of OCD

Although the typical age of onset for OCD ranges from adolescence to early adulthood (Kolada et al., 1994; Rasmussen & Tsuang, 1986), it may begin in childhood in those as young as 6 years old (Kolanda et al., 1994). An earlier age of onset (before age 18) is typically associated with greater severity and poorer prognosis (Sobin, Blundell, & Karayiorgou, 2000). Even in individuals who report an age of onset in adulthood, retrospective reports suggest that signs of the disorder are present at an earlier age (Rasmussen & Tsuang, 1986). Males have a younger mean age of onset than females, which may explain why OCD has been found to be more prevalent in boys than girls (Rasmussen & Tsuang, 1986; Zohar, 1999).

The onset of OCD tends to be insidious in nature rather than acute and attributable to a clear cause, although symptoms do generally exacerbate with stress. The course of OCD has

been reported as chronic and lifelong, with the majority of individuals experiencing a waxing and waning in their symptoms, even with treatment (American Psychiatric Association, 2000; Rasmussen & Tsuang, 1986). Findings from prospective studies on OCD are consistent with retrospective reports. For example, a 5 year follow-up study of 100 people with OCD found only 22% of patients achieved full remission of symptoms, while 53% reported partial remission (Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999). Similarly, Skoog and Skoog (1999) conducted a long-term follow-up study on 144 patients with OCD. Follow-up examinations were conducted after a mean period of 47 years since they were first assessed. They found that although improvement was evident in 83% of patients, 35% continued to suffer clinically significant symptoms and 28% experienced symptoms at a subclinical level. Only 20% of patients completely recovered (Skoog & Skoog, 1999). This suggests that despite some improvement, most OCD patients continue to experience symptoms at a clinical or subclinical level over extended periods of time.

1.5 Comorbidity

It is common for people with OCD to also have a co-existing Axis I disorder (Crino & Andrews, 1996; LaSelle et al., 2004). For example, in Crino and Andrews's (1996) study of lifetime comorbid anxiety and depressive disorders, 86% of 108 patients with OCD met criteria for an additional Axis I diagnosis. In regards to anxiety disorders, 54% of people with OCD also met criteria for lifetime panic disorder with or without agoraphobia, 42% met criteria for social phobia, and 31% met criteria for generalized anxiety disorder. Depression was also common; 50% met criteria for lifetime major depressive disorder and 19% met criteria for lifetime dysthymia (Crino & Andrews, 1996).

1.6 Treatment of OCD

OCD was once believed to be resistant to treatment. Fortunately, since the 1960s significant developments have been made and there are currently two major empirically supported treatment options for OCD: (1) pharmacotherapy, and (2) cognitive-behavioural therapy (CBT; Abramowitz, 2006).

1.6.1 Pharmacotherapy

Research shows that OCD preferentially responds to drugs that block synaptic reuptake of serotonin (Fineberg & Craig, 2009; Fineberg & Gale, 2005). Such drugs include the serotonin reuptake inhibitor clomipramine, and selective serotonin reuptake inhibitors (SSRIs) fluvoxamine, paroxetine, fluoxetine and citalopram (Fineberg & Craig, 2009; Fineberg & Gale, 2005). Findings from placebo-controlled studies support the efficacy of clomipramine (the Clomipramine Collaborative Study Group, 1991), fluvoxamine (Hollander et al., 2003a), paroxetine (Hollander et al., 2003b; Zohar & Judge, 1996), fluoxetine (Jenike, Baer, Minichiello, Raunch, & Buttolph, 1997), and citalopram (Montgomery, Kasper, Stein, Bang Hedegaard, & Lemming, 2001) in the treatment of OCD. While meta-analyses of existing OCD studies suggest superior efficacy of clomipramine over other SSRIs (e.g., Abramowitz, 1997; Ackerman & Greenland, 2002), many studies directly comparing SSRIs with clomipramine have reported equivalent efficacy (e.g., Freeman, Trimble, Deakin, Stokes, & Ashford, 1994; Koran et al., 1996; Pigott et al., 1990; Rouillon, 1998). SSRIs are often the preferred first-line pharmacotherapy treatment for OCD because they are associated with fewer side effects (Fineberg & Craig, 2009; Fineberg & Gale, 2005).

However, it is also important to note that not all OCD patients respond to the aforementioned medications. For example, Rasmussen, Eisen, and Pato (1993) found approximately 30% of OCD patients remained clinically unchanged after an adequate trial of SSRIs. In addition, studies have shown that in those OCD patients who do respond to pharmacological treatment, the majority report only partial improvement in symptoms (e.g., the Clomipramine Collaborative Study Group, 1991; Tollefson et al., 1994). For example, the mean reduction in OCD symptoms (as measured by the Yale-Brown Obsessive-Compulsive Scale) from baseline-to-endpoint was between 38% and 44% in a clomipramine study (the Clomipramine Collaborative Study Group, 1991) and between 21% to 29% in a fluoxetine study (Tollefson et al., 1994). Furthermore, relapse rates tend to be high following discontinuation of OCD medication. In a clomipramine study an 89% relapse rate was found for responsive participants within seven weeks of ceasing the medication (Pato, Zohar-Kadouch, Zohar, & Murphy, 1988). Thus, given the high relapse rates and side effects often associated with pharmacological management of OCD, psychological treatments, such as CBT, have often been utilised (Abramowitz, 2006).

1.6.2 Cognitive Behavioural Therapy

The current primary psychological treatment for OCD is a combination of behavioural and cognitive factors (Abromowitz, 2006). The behavioural approach to the treatment of OCD principally comprises exposure and response prevention (ERP). ERP requires OCD patients to confront their feared stimulus, often in a systematic and prolonged manner (exposure), without engaging in any safety behaviours or compulsions (response prevention). From a behavioural perspective ERP is hypothesised to work through conditioning principles such as habituation and

extinction to their feared stimulus. However, the cognitive model posits that ERP reduces anxiety because it results in a change in maladaptive beliefs regarding the feared stimulus (Abramowitz, Braddock, & Moore, 2009).

Cognitive approaches to OCD propose that an individual's appraisals are central to the maintenance of OCD symptoms and such obsessions and compulsions can therefore be reduced by modifying problematic cognitions (Shafran, 2005). For example, Salkovskis (1985) proposed that normal intrusive thoughts will be appraised by an individual vulnerable to OCD as indicating that they are personally responsible for causing harm to themselves or others. The distress and anxiety experienced as a consequence of this appraisal may then lead to compulsive (or preventive) behaviours aimed to neutralise the perceived threat or anxiety (e.g., checking compulsions). As cognitive models assume that misinterpretation of normal experiences causes OCD symptoms, cognitive therapy aims to help the patients identify and modify maladaptive cognitive beliefs and information processing biases that underlie and maintain their obsessions and compulsions (Abramowitz et al., 2009).

The efficacy of ERP is established with evidence from several randomly controlled studies (Fals-Stewart, Marks, & Schafer, 1993; Foa et al., 2005; Lindsay, Crino, & Andrews, 1997; van Balkom et al., 1998) and by systematic meta-analyses (Abramowitz, 1996; Foa & Kozak, 1996; O'Kearney, 2007). For example, across 12 ERP studies Foa and Kozak (1996) found 83% of patients were treatment responders (at least 30% improvement in symptoms) after a mean of 15 sessions. In addition, 76% were treatment responders at follow-up (an average of 29 months later) across 16 ERP studies (Foa & Kozak, 1996). While these results are encouraging, they also highlight that not all OCD patients respond well to ERP and in those who do, full remission from symptoms is not typical.

Compared to the many studies examining ERP, too few studies have been conducted on cognitive therapy to conclude whether it is more effective than ERP or whether it can improve the effectiveness of ERP. Studies comparing ERP and cognitive therapy have found mixed results with some reporting no difference between treatments at posttreatment (Cottraux et al., 2001; van Balkom et al., 1998) and others reporting superior efficacy for ERP (McLean et al., 2001) or cognitive therapy (van Oppen, de Haan et al., 1995). Furthermore, the combination of ERP and cognitive therapy has not been found to be more effective than ERP alone (Vogel, Stiles, & Gotestam, 2004). Whether or not cognitive therapy improves the efficacy of ERP may however, be a moot point because clinicians who use ERP will also routinely discuss cognitive distortions (Foa, Franklin, & Kozak, 1998). For example, discussions about the overestimation of danger and costs of avoidance and compulsive behaviour often occur when explaining the rationale for ERP (Abramowitz et al., 2009). Hence, although it is not known yet whether cognitive therapy can improve the effectiveness of ERP, empirical evidence demonstrates that ERP is an essential component in the psychological treatment of OCD.

In summary, empirical evidence demonstrates that CBT or medications that have an effect on serotonin are effective treatment options for adults with OCD. However, it is also apparent that not all OCD patients respond to such treatments. Given the chronicity of OCD, it is important to try to understand the aetiological factors that contribute to the development of OCD.

1.7 Aetiological Theories of OCD

The elusiveness of the specific aetiological factors involved in the development of OCD is reflected in the variety of factors hypothesised to contribute to its origin. Examples of posited aetiological factors include those based on neuropsychiatry, genetics, cognitive behavioural

theories, and information processing theories. Each of these will be explained briefly, whilst selective attention processes in OCD will be covered more comprehensively in Chapter 3 as it relates more specifically to the topic of this thesis.

1.7.1 Neuropsychiatric Theories

Neuropsychiatric theories of OCD include both neurochemical and neuroanatomical theories. In regards to neurochemical theories, abnormalities in the serotonin system, particularly the hypersensitivity of postsynaptic serotonergic receptors, have been proposed to underlie OCD symptoms (Gross, Sasson, Chorpa, & Zohar, 1998). The 'serotonin hypothesis' has been supported by different sets of research. Firstly, OCD symptoms have been found to preferentially respond to drugs that block synaptic reuptake of serotonin over drugs that primarily inhibit other neurotransmitters such as norepinephrine (e.g., Goodman et al., 1990). Secondly, acute administration of serotonin agonists has been shown to exacerbate symptoms in OCD patients (e.g., Zohar, Mueller, Insel, Zohar-Kadouch, & Murphy, 1987).

Although these findings suggest dysregulation of the serotonergic system might contribute to OCD, to date no specific abnormality in the serotonergic system has been identified as the cause (Dougherty, Rauch, & Greenberg, 2009). Furthermore, the preferential response of OCD to serotonergic medication does not necessarily indicate that OCD is caused by a dysfunction in the serotonergic system. Thus, whether the dysregulation of the serotonergic system mediates OCD symptoms remains unclear and requires further research (Dougherty et al., 2009).

Neuroanatomical theories of OCD posit that obsessions and compulsions are caused by structural and functional abnormalities in the brain. In a review of functional neuroimaging

studies Saxena and Raunch (2000) reported that most studies found evidence for elevated activity in the orbitofrontal cortex in untreated OCD patients, which decreased in response to treatment. Abnormalities in the caudate nucleus and anterior cingulate cortex were also found in those with OCD (Saxena & Raunch, 2000). Given these structures are implicated within the lateral orbitofrontal loop, Chamberlain and colleagues suggested that OCD may be characterised by abnormal processing within the lateral orbitofrontal loop (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). However, the authors assert that further work is needed to understand the neurobiology of OCD.

1.7.2 Genetics

Evidence from twin and family studies indicates that genetic factors may play a role in the aetiology of OCD. Specifically, research has shown the concordance rates of OCD are significantly higher in monozygotic twins compared to dizygotic twins (American Psychiatric Association, 2000; Carey & Gottesman, 1981). For example, Carey and Gottesman (1981) reported concordance rates of 80% and 50% for monozygotic and dizygotic twins, respectively. In addition, after reviewing OCD twin studies van Grootheest and colleagues concluded that OCD symptoms are heritable, with genetic influences ranging from 45% - 65% in children and 27% - 47% in adults (van Grootheest, Cath, Beekman, & Boomsma, 2005).

Findings from family studies also indicate that OCD is a familial disorder, as the incidence rate of OCD in relatives of OCD probands has been reported as higher than the incidence of OCD in relatives of controls (e.g., Black, Noyes, Goldstein, & Blum, 1992; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995; Nestadt et al., 2000). For example, Nestadt et al. (2000) found that the prevalence rate of OCD in relatives of OCD probands was

12%, compared to the 3% found in relatives of control participants. Thus, evidence from both twin and family studies suggests there is a genetic component to OCD. However, if OCD were solely caused by genetic factors we would expect a 100% concordance rate in monozygotic twins. Given the lack of evidence for this, it is likely that non-genetic factors are also implicated in the aetiology of OCD.

1.7.3 Cognitive Behavioural Theories

Early behavioural theories of OCD were based on Mowrer's (1960) two-stage learning theory. In this two-factor model, an obsessional fear is believed to develop when a neutral event is paired with an adversive stimulus (classical conditioning), and this fear is maintained by behaviours that prevent the natural extinction of this fear, such as avoidance or compulsive behaviours (operant conditioning). In later years this behavioural model of OCD has received various criticisms, including its inability to account for the finding that most obsessions are not the result of traumatic learning (Clark & Purdon, 2004).

More recent models have focused on both cognitive and behavioural factors that may contribute to the development and maintenance of OCD. Salkovskis's (1985) responsibility theory of OCD is an example of one such model. As outlined previously, Salkovskis (1985) proposed that normal intrusive thoughts will be appraised by an individual vulnerable to OCD as indicating that they are personally responsible for causing harm to self or others. The direct effect of such responsibility appraisals are distress and anxiety about the intrusions, attempts to neutralise the intrusions or prevent harm via various overt (e.g., checking) or covert compulsive activities (e.g., mental rituals), and attempts at cognitive control of the intrusions.

While there is some empirical support for Salkovskis's hypothesis that perceived responsibility for the occurrence and prevention of negative outcomes may be critical in OCD, Salkovskis's theory has also received various criticisms (Clark & Purdon, 2004). For example, O'Kearney (1998, 2001) argued that Salkovskis theory lacks a coherent explanation of its implied motivational properties and overlooks the role of affective factors in the phenomenology and pathogenesis of OCD. In addition, there is no evidence that responsibility appraisals have a more prominent role in the pathogenesis of obsessions than other dysfunctional beliefs such as the overimportance and control of thoughts, perfectionism, thought-action fusion, intolerance of uncertainty, or overestimation of threat (Clark & Purdon, 2004). Furthermore, Clark and Purdon (2004) highlight that it is unclear whether responsibility beliefs are a cause or a consequence of OCD, or whether the function of compulsive rituals and other neutralising behvaiour is to reduce perceived responsibility. Hence, it may be that the role and function of perceived responsibility appraisals in the pathogenesis of OCD is more limited than proposed by Salkovksis (Clark & Purdon, 2004).

1.7.4 Information Processing Accounts of OCD

Experimental psychopathologists have increasingly employed paradigms from cognitive psychology to examine whether people with anxiety disorders have information processing abnormalities that may cause or contribute to their symptoms (Amir & Kozak, 2002; McNally, 2000). Common areas of interest for information processing researchers include memory and attention processes in anxious individuals (Amir & Kozak, 2002; MacLeod & Rutherford, 2004; McNally, 2000). Research on memory processes in OCD and attentional bias will be briefly reviewed, whilst a more comprehensive review of general selective attention processes in OCD will be covered in Chapter 3, as it relates more specifically to the topic of this thesis.

1.7.4.1 Memory in OCD

The repetitive quality of OCD symptoms suggests that OCD patients often experience doubt about their memory (e.g., "Did I turn the iron off"). Research has investigated whether this doubt reflects: (a) general memory deficits; (b) a specific memory deficit for OCD-relevant material; or (c) low memory confidence (Amir & Kozak, 2002; Foa, Amir, Gershuny, Molnar, & Kozak, 1997; Tolin et al., 2001). The evidence is mixed regarding the presence of a general memory deficit in OCD. While some studies indicate OCD patients show a general deficit in verbal or nonverbal memory compared to healthy controls (e.g., Savage et al., 1996; Segalas et al., 2008; Tuna, Tekcan, & Topcuoglu, 2005; Zitterl et al., 2001), others studies have failed to find evidence for such general memory deficits (e.g., Foa et al., 1997; Jelinek, Moritz, Heeren, & Naber, 2006; Moritz, Ruhe, Jelinek, & Naber, 2009; Tolin et al., 2001). Various experimental factors may have contributed to such mixed findings across studies. Such factors include methodological differences, sample size, and contextual factors (such as speed of item administration because slow participants will display a deficit in performance which may be misinterpreted as a memory deficit; Moritz et al., 2009).

When investigating whether OCD patients display a memory deficit for OCD-related stimuli, several studies have found OCD patients are actually more accurate at remembering OCD threat-relevant actions and objects (Constans, Foa, Franklin, & Mathews, 1995; Radomsky & Rachman, 1999). In contrast to these findings, in a study conducted by Tolin and colleagues (2001) the OCD group did not show greater recall for unsafe objects compared to normal and anxious controls. A more consistent finding throughout the OCD memory literature is that OCD patients report lower levels of memory confidence compared to controls (Constans et al., 1995; Tolin et al., 2001; Tuna et al., 2005; Zitterl et al., 2001), and that enhanced responsibility

decreases memory confidence in OCD (Boschen & Vuksanovic, 2007; Moritz et al., 2007). Interestingly, several studies have also found that repeated checking leads to a paradoxical decrease in memory confidence (Boschen & Vuksanovic, 2007; Tolin et al., 2001).

In summary, although it is unclear from the empirical literature whether general or specific memory deficits are present in OCD, the evidence does suggest that people with OCD have low memory confidence which may be influenced by factors such as enhanced responsibility and repeated checking. For a more comprehensive review of memory functioning in OCD see Muller and Roberts (2005), Amir and Kozak (2002) or McNally (2000).

1.7.4.2 Attentional Bias in OCD

Like the memory literature, research on attention processes in OCD has investigated whether: (a) OCD patients have a specific attentional bias for OCD threat-relevant stimuli; or (b) OCD patients have general abnormalities in selective attention. Studies examining whether OCD patients are sensitive to and preoccupied with OCD threat-relevant stimuli have often used the emotional Stroop paradigm. The emotional Stroop task involves the presentation of words with varying emotional significance and participants are required to ignore the meaning of the word and instead name the colour ink the words are printed in as quickly as possible. Longer response latencies when colour naming emotionally threatening words compared to neutral words is said to reflect a specific attentional bias for threat because people have more difficulty inhibiting the semantic content of threatening words (compared to neutral words; Williams, Mathews, & MacLeod, 1996).

Using the emotional Stroop task several authors have found evidence for specific attentional biases in OCD (Foa, Ilai, McCarthy, Shoyer, & Murdock, 1993; Lavy, van Oppen, &

van den Hout, 1994). In a study conducted by Lavy et al. (1994) 33 OCD patients exhibited delays in colour naming words negatively related to obsessions and compulsions compared to 29 healthy controls. Importantly, OCD patients did not show deficits to words positively related to obsessions and compulsions or to general threat words. Similarly, in a study with 23 OCD washers, 10 OCD non-washers, and 14 healthy controls, OCD washers produced longer response times for contamination words than neutral words compared to non-washers and controls (Foa et al., 1993). In contrast, OCD non-washers displayed longer response times for general threat words relative to OCD washers and controls. Such differences occurred despite similarities between OCD groups in terms of OCD severity.

In contrast to these findings, several authors have also failed to find attentional bias in OCD using the emotional Stroop task (Kampman, Keijsers, Verbraak, Naring & Hoogduin, 2002; Kyrios & Iob, 1998). However, neither of these studies employed OCD threat-relevant stimuli that were individually tailored to the particular concerns of their OCD patients. In combination with Foa et al.'s (1993) results, such findings suggest that selective processing of threat stimuli may be specific to the OCD patient's idiographic concerns (Muller & Roberts, 2005).

Of interest to this thesis is whether people with OCD display *general* abnormalities in selective attention. Selective attention is posited to comprise of (i) facilitation of task-relevant stimuli and (ii) inhibition of task-irrelevant stimuli (Houghton & Tipper, 1994). Recent research has suggested that people with OCD may have both stronger facilitation and weaker inhibition, and that these selective attention abnormalities may contribute to the repetitive quality of obsessions and compulsions (Bannon et al., 2008). This area of investigation will be explored further in Chapter 3.

1.8 Summary

OCD is an anxiety disorder characterised by repetitive obsessions and/or compulsions. Although symptoms of OCD fluctuate with stress, they typically follow a chronic course with most OCD patients experiencing symptoms at a clinical or subclinical level over extended periods of time. In many cases, such symptoms are associated with distress and functional impairment in various areas such as work or academic performance, and socialising. Additionally, many individuals with OCD also experience comorbid Axis I disorders, such as depression.

Although research shows that serotonin reuptake inhibitors and CBT are effective treatment options for OCD, it is also apparent that not all OCD patients respond to such treatments, and in those who do, only partial remission of symptoms is typical. Thus, given the chronic and debilitating nature of OCD it is important to elucidate the aetiological factors that contribute to OCD symptoms to aid the development of more effective treatments for OCD. Despite the variety of factors posited to explain the aetiology of OCD, it seems likely that a combination of biological and psychological factors contribute to the origin and maintenance of OCD. However, the specific mechanisms involved in the development of OCD remain elusive. Hence, this thesis systematically examines one of these hypothesised factors, by investigating whether abnormalities in selective attention, such as facilitation and inhibition, are present in OCD.

CHAPTER 2

SELECTIVE ATTENTION PROCESSES IN A NORMAL POPULATION

2.1 Aim

In order to investigate facilitation and inhibition in OCD a thorough understanding of how these processes operate and are measured in a normal population is required. Thus, this chapter briefly reviews theories of selective attention and critically evaluates prominent paradigms used to measure selective attention.

2.2 Theories of Selective Attention

In the face of many competing stimuli efficient goal-directed behaviour depends on one's ability to select and respond to task-relevant stimuli, whilst ignoring task-irrelevant stimuli (Houghton & Tipper, 1994; Neumann & DeSchepper, 1991). Consistent with the important role this process plays in adaptive behaviour, selective attention is arguably one of the most prolific topics within cognitive psychology (Driver, 2001; Milliken, Joordens, Merikle, & Seiffert, 1998).

Various theories of selective attention have been proposed (e.g., Broadbent, 1958; Deutsch & Deutsch, 1963; Houghton & Tipper, 1994). The two main elements of variation in theories of selective attention are the locus of selection (i.e., early versus late selection) and mechanisms of selection (Houghton & Tipper, 1994).

2.2.1 Early versus late selection theories

'Early selection' theories of selective attention propose that selection occurs prior to categorisation of stimuli and that attention is therefore necessary for the categorisation process (Houghton & Tipper, 1994). Broadbent's (1958) filter theory is an example of an 'early selection' approach. Broadbent postulated that one channel within a particular sensory modality (e.g., sight) is selected for attention based on physical features and that after this selection task-relevant information would be further processed for meaning. In contrast, task-irrelevant information would be filtered out before it could be processed for meaning, which protects the limited capacity system from overloading (Broadbent, 1958).

'Late selection' theories of selective attention (e.g., Deutsch & Deutsch, 1963; Norman, 1968) assert that both task-relevant and irrelevant stimuli are fully analysed prior to selection. In Deutsch and Deutsch's (1963) model all information is analysed, but only information that is important to the individual at that time is selected for full awareness. In contrast to the early selection theories in which attention is said to be necessary for categorising stimuli, late selection theories posit that attention serves the purpose of selecting one's response to stimuli (Houghton & Tipper, 1994).

2.2.2 Single versus dual mechanism theories

Facilitation and inhibition are the two mechanisms by which selective attention may be achieved (Houghton & Tipper, 1994). Early theories of attention (e.g., Broadbent, 1958; van der Heijden, 1981) held that the selection was achieved via the facilitation or excitation of goal-relevant stimuli, while unattended stimuli passively decayed back to resting levels. 'Spotlight' models of attention provide a good example of a single mechanism theory (Houghton & Tipper,

1994). In such models attention is likened to a beam of spotlight, which amplifies the information within its beam allowing such information to be processed further. Hence, selection of target from distractor stimuli appears to depend on facilitation processes, with no active role proposed for inhibitory processes (Broadbent, 1958; van der Heijden, 1981).

In contrast, more recent models of selective attention contend that selective attention requires both the amplification of task-relevant stimuli *and* active suppression of task-irrelevant stimuli (Houghton & Tipper, 1994; Neumann & DeSchepper, 1991). These dual mechanisms are said to allow for more efficient selection as the target stimulus can be differentiated from distractor stimuli faster than if a single mechanism was operating (Houghton & Tipper, 1994). Such dual mechanisms are argued to be independent processes operating in selective attention (Houghton & Tipper, 1994; Neumann & DeSchepper, 1991), and preliminary findings from brain imaging research appear to support this contention. Wright et al. (2006) examined the brain correlates of inhibition and facilitation processes being tapped in a visuospatial priming task. They found these processes activated and deactivated non-overlapping areas of the prefrontal cortex, suggesting such processes operated independently.

2.2.3 Houghton & Tipper's (1994) selective attention model

Houghton and Tipper (1994) developed a comprehensive connectionist model of selective attention. It asserts that both excitation and inhibition mechanisms are used in selection, and that this selection occurs after perceptual grouping and meaning analysis (late selection). This model will be examined more closely as it has been referred to extensively in explaining the phenomenon of negative priming.

Houghton and Tipper's (1994) model asserts that stimuli in our environment are processed by both external perceptual inputs (bottom up information) and internal goal-driven inputs (top down information). For example, when two objects (target and distractor) are presented both will receive excitatory activation from external perceptual inputs while they are present. Such objects will also be compared to goal-driven templates. That is, individuals are said to hold a template or schema for target stimuli based on their current goals. Stimuli are compared to this template and if they match they receive excitatory feedback from internal inputs; if they don't match they receive inhibitory feedback (see Figure 2.1; Houghton & Tipper, 1994, p. 72).

The combined excitatory activation from external and internal inputs for the target stimulus increases its activation level and makes it easier to select and respond to. If the target stimulus is repeated a faster response time is observed (Houghton & Tipper, 1994). Response time differentials are said to be a reflection of the activation state for the target's internal representation (Milliken & Joordens, 1996). Hence, the response time advantage for repeated target stimuli is used as a measure of facilitation processes.

In regards to the distractor, when target and distractor stimuli are presented the distractor stimulus receives both excitatory activation from external inputs and inhibitory feedback from internal inputs. Hence, although its activation level is lower than the target's, its activation level is still above baseline. However, when the target and distractor stimuli are removed, the external excitatory activation ceases for the distractor stimulus while its internal inhibitory feedback continues. This results in the activation level for the distractor stimulus being suppressed below baseline, and has been referred to as an 'inhibitory rebound' (see Figure 2.2; Houghton & Tipper, 1994, p. 92). If the distractor is subsequently presented as a target during this inhibitory rebound

period Houghton and Tipper argue that response times will be delayed, because the individual needs time to overcome the active suppression of the internal representation of the distractor in order to respond to it as a target. In priming studies this response time cost has been termed 'negative priming' and is said to be a measure of inhibition processes (Tipper, 1985).

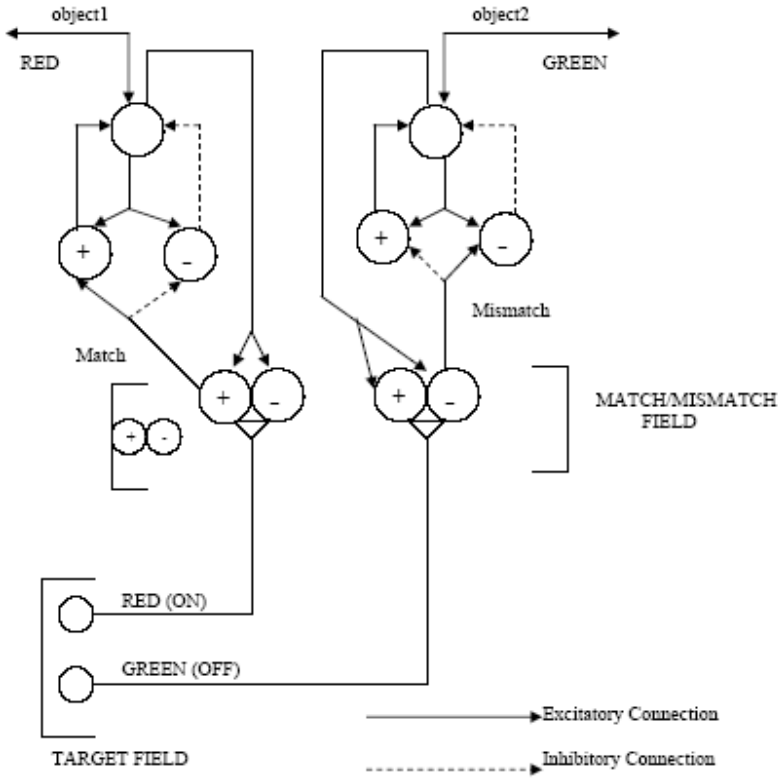


Figure 2.1. **The excitation and inhibition of target and distractor stimuli in Houghton and Tipper's (1994) connectionist model. Two objects are presented, one in red and one in green. During the presentation of the objects, excitatory signals from both colour cells in the object field are sent to the match/mismatch field. Information for the goal-driven template (red) is held in the target field, which sends excitatory signals to the red object in the match/mismatch field. In contrast, inhibitory signals are sent to the green object. The red object receives only excitatory feedback, whereas the green object receives both excitatory feedback from the object field and inhibitory feedback from the goal-driven target field. This results in a lower level of activation for the distractor object compared to the target. Figure from "A Model of Inhibitory Mechanisms in Selective Attention" by G. Houghton & S. P. Tipper, 1994, In D. Dagenbach & T. H. Carr (Eds.),** ***Inhibitory Processes in Attention, Memory and Language,*** **p. 72. Copyright 1994 by Academic Press.**

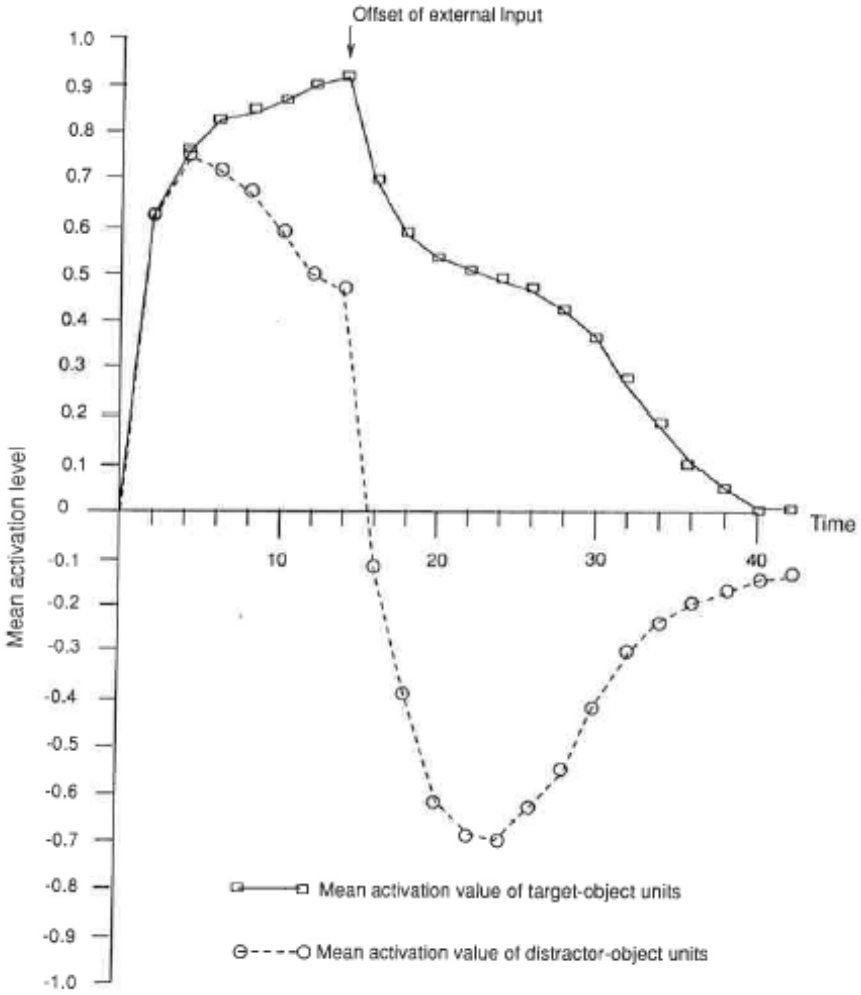


Figure 2.2. **The temporal course of facilitation and inhibition effects as shown in Houghton and Tipper's (1994) model. The two lines show the mean activation level of target and distractor stimuli across time in a priming task. When the target and distractor stop being presented external excitatory input for the distractor ceases while the internal inhibitory feedback continues, this causes the activation level for the distractor representation to be suppressed below resting levels. This is called the inhibitory rebound. Figure from "A Model of Inhibitory Mechanisms in Selective Attention" by G. Houghton & S. P. Tipper, 1994, In D. Dagenbach & T. H. Carr (Eds.),** ***Inhibitory Processes in Attention, Memory and Language,*** **p. 92. Copyright 1994 by Academic Press.**

Hence, although both inhibition and facilitation processes are deemed to work together in selective attention, Houghton and Tipper proposed that they are independent mechanisms that have different temporal courses (as can be seen in Figure 2.2; Houghton & Tipper, 1994, p. 92). Consistent with this, other researchers have also suggested inhibition and facilitation have

different time courses (Kok, 1999; Salo, Henik, Nordahl & Robertson, 2002), with findings from priming studies suggesting facilitation is a stable process across time (Salo et al., 2002; Waechter, Stolz, & Besner, 2010), whereas inhibition may decay with time (Neill & Westberry, 1987).

2.3 Measures of Selective Attention

To understand facilitation and inhibition processes further we must examine two of the prominent experimental methods used to measure such processes: (1) Priming paradigm (Milliken et al., 1998); (2) Go/Nogo paradigm (Simmonds, Pekar, & Mostofsky, 2008).

2.3.1 Priming Paradigm

In a traditional priming task, target and distractor stimuli are presented simultaneously in the initial prime trial and subsequent probe trial. Participants are required to respond to the target stimulus and ignore the distractor stimulus in these trials (Tipper, 1985). For example, in Tipper's (1985) seminal study participants were presented with two superimposed pictures on prime and probe trials, and were instructed to select (i.e., name) the drawing in the colour red, while ignoring the drawing in the colour green. Manipulating the relationship between stimuli presented in the prime and probe trials is said to enable the examination of facilitation and inhibition processes. That is, responses on attended repetition and ignored repetition probe trials are compared to control trials and differences in response accuracy and latency are referred to as priming effects (Milliken et al., 1998). In the attended repetition condition the target stimulus is repeated on prime and probe trial, whereas in the ignored repetition condition the distractor stimulus from the prime trial is re-presented as the target in the probe trial. These conditions are

compared to the control condition in which no target or distractor stimuli are repeated in prime and probe trials (see Figure 2.3).

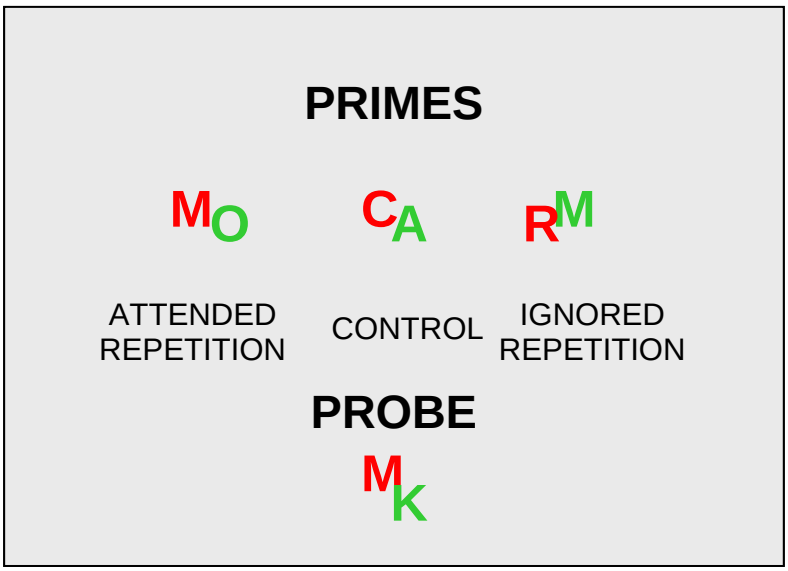


***Figure 2.3*. Examples of prime and probe displays in a priming paradigm. Letters in red are selected (target stimulus) and letters in green are ignored (distractor stimulus).**

A faster response time in the attended repetition condition relative to the control is called positive priming. Positive priming effects are used as a measure of facilitation because it is believed that the ***activation level of the target from the prime display carries over and facilitates activation*** of the same stimulus in the probe trial (Stadler & Hogan, 1996). In contrast, a slower response in the ignored repetition trial relative to the control is called negative priming. Tipper and colleagues argue that the negative priming effect reflects inhibition processes, as the ***internal representation of the distractor object is actively suppressed*** in the prime trial and the suppression of its activation lingers and subsequently causes a slowed response time to this same object when it becomes a target in the probe trial (Houghton & Tipper, 1994; Tipper, 1985; Tipper, Weaver, Cameron, Brehaut, & Bastedo, 1991).

The example of Tipper's (1985) identification task is just one of the many forms priming tasks can take. Other forms include word or letter naming, semantic categorisation, lexical decision, letter or shape matching, and localisation tasks (Neill, Valdes & Terry, 1995). The priming literature makes a distinction between ***identity priming*** and ***location priming*** tasks (Gibbons & Rammsayer, 2004). In identity priming tasks participants base their response on the identity of the stimuli (e.g., colour or name), whereas location priming tasks require participants to report the location of the relevant stimulus. For example, in a study conducted by Tipper and colleagues (1991), participants had to press one of four buttons on the keyboard to indicate the location of the character 'O' on the screen. They found negative priming effects in the probe trial when 'O' was re-presented in the same position as the distractor character '+' was in the prime trial (Tipper et al., 1991).

A distinction is often made between identity and location priming paradigms because the processes underlying performance on such tasks may be different (Gibbons & Rammsayer, 2004). Findings from neurophysiological data suggest that object identity and object location are processed by separate cortical systems (Ungerleider & Mishkin, 1982). Several authors have cautioned against the generalisation of identification to localisation tasks, and vice versa (Fox, 1995; May, Kane, & Hasher, 1995). Although there is a body of literature on each of these areas of investigation, models of identity priming are more relevant to theoretical conceptualisations of OCD, so the current review focuses on facilitation and inhibition effects found in identity priming paradigms.

2.3.1.1 Priming Paradigm and Facilitation

As stated previously, the priming paradigm has been a popular method for measuring facilitation processes. The positive priming effect is a robust finding in identity priming tasks

(Gibbons, Rammsayer, & Stahl, 2006; Groh-Bordin & Frings, 2009; Koshino, Boese, & Ferraro, 2000; Lowe, 1979; Neumann & DeSchepper, 1991; Salo et al., 2002; Stadler & Hogan, 1996; Tipper, 1985; Troche, Gibbons, & Rammsayer, 2008; Waechter et al., 2010). For example, Neumann & DeSchepper (1991) utilised an identity priming task in which two letters were shown in prime and probe trials, one presented in red (target) and the other presented randomly in one of four distractor colours. Participants were required to remember the target letter in the prime trial, name the target letter in the probe trial and subsequently recall the target letter in the prime trial. As predicted, the repetition of target stimuli in the prime and probe trial produced a response time advantage compared to the control (Neumann & DeSchepper, 1991). This finding has been a consistent one despite experimental variations in response modality (e.g., vocal naming versus keypress response) and differences in the types of stimuli used including letters, words/nonwords, pictures, numbers, and colours (Lowe, 1979; Neumann & DeSchepper, 1991; Stadler & Hogan, 1996; Tipper, 1985; Waetcher et al., 2010).

In regards to temporal course, studies examining positive priming have found positive priming effects across a range of time intervals between the presentation of prime and probe trials. Specifically, positive priming effects have been found in studies using response or interstimulus intervals of 400ms, 500ms, 600ms, 1000ms, 1200ms, and 2000ms (Gibbons et al., 2006; Koshino et al., 2000; Lowe, 1979; Neumann & DeSchepper, 1991; Salo et al., 2002; Stadler & Hogan, 1996; Tipper, 1985). These findings appear to be in line with Houghton and Tipper's (1994) assertion that repetition priming effects persist for "some time" (p. 89). Exactly what 'some time' means however, is unclear as only two positive priming studies have utilised multiple time intervals between prime and probe to examine the temporal course of facilitation (Salo et al., 2002; Waetcher et al., 2010). Salo and colleagues (2002) examined the time course

of facilitation using a Stroop priming task at two response stimulus intervals (RSI): 500ms and 2000ms. They found facilitation priming effects at both RSIs within the normal control group (Salo et al., 2002). Similarly, Waetcher and colleagues (2010) found a response time benefit for repeated targets compared to unrepeated targets in a word identification task at all three interstimulus intervals (ISI; 50ms, 200ms, and 650ms). These studies indicate that facilitation is a consistent finding across different time intervals in a normal population. However, further research examining the temporal course of facilitation in healthy controls and clinical populations (e.g., OCD) is needed to corroborate and extend these findings. That is, the temporal course of facilitation in OCD may be different to healthy controls (e.g., facilitation persists for longer in OCD). Such differences in the temporal course of facilitation may contribute to the repetitive nature of obsessions and compulsions. This will be examined in Study 2.

Another important question is whether facilitation is a reliable finding in identity priming tasks. If a task measuring selective attention has low reliability it reduces the chance of finding a significant difference between groups (Waetcher et al., 2010). This is extremely pertinent to the priming literature which has found differences in negative or positive priming effects between normal controls and clinical disorders (e.g., obsessive compulsive disorder, schizophrenia) and interpreted such differences as evidence of reduced inhibition (Beech, Powell, McWilliams, & Claridge, 1989; Enright & Beech, 1993a, 1993b) or excessive facilitation processes (Bannon et al., 2008; Hartston & Swerdlow, 1999).

As far as the author is aware only two studies to date have examined the reliability of the positive priming effect, with both suggesting facilitation is a reliable finding (Troche et al., 2008; Waetcher et al., 2010). To examine the reliability of repetition priming, Troche and colleagues (2008) had young and older participants complete a number identification task twice (two weeks

apart). Test-retest coefficients suggested temporal stability for facilitation effects in both young (r = 0.63) and older (r = 0.89) adults (Troche et al., 2008).

In addition, Waetcher and colleagues (2010) examined the within subject reliability of repetition priming by correlating participant's repetition priming scores on Block 1 of a word identification task with their scores on Block 2. In this task participants were required to silently read the word presented in the prime trial and then press one of two keys on the keyboard to indicate whether the word presented in the probe was a word or non-word at three ISIs: 50ms, 200ms, and 650ms. Waetcher et al. (2010) repeated this experiment three times, using three different repetition proportions (RP; 25%, 50% & 75%). They found repetition priming effects were reliable at all three ISIs when the likelihood of the prime being repeated in the probe was low (25%). At a RP of 50% reliable repetition priming was found at ISIs of 50ms and 200ms, but not 650ms. Similarly, only medium and long ISIs (200ms and 650ms) exhibited reliable repetition priming at an RP of 75% (Waetcher et al., 2010).

Waetcher and colleagues (2010) concluded that overall these results indicate that repetition priming is reliable in most RP and ISI conditions, and an interplay of controlled and automatic processes may explain the absence of reliability in the other two conditions. For example, using controlled processing when the RP is high (75%) and the ISI is short (50ms) may not have given participants enough time to succeed, resulting in low reliability of scores in that priming condition. Hence, overall it appears that facilitation effects in identity priming tasks are a consistent and reliable finding within subjects and across tasks. However, given the limited studies examining the time course of facilitation, more research examining its temporal course is needed to determine whether it is stable over time. This thesis aims to examine this further in Study 1.

2.3.1.2 Priming Paradigm and Inhibition

The negative priming effect has been argued to reflect inhibition processes (Tipper, 1985). As can be seen in Table 2.1, of 23 studies involving 57 identity priming experiments, 37 experiments reported negative priming effects and 20 did not.

The presence or absence of negative priming effects has been attributed to variations in experimental factors such as experimental instruction (e.g., emphasis on speed instead of accuracy), design (e.g., lack of probe distractor stimuli, repetition and number of stimuli used, contextual similarity between prime and probe trials) and temporal manipulation (Lowe, 1998; Malley & Strayer, 1995; Moore, 1994; Neill & Valdes, 1992; Neill & Westberry, 1987). These factors are explored in greater detail below.

Experimental instruction: Several studies have found that emphasising speed or accuracy affects negative priming effects (Neill & Westberry, 1987; Neumann & DeSchepper, 1992). Generally it has been found that when instructing participants to respond as fast as possible, making accuracy a lower priority, positive priming effects are observed (Neill, 1977, Experiment 2; Neill & Westberry, 1987, Experiment 1; Neumann & DeSchepper, 1992, Experiment 2). In contrast, emphasising accuracy over speed produces the expected negative priming effect (Neill & Westberry, 1987, Experiment 1 and 2; Neumann & DeSchepper, 1992, Experiment 2).

Table 2.1. *Summary of Findings from Studies Examining Identity Negative Priming*

Study	*n*	Stimuli	NP observed
Neill			
(1977; Exp. 1)	8	Stroop colour words	✓
(1977; Exp. 2)	6	Stroop colour words	✗
Lowe			
(1979; Exp. 1, 2)	8, 20	Stroop colour words	✓
(1979; Exp. 3, 4)	16, 8	Stroop colour words, colour patches & letters	✗
Allport et al.			
(1985; Exp. 1,2,3)	20, 34, 24	Letters and pictures	✓
(1985; Exp. 9)	20	Pictures	✗
Tipper			
(1985; Exp. 1, 2, 3)	24, 22, 11	Pictures	✓
Tipper & Cranston			✓
(1985; Exp. 1, 2)	20, 12	Letters	
(1985; Exp. 3)	24	Letters	✗
Neill & Westberry			
(1987; Exp. 1)	16	Stroop colour words	✗
(1987; Exp. 2)	12	Stroop colour words	✓
Neumann & DeSchepper			
(1991; Exp 3)	55	Letters	✓
Tipper et al.			
(1991; Exp. 1, 2)	84, 28	Pictures	✓
Neill & Valdes			
(1992; Exp. 1, 2, 3)	26, 20, 24	Letters	✓
Moore			
(1994; Exp. 1, 2, 3, 4)	16	Letters	✗
Tipper, Weaver & Houghton			
(1994; 1, 2)	30, 30	Letters	✗
(1994; 3)	30	Letters	✓
Malley & Strayer			
(1995; Exp. 1,2)	35, 40	Words	✗
(1995; Exp. 3,4)	40, 40	Words	✓
Milliken & Joordens			
(1996; Exp. 1,2)	16, 16	Words	✓
(1996; Exp. 3)	16	Words	✗

Stadler & Hogan (1996)	19	Numbers	✓
Lowe (1998; Exp. 2,3)	40	Words	✓
(1998; Exp. 1)	40	Words	✗
Milliken et al. (1998; Exp. 1a, 2a-c, 4)	20, 24, 20	Words	✓
(1998; Exp. 1b, 3)	20, 20	Words	✗
Bestgen & Dupont (2000; Exp. 1, 2)	36, 150	Stroop colour words & letters	✓
Koshino et al. (2000)	40	Letters	✓
MacLeod, Chiappe & Fox (2002)	42	Words	✓
Gibbons et al. (2006)	36	Numbers	✗
Troche et al. (2008)	74	Numbers	✓
Chao (2009; Exp. 1, 2a-c, 3)	24, 24, 36	Stroop colour words	✓
Groh-Bordin & Frings (2009)	44	Letters	✗

Note. NP = negative priming; ✓ = study reported negative priming effects; ✗ = study failed to report negative priming effects.

Experimental Design: Findings from several studies suggest that a distractor stimulus is required on probe trials in order to produce negative priming effects (Moore, 1994; Tipper & Cranston, 1985). That is, negative priming is said to depend on high-conflict probes, in which distractor stimuli interfere directly with the correct response (Moore, 1994; Tipper & Cranston, 1985). After failing to find negative priming effects on their letter-naming task when no probe distractor was present, Tipper and Cranston (1985) concluded that a selection state is not

maintained when the probe display contains no conflicting information (e.g., single probes or distractor probes are easy to distinguish from target), and a response can be output automatically (thus no inhibition needs to be overcome and no negative priming is observed). Consistent with this, Moore (1994) conducted a series of experiments using a letter identification task. In high conflict trials the target overlapped the distractor letter associated with an incompatible response, whereas the low-conflict trials consisted of (a) distractors not associated with a response, (b) # sign, (c) random dots, or (d) no distractor at all. Negative priming was found to occur on high conflict probes and only on low conflict probes when they were intermixed with high conflict probes. However, while some studies have failed to find negative priming in the absence of a probe distractor (Allport, Tipper, & Chmiel, 1985; Groh-Bordin & Frings, 2009; Milliken & Joordens, 1996; Moore, 1994; Tipper & Cranston, 1985), other studies have reported negative priming effects (Bannon et al., 2008; Milliken et al., 1998, Experiment 4; Neill, Terry, & Valdes, 1994).

In summary, it seems the absence of distractor stimuli in the probe trial fails to produce negative priming effects in some experiments but not others. It is possible that these inconsistent results are due to other methodological differences between studies. That is, these studies differed from each other on many experimental design factors such as whether or not a prime distractor was presented and the task for the prime trial (i.e., whether it was responded to at the time of selection or after the probe, or not at all). Given the number of other methodological differences between these studies the underlying reasons for the discrepant negative priming findings regarding the presentation of a simultaneous probe distractor remains elusive.

Findings from two studies suggest that a small set of stimuli may be needed to produce negative priming effects (Chiappe & MacLeod, 1995; Malley & Strayer, 1995). Malley and

Strayer (1995) showed negative priming occurred only for small sets of stimuli (a dozen or less) and failed to be observed in larger sets. Similarly, Chiappe, and MacLeod (1995) used a smaller stimuli set (10 items) and found negative priming effects, both when participants were required to name items and categorise them. In contrast, MacLeod, Bibi, and Stamenova (as cited in MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003) used 20 items (5 categories) and failed to find negative priming effects in either naming or categorisation tasks. This may be because when using a smaller number of stimuli, they are likely to be repeated a greater number of times throughout the task. That is, Malley and Strayer (1995) found negative priming effects with repeated stimuli, but not with novel stimuli. They suggested that when stimuli are repeated they will obtain higher levels of activation, and create negative priming effects when they compete for a response in probe trials. However, limited research has been conducted regarding this experimental factor.

Lowe (1998) suggested that increased contextual similarity between prime and probe trials may also influence the likelihood of negative priming effects. Using a word naming task Lowe (1998) manipulated the degree of familiarity between prime and probe items. Negative priming was observed when distractors became targets in a familiar context (i.e., presented in the same word pair in which they were initially ignored). However, positive priming was found when distractors become targets in an unfamiliar context (i.e., in a new word pair). Lowe (1998) suggested that such negative priming effects are sensitive to contextual differences between prime and probe trials.

Temporal manipulation: The temporal interval between the presentation of prime and probe stimuli has also been shown to influence negative priming effects (Neill & Valdes, 1992; Neill & Westberry, 1987). That is, the temporal interval between prime and probe stimuli has

been manipulated in various experiments to examine the temporal course of processes underlying negative priming (Neill & Valdes, 1992; Neill & Westberry, 1987; Tipper et al., 1991). Neill and Westberry (1987) initially investigated this issue with a Stroop task using response-stimulus intervals (RSI) of 20, 520, 1020, and 2020ms. They found negative priming effects at all intervals except 2020ms. Neill and Westberry concluded that negative priming decays with time, and disappears after 2 seconds. In contrast, Tipper and colleagues found no change in negative priming effects across three RSIs (1350ms, 3100ms, and 6600ms) in a picture naming task. Neill and Valdes (1992) hypothesised that Tipper and colleagues may have found RSI effects if they had also included shorter intervals, as decay effects at longer intervals may be too small to detect. To explore this further Neill and Valdes (1992) conducted a letter matching task across five randomly presented RSIs (500ms, 1000ms, 2000ms, 4000ms, and 8000ms). They found negative priming effects decreased across RSIs from 70ms at an RSI of 500ms to 8ms at an RSI of 8000ms, with the greatest amount of change occurring between RSIs of 500ms and 1000ms. However, Hasher, Stoltzfus, Zacks, and Rympa (1991) found no difference between negative priming effects using a letter naming task at RSIs of 500ms and 1200ms.

In summary, the results appear inconsistent, with some studies finding a decay in negative priming over time whereas others do not. Neill and Valdes (1992) suggested that this discrepancy in findings may be due to methodological differences between the studies (i.e., between versus within subjects design). It is evident that further research is required to determine whether negative priming effects decay over time, and Study 1 aims to examine this issue further. Given that Neill and Valdes (1992) found the greatest amount of decay in negative priming occurred between 500ms and 1000ms, Study 1 will examine the temporal course of inhibition within this temporal interval. Information regarding the temporal course of inhibition

also has important implications for research on inhibition in clinical populations, such as OCD. That is, it is important to clarify whether differences between OCD and control participants negative priming effects are the result of differences in the strength of their active suppression processes and/or differences in the temporal course of their active suppression. Study 2 aims to examine this issue further.

Given such inconsistent results throughout the negative priming literature, it is not surprising that the reliability and validity of negative priming as a measure of inhibition has been questioned. To examine reliability Bestgen and Dupont (2000) conducted a spilt-half correlation analysis on two identity negative priming tasks. Both studies failed to find sufficient levels of reliability (r_s = 0.00), leading the authors to conclude that the negative priming paradigm is not a reliable measure of inhibition. In addition, Troche and colleagues (2008) examined the test-retest reliability of a number identification task in both young and older adults. The results revealed low test-retest coefficients for negative priming in both young (r = -0.12) and older adults (r = 0.45), leading the authors to speculate that negative priming effects are a state-dependent phenomenon rather than a trait marker for inhibition.

Theoretical Accounts of Negative Priming: The mechanisms that underlie negative priming effects are in dispute with several theories proposing that retrieval mechanisms account for negative priming effects instead of distractor suppression. The two most prominent retrieval theories (*episodic retrieval* and *feature mismatch*) will be briefly described.

The **episodic retrieval account** (Neill & Valdes, 1992; Neill, Valdes, Terry, & Gorfein, 1992) holds that negative priming effects are the result of retrieval of information from the prime trial which conflicts with the correct response in the probe trial. According to this account the distractor stimulus receives a "do not respond" tag on the prime trial. When this stimulus is re-

presented in the probe trial the participant is said to retrieve from memory its previous processing episode (prime trial) in which it was ignored. The previous "do not respond" tag conflicts with the "respond" tag that is required for the same stimulus on the probe trial because it is now the target. The delay in responding found in negative priming tasks is said to reflect the time needed to resolve this conflict between the "do not respond" and "respond" tags attached to the same stimulus in different processing episodes (Neill & Valdes, 1992; Neill et al., 1992).

While the episodic retrieval account attributes negative priming to response conflict, the **feature mismatch account** (Park & Kanwisher, 1994) posits that such effects are the result of stimulus feature conflict. To illustrate, on a typical negative priming task prime and probe trials have two words (TABLE, GRASS), each presented in different colours (red and green) with the participant responding to the word presented in red in both trials. In a negative priming trial the distractor word 'grass' is presented in green in the prime trial, and then re-presented as the target in red in the probe trial. This mismatch in the colour of the word 'grass' from prime to probe trials is an example of a stimulus feature conflict that is said to account for a delay in response time. Although there is some empirical support for these retrieval based theories of negative priming (MacLeod et al., 2003), Tipper (2001) argued "there is no firm evidence to discount inhibition models" of negative priming (p. 321). Furthermore, it is evident that no one theory can completely explain the inconsistent results throughout the negative priming literature (Fox, 1995).

An additional criticism of the traditional priming paradigm is that it actually confounds constructs of inhibition and facilitation. That is, because target and distractor stimuli are presented simultaneously in prime and probe trials, both facilitation and inhibition processes are theoretically active in each trial. Specifically, positive priming trials which are said to measure

facilitation processes, theoretically also require inhibition processes and the reverse can be argued for negative priming trials. Consequently, these processes cannot be measured independently using a traditional priming task. This thesis aims to tease facilitation and inhibition effects apart and measure them separately in a modified priming task.

2.3.2 Go/Nogo Paradigm

2.3.2.1. Go/Nogo Paradigm and Inhibition

Go/Nogo tasks are generally credited as being robust measures of response inhibition, and are used extensively within neuropsychological studies (Simmonds et al., 2008). A traditional Go/Nogo task employs a frequent Go and infrequent Nogo stimulus. Participants are required to respond quickly to Go stimuli, typically with a button press, and withhold a response to Nogo stimuli. One's ability to withhold a response to Nogo stimuli is used to measure response inhibition. That is, commission errors to Nogo stimuli are typically interpreted as reflecting a failure in response inhibition (Simmonds et al., 2008).

To illustrate, a traditional Go/Nogo task was used in a study examining inhibition processes in children with attention deficit hyperactivity disorder (ADHD) and typically developing children (Wodka et al., 2007). The children were required to press a button quickly to the frequently presented green spaceships, and withhold a response to the infrequent red spaceships. The percentage of commission errors was used as a reflection of response inhibition. The greater number of commission errors made by the ADHD group compared to controls was taken to indicate that children with ADHD have a primary deficit in response inhibition (Wodka et al., 2007). Similarly, many other studies have interpreted an increased number of commission errors in Go/Nogo tasks as a reflection of inhibitory deficits in various populations such as OCD

(Bannon, Gonsalvez, Croft, & Boyce, 2002), schizophrenia (Weisbrod, Kiefer, Marzinzik, & Spitzer, 2000), and ill Gulf War veterans (Tillman et al., 2010).

Many variations of the Go/Nogo task exist throughout the inhibition literature. Such modifications include the use of different Go/Nogo stimuli, the number of Go/Nogo stimuli, continuous versus static presentation of stimuli, timing of stimuli, proportion of Go versus Nogo stimuli, and whether Go/Nogo stimuli are based on context rule (Langenecker, Zubieta, Young, Akil, & Nielson, 2007). Because of the many variations of Go/Nogo tasks, Langenecker et al. (2007) criticised the Go/Nogo paradigm for its lack of standardisation and validation. To counter this, they examined the convergent validity and reliability of three types of Go/Nogo tasks: (1) Three Go stimuli only; (2) Two context based Go stimuli, in which participants respond to 'x' and 'y' only when they appear in alternation; and (3) Three context based Go stimuli, in which participants respond to 'x', 'y' and 'z' only if they occur in a nonrepeating order (e.g., double 'x' would be a Nogo stimulus on the second presentation). Langenecker and colleagues (2007) argue that context based inhibition provides a more specific measure of inhibitory control, whereby a previous response can result in "real time" changes in target or distractor sets. The authors found the Go/Nogo tasks demonstrated strong test-retest reliability and modest convergent validity with other executive functioning tests. In addition to criticisms about its lack of standardisation and validation, the Go/Nogo paradigm has also recently been criticised for confounding facilitation and inhibition processes (Bannon et al., 2008; Thomas, Gonsalvez, & Johnstone, 2009). This critique is discussed further below.

2.3.2.2. Go/Nogo Paradigm and Facilitation

The frequent repetition of Go stimuli in the Go/Nogo paradigm also provides the opportunity to measure facilitation processes. Yet surprisingly only one study examined facilitation processes using a Go/Nogo task, finding facilitation effects were produced to initial Go stimulus repetitions (Thomas et al., 2009). To elaborate, using a modified Go/Nogo task Thomas and colleagues (2009) examined facilitation processes using stimulus repetitions within stimuli trains. That is, stimuli were presented in a sequence with Go stimulus repetitions occurring between 1-4 times, followed by the presentation of a Nogo stimulus (e.g., GNG, GGNG). Response times to Go stimulus repetitions were found to decrease initially (e.g., G, GG), but increase with latter repetitions (e.g., GGG, GGGG; Thomas et al., 2009). This reversal in facilitation effects at latter repetitions was posited as being caused by participants' anticipation of a change in stimulus at latter Go stimulus repetitions.

In addition to reductions in response time, commission errors may be another measure of facilitation processes in the Go/Nogo task. That is, although they have typically been interpreted as a failure of response inhibition, it is possible commission errors are actually a reflection of facilitation instead of inhibition. The more frequent occurrences of Go stimuli compared to Nogo stimuli may prime Go stimuli and lead to a bias to respond, making commission errors more likely. If this is the case, then commission errors could be argued to be a reflection of the strength of facilitation processes (overactivation of the Go-stimulus) rather than a measure of the failure of inhibition processes (weak suppression of the Nogo stimulus).

Two studies that have examined the impact of sequence effects on commission errors have reported conflicting results (Durston, Thomas, Worden, Yang, & Casey, 2002; Thomas et al., 2009). In an fMRI study Nogo stimuli were preceded by one, three or five repetitions of Go

stimuli. Durston and colleagues (2002) found that more commission errors were made to the Nogo stimuli preceded by a greater number of Go stimuli (Durston et al., 2002). In contrast, Thomas and colleagues (2009) found a linear reduction in commission errors as the number of Go stimuli preceding the Nogo stimulus increased. Such contradictory findings may have been due to methodological differences between the studies such as sample differences (e.g., Thomas and colleagues used psychology students who may have been familiar with Go/Nogo task, whereas Durston and colleagues used adults from the general population) or differences in task stimuli (e.g., Durston and colleagues employed Pokemon characters which may have made the task more difficult compared to the ✓ and × stimuli utilised by Thomas and colleagues). Hence, it is evident that further investigation regarding sequence effects on commission errors is needed to help provide some insight about whether they reflect facilitation or inhibition effects.

In summary, although the Go/Nogo task is a prominent measure of inhibition, it has recently been criticised for confounding inhibition and facilitation effects (Bannon et al., 2008; Thomas et al., 2009). Specifically, although commission errors have typically been interpreted as a reflection of inhibition failure, they may actually reflect facilitation effects given the high frequency of Go stimuli. This thesis aimed to further examine sequence effects on commission errors to determine whether they reflect inhibition or facilitation.

2.4 Summary

Within the identity priming literature facilitation effects appear to be a consistent and reliable finding across tasks and within subjects (e.g., Salo et al., 2002; Troche et al., 2008; Waechter et al., 2010). Findings in the negative priming literature are not as consistent, with the presence or absence of the negative priming effect seeming to depend on the design of the

experiment. There is limited literature on the temporal determinants of facilitation and inhibition, which is surprising given that such processes may be time-bound. In regards to facilitation, priming studies suggest positive priming effects are a consistent finding from 50ms to 2000ms temporal intervals (Salo et al., 2002; Waetcher et al., 2010). In contrast, negative priming studies are less consistent with some indicating this effect is stable over time (e.g., Tipper et al., 1991), whereas others suggest it rapidly decays (e.g., Neill & Valdes, 1992). Such findings suggest that facilitation may be the more stable trait-like mechanism of selective attention, whereas inhibition may be a state-dependent mechanism that operates when required.

Several traditional priming tasks (e.g., Go-Nogo) used to investigate facilitation and inhibition can be criticised for confounding facilitation and inhibition. Study 1 in this thesis aims to address some of these problems by designing modified tasks. Details of Study 1 are described in Chapter 4. The next chapter reviews selective attention in OCD.

CHAPTER 3

SELECTIVE ATTENTION ABNORMALITIES IN OBSESSIVE - COMPULSIVE DISORDER

3.1 Aim

The aim of this chapter is to review research examining inhibition and facilitation processes in OCD. Because people with generalized anxiety disorder (GAD) will be included as the anxious control group for Study 2, the current chapter will also briefly review the nature of GAD and research examining inhibition and facilitation in GAD.

3.2 General Selective Attention in OCD

As was outlined in Chapter 2, more recent models of selective attention assert that both facilitation of task-relevant stimuli and inhibition of task-irrelevant stimuli are needed for efficient goal-directed behaviour (Houghton & Tipper, 1994). It has been suggested that people with OCD may have a disturbance in these two processes (Amir & Kozak, 2002). That is, the recurring properties of OCD symptoms may indicate inhibition and/or facilitation abnormalities (Bannon et al., 2008).

3.2.1 Inhibition in OCD

The uncontrollability and recurrence of thoughts and behaviours experienced by people with OCD is suggestive of inhibitory deficits (Bannon et al., 2002; Bannon et al., 2008; Chamberlain et al., 2005; Penades et al., 2007; Rankins, Bradshaw, & Georgiou-Karistianis,

2006). Findings from various experimental paradigms support this contention (e.g., Amir, Cobb, & Morrison, 2008; Bannon et al., 2008; Enright & Beech, 1990; Georgiou-Karistianis, Howells, & Bradshaw, 2003; Moritz et al., 2004; Penades et al., 2007; Rankins et al., 2006). This chapter will focus on the experimental paradigms most commonly used to investigate inhibitory processes in OCD. Such paradigms include the Stroop task, Go/Nogo task, Stop signal task, and the negative priming task. Because these paradigms assess inhibition in different ways, findings from OCD studies using the aforementioned paradigms will each be discussed in turn below. Such results are summarised in Table 3.1. As is evident from Table 3.1, behavioural data from studies examining inhibition processes in OCD are inconsistent, with some reporting an inhibitory deficit for OCD participants and others not.

Stroop Task: This inconsistent pattern of results is present in studies utilising the Stroop task. The interference condition of the Stroop task (Stroop, 1935) has been used to examine cognitive inhibition in OCD (e.g., Bannon et al., 2002; Penades et al., 2007). The interference condition of the Stroop task typically requires participants to name the colour ink of the presented word which is the name of another colour (e.g., say "red" when the word 'BLUE' is presented in red ink). Response times are typically longer in this condition, compared to naming the colour ink of non colour words, because participants are required to attend to one set of information (colour value) and inhibit the tendency to read the word (semantic value).

Table 3.1. *Summary of Findings from Studies Examining Inhibition Processes in OCD*

Experimental Paradigm	Study	*n* (OCD)	(NC)	(Other)	Deficit in OCD
Stroop	Martinot et al. (1990)	16	8		✓
	Hartston & Swerdlow (1999)	76	62		✓
	Bannon et al. (2002)	20		20 (PD)	✓
	Moritz et al. (2004)	35	20		✗
	Penades et al. (2005)	35	33		✓
	Penades et al. (2007)	27	25		✓
	Moritz et al. (2008)	23	23		✗
Go/Nogo	Bannon et al. (2002)	20		20 (PD)	✓
	Hermann et al. (2003)	12	12		✗
	Penades et al. (2007)	27	25		✓
	Kim et al. (2007)	15	15		✗
Stop	Johannes et al. (2001).	10	10		✗
	Krikorian et al. (2004)	7	10		✗
	Chamberlain et al. (2006)	20	20		✓
	Penades et al. (2007)	27	25		✓
	Menzies et al. (2007)	31	31		✓
Negative Priming	Enright & Beech (1990)	16		15 (OAD)	✓
	Enright & Beech (1993a)	36		60 (OAD)	✓
	Enright & Beech (1993b)	36		60 (OAD)	✓
	Enright et al. (1995) (pres.=100ms)	32		32 (OAD)	✓
	Enright et al. (1995) (pres.=250, 350ms)	32		32 (OAD)	✗
	Hartston & Swerdlow (1999)	76	62		✗
	MacDonald et al. (1999)	24	12		✗
	McNally et al. (2001)	26	19		✗
	Hoenig et al. (2002)	15	16		✗
	Amir et al. (2008) (neutral stimuli)	19	19		✗
	Bannon et al. (2008)	20	20	20 (PD)	✓
	Moritz et al. (2010)	18	28		✗

Note. NC = normal controls; PD = panic disorder controls; OAD = other anxiety disorder controls; pres. = stimulus presentation; ✓ = study reported significant deficits; ✗ = study failed to report any significant deficits between OCD and control participants.

From Table 3.1 it is evident that five studies employing the Stroop paradigm reported higher interference costs for OCD patients (Bannon et al., 2002; Hartston & Swerdlow, 1999; Martinot et al., 1990; Penades, Catalan, Andres, Salamero, & Gasto, 2005; Penades et al., 2007). For example, in a vocal naming Stroop paradigm, OCD participants completed significantly fewer items on the Stroop interference condition compared to normal controls (Hartston & Swerdlow, 1999; Penades et al., 2005). Similarly, OCD participants showed increased response times and increased error rates on Stroop interference trials compared to normal controls in vocal naming and motor Stroop tasks (Martinot et al., 1990; Penades et al., 2007). However, in the absence of an anxious control group, it is difficult for these studies to determine whether these deficits are specific to OCD or anxiety disorders in general (Kuelz, Hohagen, & Voderholzer, 2004). Accordingly, Bannon et al. (2002) employed the Stroop task to measure cognitive inhibition in OCD and panic disorder patients. The authors found that OCD participants made significantly more errors and produced longer reaction times on the Stroop interference condition compared to panic disorder participants.

In contrast to these findings, Moritz and colleagues (2004, 2008) failed to find a difference between OCD and healthy controls on Stroop interference trials (Moritz et al., 2004; Moritz et al., 2008). The lack of difference between OCD and normal controls in these studies may have been due to the methodological design of the Stroop task used. That is, unlike studies of Bannon et al. (2008), Penades et al. (2005) and Hartston & Swerdlow (1999), Moritz and colleagues (2004, 2008) used a larger number of conditions, and presented these conditions randomly instead of blockwise. As no differences were found between OCD and controls, Moritz and colleagues (2004, 2008) suggested a blockwise administration may be a more powerful method for eliciting Stroop effects.

In summary, although there are some inconsistent results, Stroop task studies suggest that individuals with OCD demonstrate performance deficits on the interference condition of the Stroop task compared to normal and anxious controls. Such findings have been interpreted as evidence for the presence of inhibitory deficits in OCD (Bannon et al., 2002; Hartston & Swerdlow, 1999; Penades et al., 2007).

Go/Nogo Task: Results from behavioural data in studies using the Go/Nogo task are equivocal regarding whether or not OCD participants display an inhibitory deficit. The Go/Nogo task has been used as a measure of behavioural response inhibition in people with OCD (Bannon et al., 2002; Penades et al., 2007). In the Go/Nogo task participants are required to respond quickly to Go stimuli, typically with a button press, and withhold a response to Nogo stimuli. An individual's ability to withhold a response to Nogo stimuli is used to measure response inhibition. That is, commission errors to Nogo stimuli are typically interpreted as reflecting a failure in response inhibition (Simmonds et al., 2008).

Table 3.1 reveals two studies using the Go/Nogo task have reported OCD patients exhibit performance deficits compared to controls (Bannon et al., 2002; Penades et al., 2007). Using a Go: Nogo stimuli ratio of 75:25% Bannon and colleagues (2002) found that OCD participants produced faster responses to Go stimuli and made more commission errors to Nogo stimuli compared to panic disorder patients. A faster response time to Go stimuli is not consistent with an inhibition hypothesis, but instead may reflect stronger facilitation in OCD. This will be explored in Study 2. Penades et al. (2007) also reported a smaller percentage of successful inhibition for OCD participants compared to normal controls using a Go: Nogo stimuli ratio of 73:27%. In contrast, Kim and colleagues (2007) failed to find a difference between the error rates of OCD and normal control groups using a Go/Nogo ratio of 50:50% (Kim, Kim, Yoo, &

Kwon, 2007). Although OCD participant's response times were significantly quicker to Go stimuli, Herrmann and colleagues (2003) failed to find a significant difference between the error rates of OCD and normal control groups. Interestingly, they used an unusually low Go: Nogo ratio of 10:90% (Herrmann, Jacob, Unterecker, & Fallgatter, 2003). In explaining these discrepant results, it is evident that OCD participants perform significantly worse in Go/Nogo studies employing a higher percentage of Go stimuli relative to Nogo stimuli (Bannon et al., 2002; Penades et al., 2007). Hence, it may be the case that the lower frequency of Go stimuli in the latter two studies was not enough to detect a difference between the groups' response inhibition abilities.

The increased number of commission errors on Go/Nogo tasks has typically been interpreted as evidence of an inhibitory deficit in OCD (Bannon et al., 2002; Penades et al., 2007). However, as discussed in Chapters 2 and 3, the frequent presentation of Go stimuli in Go/Nogo tasks may lead to a bias to respond, making commission errors more likely. Hence, commission errors may reflect facilitation effects. Study 2 will examine whether enhanced facilitation effects in OCD are reflected in an increased commission error rate.

Stop Task: Differences in the dependent variable used to measure inhibition may account for the inconsistent results found for OCD participants in studies using the Stop task. The Stop task has been administered to assess participants' ability to inhibit a prepotent motor response (Logan, Cowan, & Davis, 1984). In the Stop task participants are required to press a button to 'Go' stimuli and attempt to inhibit a response to 'Go' stimuli when a 'Stop' signal is presented after the presentation of a 'Go' stimulus. For example, Menzies et al. (2007) required participants to press one of two buttons to indicate whether the arrows presented (Go stimuli) were pointing left or right. In 25% of the trials presented, an audible stop-signal was presented

after the presentation of the arrows and participants were directed to attempt to inhibit their motor response to the arrows. The dependent variable Menzies and colleagues (2007) used was the stop-signal reaction time (SSRT), which is the processing time required by each participant to inhibit a prepotent response. They found that patients with OCD and their unaffected first-degree relatives had a significantly longer SSRT than healthy controls.

Consistent with this finding, two additional studies utilised the SSRT as the dependent measure in the Stop task and reported OCD participants produced significantly longer SSRT than healthy controls (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Penades et al., 2007). These studies suggest that a response to Go stimuli may be more highly primed (or activated) in those with OCD, which means they need a shorter interval between the presentation of the Go stimuli and Stop signal to be successful at inhibiting their response.

In contrast to these findings, Table 3.1 reveals that two studies failed to report a deficit for OCD participants on the Stop task compared to normal controls (Johannes et al., 2001; Krikorian, Zimmerman, & Fleck, 2004). These studies did not use the SSRT as the dependent measure, but instead compared groups on number of correct hits, commission and omission errors, and general reaction time. Thus, the comparable performance of OCD participants to controls on the Stop task in these two studies might be due to differences in dependent measures rather than a genuine absence of difference between groups. Overall, findings from studies using the Stop task suggest people with OCD need a shorter time interval between Go stimuli and the Stop signal to be successful at inhibiting a response to the primed Go stimuli compared to normal controls.

Negative Priming Task: Like the previous three paradigms, the negative priming paradigm has also produced inconsistent findings throughout the OCD literature. In the

traditional negative priming task, target and distractor stimuli are presented simultaneously in the initial prime and subsequent probe trial. Participants are instructed to respond to target stimulus (e.g., name the red letter) and ignore the distractor stimulus in the prime and probe (Tipper, 1985). A slower response time to a target in the probe trial that was previously a distractor in the prime trial is called negative priming (Tipper, 1985). This effect has been argued to reflect inhibition processes, as the internal representation of the distractor stimulus is actively suppressed in the prime trial and this suppression lingers, producing a slowed response time in the probe trial (Houghton & Tipper, 1994; Tipper, 1985; Tipper et al., 1991). While other theories regarding the mechanism underlying negative priming have been proposed, such as episodic retrieval (Neill & Valdes, 1992; Neill et al., 1992), distractor suppression is the preferred theory to explain negative priming in the OCD literature (Amir et al., 2008).

Studies examining inhibition processes in OCD via the negative priming paradigm have employed a range of negative priming tasks. Such studies have varied in task parameters including priming type (spatial versus identity priming), stimuli used (letters, digits, words and Stroop words), duration of stimulus presentations or time between prime and probe trials, and whether or not a probe stimulus is simultaneously presented (e.g., Bannon et al., 2008; Enright, Beech, & Claridge, 1995; Hoenig, Hochrein, Muller, & Wagner, 2002). In regards to priming type, only two studies have examined spatial negative priming in OCD (Hartston & Swerdlow, 1999; Hoenig et al., 2002). In such studies participants were required to press one of four buttons corresponding to the location of the target stimuli. Both Hartston and Swerdlow (1999) and Hoenig et al. (2002) found no differences between OCD participants and normal controls in regards to spatial negative priming.

Whilst a greater number of studies have examined identity negative priming in OCD, the mixed results have increased confusion regarding whether or not OCD participants display a negative priming deficit and what that actually means. Table 3.1 reveals that five studies have reported negative priming abnormalities in OCD, suggesting they have difficulty inhibiting distractor stimuli in the prime trial (Bannon et al., 2008; Enright & Beech, 1990; Enright & Beech, 1993a, 1993b). For example, a series of studies conducted by Enright and colleagues (Enright & Beech, 1990; Enright & Beech, 1993a, 1993b) compared negative priming in OCD participants to those with other anxiety disorders. The different stimuli used across their studies included single letters, words, and Stroop colour words. At stimulus presentations of 100ms, each study found OCD participants exhibited impaired negative priming effects relative to anxious controls (Enright & Beech, 1990; Enright & Beech, 1993a, 1993b; Enright et al., 1995). Similarly, Bannon and colleagues (2008) also used a stimulus presentation of 100ms and found reduced negative priming in symptomatic and remitted OCD participants compared to normal controls and panic disorder patients with neutral words.

In contrast to these findings, six studies employing stimulus presentation durations of longer than 100ms reported comparable negative priming effects for OCD patients and controls (Amir et al., 2008; Enright et al., 1995; Hoenig et al., 2002; MacDonald, MacLeod, Antony, & Swinson, 1999; McNally, Wilhelm, Buhlmann, & Shin, 2001; Moritz, Kloss, & Jelinek., 2010). In the aforementioned study conducted by Enright et al. (1995), three stimulus presentation times were used for Stroop words (100, 250 and 350ms). At stimulus durations of 250ms and 350ms no significant difference in negative priming effects were observed between OCD and anxious controls. McNally et al. (2001) also reported that the marginally significant difference between

OCD and controls found at a stimulus duration of 100ms disappeared at a stimulus duration of 500ms.

Furthermore, studies allowing priming stimuli to stay on screen until participants have responded failed to report differences between OCD and normal controls (Amir et al., 2008; MacDonald et al., 1999). Similarly, two studies using stimulus presentation durations of 150ms failed to find differences between OCD and normal controls (Hoenig et al., 2002; Moritz et al., 2010). These studies are also the only two that examined the effect of multiple temporal intervals between prime and probe trials on negative priming in OCD. After conducting a subgroup analyses Hoenig and colleagues (2002) reported OCD checkers showed negative priming impairments compared to non-checkers at an RSI of 500ms, but normal negative priming at an RSI of 2000ms. In contrast, OCD non-checkers displayed the opposite results. However, Moritz and colleagues (2010) did not find any interaction between group and RSI interval (400ms, 1000ms). Thus, while Hoenig et al.'s (2002) findings suggest the temporal course of negative priming may be different between OCD subtypes, Moritz et al.'s (2010) results suggest the temporal course of negative priming in OCD does not differ from normal controls. Further research is required to determine whether the temporal course of negative priming is different from normal and anxious controls.

Overall, preliminary findings indicate OCD participants do not display a deficit in spatial negative priming. Further, it appears that identity negative priming studies employing a stimulus presentation of 100ms find negative priming deficits in OCD participants, whereas a comparable negative priming effect is found in tasks using a stimulus presentation of greater than 100ms. Enright et al. (1995) speculated that this finding may be because people with OCD have reduced pre-attentive inhibition that is not evident a longer stimulus durations, as more attended strategies

of information processing mask the problem. However, MacDonald et al. (1999) criticised this explanation for being inconsistent with (i) the inhibition account of negative priming (as inhibition is argued to occur after selection, and not in the first 100ms), and (ii) the negative priming literature in general. Instead, they suggested individuals with OCD have visual attention deficits that disadvantage their performance at short (100ms), but not longer stimulus presentations (> 100ms). Future research is required to investigate the importance of this stimulus duration for OCD. Furthermore, given the methodological differences in negative priming tasks, researchers should also attempt to replicate studies to examine whether these results are reliable in the OCD population.

Summary: Given the heterogeneity of OCD samples and the methodological differences between studies investigating inhibition processes in OCD, such inconsistent results are unsurprising. Overall, converging evidence from the Stop paradigm indicates that people with OCD have impaired ability to inhibit a prepotent response compared to controls. Similarly, data from the Stroop paradigm suggests people with OCD exhibit deficits in cognitive inhibition compared to controls. Findings from the Go/Nogo paradigm are less clear. The research suggests that a high Go stimulus ratio is required for OCD participants to make significantly more commission errors than controls. As to whether this outcome is the result of impaired inhibition or enhanced facilitation processes is yet to be investigated. The negative priming effect also needs further examination in those with OCD. To date, only studies using short stimulus presentations (100ms) have reported inhibitory deficits for OCD participants. Reasons for this are still unclear.

3.2.2 Facilitation in OCD

Stronger facilitation processes may also contribute to the recurring properties of obsessions and compulsions in OCD (Bannon et al., 2008). That is, if stimuli or responses are more highly activated or primed in individuals with OCD, this may result in a continued focus on primed thoughts and behaviours. This continued focus may lead to the re-activation and repetition of such primed thoughts and behaviours (Bannon et al., 2008). For example, once an individual with OCD has completed a compulsion, this initial behaviour creates a prime for itself and its higher level of activation increases the likelihood that the individual will continue to focus on and subsequently repeat this behaviour. Because OCD patients only exhibit obsessions and compulsions to a narrow range of concerns, enhanced facilitation may be more of a generalised or trait-like vulnerability rather than specific vulnerability to developing OCD. That is, the interaction between this generalised vulnerability and other specific factors may result in the narrow range of obsessions and compulsions. Such specific factors may include cognitive threat schemas (Bannon et al., 2008) or thought suppression coping strategies.

However, in contrast to the plethora of empirical research examining inhibition processes in OCD, only two studies to date have examined facilitation processes. Both studies used the priming paradigm to measure facilitation processes (Bannon et al., 2008; Hartston & Swerdlow, 1999). As described previously, in a traditional priming task target and distractor stimuli are presented simultaneously in the initial prime trial and subsequent probe trial (Tipper, 1985). Trials in which the target stimulus is repeated in the prime and subsequent probe are called positive priming trials. Response times on positive priming trials are typically faster than control trials, in which no target or distractor stimuli are repeated in prime and probe trials (Tipper, 1985). In this paradigm facilitation is measured by subtracting the mean response time on

positive priming trials from the mean response time on control trials. Positive priming effects are used as a measure of facilitation because it is believed that the activation level of the target from the prime display carries over and facilitates activation of the same stimulus in the probe trial (Stadler & Hogan, 1996; for further detail see Chapter 2).

Using a visuospatial priming paradigm, Hartston and Swerdlow (1999) examined facilitation processes in OCD. They found that compared to normal controls, people with OCD exhibited a larger facilitation differential. In addition, significantly elevated facilitation correlated with specific OCD subgroups including those who experienced violent images, a history of motor or vocal tics, a history of "just right" obsessions, or a history of checking compulsions (Hartston & Swerdlow, 1999). Such findings led these authors to speculate that enhanced facilitation processes contribute to the repetitive and automatic quality of OCD symptoms. Specifically, the automatic and repetitive nature of obsessions may be the result of the obsession establishing a prime for itself and facilitating its own recurrence, like "a groove in the brain" (Hartston & Swerdlow, 1999, p. 454). A similar process was suggested to contribute to the automatic and self-facilitating nature of compulsions.

Evidence from thought suppression studies appears to support the hypothesis that stronger facilitation processes may contribute to the recurrence of obsessions. Janeck and Calamari (1999) investigated the frequency of an idiographic negative thought in three stages: (1) 5 minute monitoring period (i.e., think about anything you like), (2) 5 minute thought suppression period (i.e., try not to think about target thought), and (3) another 5 minute monitoring period. OCD participants reported a higher frequency of the target thought compared to normal controls irrespective of the condition, suggesting it was more highly activated (or primed) in those with OCD even in the initial monitoring period when no thought suppression

was attempted. That is, it seems that once a thought occurs in someone with OCD, the likelihood of it recurring may be increased because of its increased activation strength.

Attempts to suppress thoughts have been shown to create a paradoxical increase in the frequency of the thought being "suppressed" (Wegner, Schneider, Carter, & White, 1987). This effect also appears to be enhanced in OCD. For example, using the same design as Janeck and Calamari (1999), Tolin and colleagues (2002) conducted a thought suppression study with a neutral thought (Tolin, Abramowitz, Przeworski, & Foa, 2002). They found the OCD group reported more target thoughts in all conditions compared to social anxiety and normal control groups, once again suggesting that thoughts are more highly activated in individuals with OCD even when thought suppression is not attempted. In addition, the OCD group also reported significantly more target thoughts during the thought suppression condition compared to the baseline monitoring condition, unlike the anxious and normal control groups (Tolin et al., 2002).

Based on these findings it is conceivable that thoughts are more highly activated or primed in people with OCD, leading to hyperaccessibility of the thought and a greater likelihood of it intruding into consciousness (Tolin et al., 2002). If pre-existing cognitive schemas bring anxiety provoking thoughts to attention, abnormally strong facilitation mechanisms could underlie the recurrence of such thoughts. If thought suppression is a way that people with OCD try to cope with the recurrence of such personally distressing thoughts, it makes sense that such thoughts are experienced with higher frequency and intensity. Hence, as obsessions have their origin in normal intrusive cognitions (Rachman & de Silva, 1978) it may be that the combination of stronger facilitation and subsequent thought suppression attempts that increases one's vulnerability to developing OCD.

A study conducted by Bannon and colleagues (2008) also found evidence for stronger facilitation processes in OCD. They developed a modified identity priming task to examine facilitation and inhibition processes independently in OCD. In this task neutral and idiographic threat words were presented individually and sequentially (see Chapter 4). Both symptomatic and remitted OCD groups showed enhanced facilitation to the repetition of target words compared to normal controls and panic disorder patients. As this effect was evident in both remitted and symptomatic OCD groups Bannon et al. (2008) suggested it may be a trait (as opposed to state) marker of the disorder. Bannon and colleagues (2008) also found impaired negative priming effects in OCD compared to controls. This lead the authors to propose that the combination of abnormal inhibition and facilitation processes make the recurrence of OCD symptomatology more likely because stimuli are both strongly encoded and cannot be inhibited as effectively. Bannon and colleagues (2008) offer an ink-and-eraser analogy to further explain these facilitation and inhibition abnormalities in OCD. Specifically, they relate enhanced facilitation effects to the strength and permanence of the ink used to encode information, and inhibition effects as the erasing fluid used to erase information that needs suppressing. Hence, people with OCD are posited as having stronger ink and weaker erasing fluid. Possessing the combination of these abnormalities may further increase one's vulnerability to developing OCD (Bannon et al., 2008).

In summary, preliminary findings from studies using the priming paradigm suggest stronger facilitation processes in those with OCD. Evidence from thought suppression studies also indicate that thoughts may be more highly activated in OCD, and when combined with thought suppression a paradoxical increase in thought frequency results. However, more research

on facilitation processes in OCD is needed in order to further our understanding of the potential underlying causes of OCD.

3.3 The Nature of GAD

3.3.1 Clinical Features of GAD

GAD as an anxiety disorder characterised by excessive and persistent anxiety or worry about multiple "understandable" domains or activities (e.g., work performance, finances, family and health). This worry is experienced as difficult to control and is associated with physiological symptoms such as irritability, fatigue, restlessness, muscle tension, and difficulty concentrating or sleeping (American Psychiatric Association, 2000).

3.3.2 GAD Prevalence and Course

An ECA study conducted in the United States found 12-month and lifetime prevalence rates for GAD across three sites ranged from 2.0%-3.6% and 4.1%-6.6%, respectively (Blazer, Hughes, George, Swartz, & Boyer, 1991). Similar prevalence rates were reported in Taiwan with 12-month and lifetime prevalence rates of 3.4% and 3.7%, respectively (Hwu, Yeh, & Chang, 1989). Consistent with these findings, an Australian study also reported a twelve-month prevalence rate of 3.6% (Hunt, Issakidis, & Andrews, 2002). In regards to gender, GAD tends to occur with double the frequency in women than men (Blazer et al., 1991; Carter, Wittchen, Pfister, & Kessler, 2001; Hunt et al., 2002; Wittchen, Zhao, Kessler, & Eaton, 1994).

Retrospective reports from GAD patients indicate that the average age of onset of GAD is between adolescence and late 20s (Barlow, Blanchard, Vermilyea, Vermilyea, & DiNardo, 1986; Rogers et al., 1999). The onset of GAD tends to be gradual, with patients often reporting they

have been anxious and nervous throughout their life (Rapee, 1991). Furthermore, like OCD, the course of GAD is reported to be chronic and fluctuating, with an exacerbation in symptoms evident during times of stress (Blazer et al., 1991; Brown, Barlow, & Liebowitz, 1994). These findings from retrospective reports are consistent with those from prospective studies. A 5 year follow-up study conducted with 167 patients involved in the Harvard/Brown Anxiety Research Program (HARP) found only 38% of patients experienced full remission of symptoms for 2 months or longer at any time over 5 years (Yonkers, Dyck, Warshaw, & Keller, 2000). They also found among those who achieved full remission, 27% relapsed to meet full criteria for GAD again during the three years of follow-up. These findings led Yonkers and colleagues (2000) to suggest that the course of GAD may best be described as chronic and relapsing.

3.3.3 Comorbidity

Like other anxiety disorders, it is common for people with GAD to have a comorbid Axis I disorder (Carter et al., 2001; Wittchen et al., 1994). An epidemiological study found that 93% of patients with GAD met criteria for an additional Axis I disorder over a 12 month period. Specifically, 59% met criteria for major depressive disorder and 56% fulfilled criteria for another anxiety disorder, such as specific phobia (30%), social phobia (29%), and panic disorder (22%; Carter et al., 2001). Similarly, a national comorbidity study conducted in the United States found 90.4% of individuals with GAD had lifetime comorbidity with another Axis I disorder. Major depressive disorder was the most common (62%; Wittchen et al., 1994).

3.4 Similarities between OCD and GAD

Based on the clinical descriptions above it is apparent that some clinical features are common to both OCD and GAD. One of the main similarities is the perseverative thinking component of both obsessions and worry, which has lead some authors to query whether these constructs represent distinct cognitive phenomena or the same mental process (Turner, Beidel, & Stanley, 1992). Subsequently, the similarities and differences between obsessions and worry have been investigated (Cormer, Kendall, Franklin, Hudson, & Pimentel, 2004; Turner et al., 1992), and are outlined below.

After examining obsessions in a clinical and non-clinical population, Rachman and de Silva (1978) discovered that obsessions are a common experience in a non-clinical population. They reported that the main differences between 'abnormal' and 'normal' obsessions were that abnormal obsessions were more frequent, more discomforting and intense, lasted longer, and were more strongly resisted and harder to dismiss. 'Normal" and 'abnormal' obsessions were also found to be similar in regards to the form (e.g., thoughts, images) and content (Rachman & de Silva, 1978). Like abnormal and normal obsessions, the content of worry in patients with GAD was found to be similar to the content of worry in a non-clinical population. However, people with GAD report greater difficulty controlling their worry and less success in resisting or reducing their worry (Craske, Rapee, Jackel, & Barlow, 1989).

Hence, it is evident that obsessions and worry share several characteristics; (i) both are present in clinical and non-clinical populations; (ii) both occur with greater frequency and less controllability in clinical populations; and (iii) both are associated with negative affect (Turner et al., 1992). However, the form and content of these cognitive phenomena help differentiate obsessions from worry (Cormer et al., 2004). That is, although the content of worry in GAD is

related to everyday matters, obsessions in OCD frequently include themes such as contamination or aggression which are experienced as ego-dystonic and are "not simply excessive worries about real-life problems" (American Psychiatric Association, 2000, p. 462). In addition, while they are both experienced as repetitive cognitive activity, obsessions are perceived as intrusive and may manifest in the form of thoughts, images or impulses, whereas worry generally occurs in the form of a thought. Furthermore, obsessions often trigger neutralising behaviour that is ritualistic, whereas worries in GAD do not (Cormer et al., 2004; Turner et al., 1992).

Given the similarities between OCD and GAD it is unsurprising that the co-occurrence rate of these two disorders is quite high, especially among individuals with OCD. Two studies have reported comorbidity rates of approximately 30% for adults with OCD who also met lifetime criteria for GAD (Andrews, Stewart, Morris-Yates, Holt, & Henderson, 1990; Crino & Andrews, 1996). Additionally, a clinical study found 20% of patients with OCD also met criteria for GAD (Abramowitz & Foa, 1998). Given the data on the proximity of OCD and GAD, Cormer and colleagues (2004) suggest that they may be neighbouring disorders in the context of anxiety disorders. Consequently, similar mechanisms may underlie the development and maintenance of these disorders. While there is a plethora of neurobiological studies suggesting neurobiological abnormalities may underlie clinical symptoms of OCD, few neuroimaging studies have been conducted with GAD patients (Britton & Rauch, 2009). Hence, while these disorders have clinical similarities, whether they share neurobiological similarities is yet to be determined.

3.5 Selective Attention in GAD

Given the similarities in the repetitive cognitive activity of OCD and GAD, it is important to investigate whether the hypothesised inhibition and facilitation abnormalities are specific to OCD, or also occur in GAD. The majority of research examining attention processes in GAD has focused on whether such individuals preferentially encode threatening information (MacLeod & Rutherford, 2004). This has been demonstrated in studies utilising the emotional Stroop paradigm. In this task participants are presented with threatening and neutral words and asked to name the ink colour the word is printed in while ignoring the semantic content. The selective encoding of threatening stimuli is revealed by slowed colour naming to threat words in comparison to neutral words, because people have more difficulty inhibiting the semantic content of threatening words (compared to neutral words) to instead name the colour ink the word is printed it. This effect has been demonstrated by GAD patients in a number of studies (e.g., Albu, 2008; Bradley, Mogg, Millar, & White, 1995; Mathews & MacLeod, 1985). These findings suggest that GAD patients have a selective attention bias for threatening information.

In contrast to the large amount of empirical research examining general inhibitory processes in OCD, relatively little research has been conducted on inhibition in GAD. Furthermore, research is yet to investigate facilitation in GAD. In regards to inhibition, no significant differences were found between GAD participants and normal controls on the Stroop colour-word interference effect (Albu, 2008; Price, & Mohlman, 2007). Similarly, Dorahy and colleagues (2006) failed to detect differences between the performance of GAD and normal control participants on a negative priming task (Dorahy, McCusker, Loewenstein, Colbert, & Mulholland, 2006). A small group of GAD patients (n =10) also participated in two of the negative priming studies conducted by Enright and colleagues (as part of the anxiety control

group). Compared to the OCD group, GAD participants showed significantly greater negative priming effects (i.e., less negative priming abnormalities) on negative priming tasks that utilised words as stimuli (Enright & Beech, 1993a, 1993b). Conversely, no significant differences were found between OCD and GAD participants on negative priming tasks employing overlapping letters (Enright & Beech, 1993a).

Collectively, these results suggest that people with GAD do not display a deficit in inhibition compared to OCD or normal controls. However, given the small number of participants in these studies and the limited research on general inhibition and facilitation processes in GAD, further research is required to explore whether inhibition and facilitation abnormalities are specific to OCD. Study 2 aims to address this issue.

3.6 Summary

Recent research suggests that abnormalities in facilitation and/or inhibition processes are frequently detected among persons with OCD. Researchers have proceeded to theorise that these deficits may underlie and contribute to clinical symptoms in general, but specifically the repetitive properties of obsessions and compulsions in OCD (Bannon et al., 2008). More specifically, findings from studies assessing facilitation in OCD indicate that people with OCD display stronger facilitation (Bannon et al., 2008; Hartston & Swerdlow, 1999). In addition, behavioural data from studies examining inhibition in OCD using the Stroop and Stop paradigms suggest people with OCD have inhibitory deficits (e.g., Bannon et al., 2002; Penades et al., 2007; Menzies et al., 2007). However, results from studies employing the Go/Nogo and negative priming paradigm to assess inhibition processes in OCD are less clear (e.g., Bannon et al., 2002; Enright et al., 1995; Kim et al., 2007; Moritz et al., 2010).

As to whether stronger facilitation and weaker inhibition is specific to OCD or whether these deficits could occur in other anxiety disorders in general or in some anxiety disorders (e.g., GAD) is yet to be conclusively determined. Table 3.1 highlights that to date only one study investigating inhibition and facilitation in OCD has included both healthy and anxious control groups. Because of similarities between OCD and GAD at a phenomenological level, any claim that these processes are specific to OCD must be backed up by comparisons with GAD groups. GAD patients will be included in Study 2 as an anxious control group. Limited research on facilitation and inhibition in GAD highlights the need for further research in this area.

3.7 The Present Study

An important research question arises from this literature review. Are facilitation and/or inhibition abnormalities present in and specific to OCD? That is, although recent research suggests enhanced facilitation and weaker inhibition may contribute to the repetitive nature of obsessions and compulsions in OCD (Bannon et al., 2008), few studies have examined both facilitation and inhibition in OCD. Hence, the primary aim of Study 2 was to address this gap in the literature and determine whether facilitation and inhibition abnormalities are present in OCD. However, because tasks measuring selective attention (e.g., Go/Nogo task) have been criticised for confounding facilitation and inhibition, before this thesis can examine selective attention in OCD using such paradigms, it firstly needs to establish selective attention paradigms that disentangle facilitation and inhibition. Hence, the primary aim of Study 1 was to address some of these problems by (i) designing tasks that were modified to disentangle facilitation and inhibition and (ii) evaluating which of these paradigms would provide the best information about facilitation and inhibition in OCD. The rationale, aims and hypotheses of Study 1 and 2 are presented in Chapters 4 and 5, respectively.

CHAPTER 4

STUDY 1: THE TEMPORAL COURSE OF FACILITATION AND INHIBITION IN NORMAL CONTROLS

4.1 Rationale and Aims of Study 1

Commonly used measures of selective attention (e.g., Go/Nogo task) have been criticised for confounding facilitation and inhibition (Bannon et al., 2008; Thomas et al., 2009). Given that facilitation and inhibition abnormalities may contribute to the recurrent nature of OCD symptoms (Bannon et al., 2008), it is important to have tasks that can measure these processes adequately. Hence, the primary aim of the current study was to modify priming and Go/Nogo paradigms, to try and disentangle facilitation and inhibition and measure them independently in normal controls. This series of experiments will determine which paradigms are the most appropriate to use when assessing facilitation and inhibition in OCD in Study 2.

The temporal course of facilitation and inhibition is also of paramount interest. Limited research has examined the temporal course of facilitation and conflicting findings have been reported regarding the temporal course of inhibition. Thus, the second aim of the present study was to capture the temporal course of these processes, to try and determine whether facilitation is stable across time and whether inhibition rapidly decays.

The third aim of the current study was to assess sequence effects on commission errors in the modified Go/Nogo task to determine whether commission errors are a reflection of inhibition failure or facilitation.

4.2 Experiment 1

As outlined in Chapter 2, the traditional priming task can be criticised for confounding constructs of facilitation and inhibition. That is, because target and distractor stimuli are presented simultaneously, both facilitation and inhibition processes are theoretically active in each trial. However, Bannon et al. (2008) developed a priming type paradigm to tease out these processes and measure them independently in participants with OCD. They modified the traditional priming paradigm by presenting target and distractor stimuli sequentially instead of simultaneously. In this task one word was presented at a time in prime and probe trials and participants were required to press the spacebar as quickly and accurately as possible each time the target word was presented on the screen. Bannon et al. (2008) found both facilitation and inhibition effects in the normal control (n = 20) and OCD (n = 20) groups using this sequential methodology. Given this is a newly developed paradigm, Bannon et al.'s results need to be corroborated. An added advantage of the sequential methodology, instead of simultaneous, is that it is a simpler task that can be used more easily with clinical populations.

Milliken and colleagues have also demonstrated that negative priming can be observed without requiring the overt selection of target against distractor stimulus in each trial (Milliken & Joordens, 1996; Milliken et al., 1998). In one of a series of experiments conducted by Milliken et al. (1998), 20 participants were shown a single prime word and a single probe word. They were instructed to either read or ignore the prime word, and then name the red probe word on each trial. In trials when the prime word was repeated as the probe word, the read group displayed positive priming and the ignore group displayed negative priming (Milliken et al. 1998). These results suggest that inhibition and facilitation processes can be measured without overt selection

against a distractor being required in each trial. The added benefit of the modified methodology is that it provides a way for these processes to be examined independently.

The current experiment aimed to replicate and extend Bannon et al.'s findings by attempting to capture these processes over time. That is, participants completed the modified priming task twice at time intervals of 600ms and 1000ms, as prior research suggests that the greatest amount of change in negative priming effects occurs between 500ms -1000ms (Neill & Valdes, 1992). Given Bannon et al.'s (2008) findings it was predicted that compared to control trials, participants would produce faster response times on positive priming trials (Hypothesis 1) and slower response times on negative priming trials (Hypothesis 2). This study also examined whether temporal determinants influence facilitation and inhibition.

4.2.1 Method

4.2.1.1 Participants. Fifteen undergraduate psychology students (13 females) aged between 18 and 57 years (M = 24.87, SD = 12.40) participated in Experiment 1 for partial course credit.

4.2.1.2 Task -Modified Priming Paradigm. This paradigm previously reported by Bannon et al. (2008) includes two independent tasks: facilitation and inhibition tasks. In this paradigm words were presented on a laptop computer using Java software. Three randomly selected neutral words from Bannon et al.'s (2008) previously trialed word list (see Appendix D) were presented in each condition. In each task, a trial consisted of the presentation of a cross (warning event) and subsequent presentation of a word at Time 1 and a word at Time 2. Stimuli were presented in black on a white background (see Figure 4.1). Participants completed two ISI conditions for each task: 600ms and 1000ms.

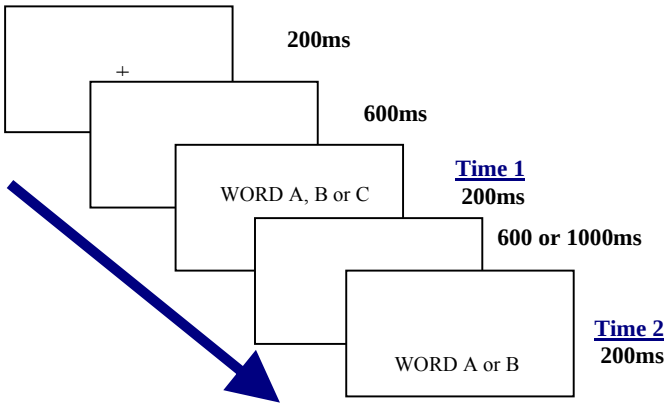


***Figure 4.1*. Schematic representation of the timeline of events for the modified priming paradigm.**

Facilitation Task: In the facilitation task Word A was randomly selected as the target and Words B and C subsequently became distractors. At Time 1 Word A or C was presented randomly with equal probability, whereas at Time 2 either Word A or B was presented randomly with equal probability. Participants were asked to press the mouse button as quickly and accurately as they could each time the target word (Word A) appeared. There are two key trials of interest in this task: (i) *control trials*: Word A was presented at Time 2 but not Time 1; (ii) *positive priming trials*: Word A was presented at both Time 1 and Time 2 (see Figure 4.2). The facilitation differential was calculated by subtracting the mean response time (RT) at Time 2 in the positive priming condition from the control condition. The task consisted of 80 trials plus a short practice block of 16 trials that participants were required to perform at ≥ 80% accuracy before progressing to the actual experiment. Trials resulting in incorrect or outlier responses (+/- 1.5 standard deviations; *SD*) were excluded before the mean RT was calculated.

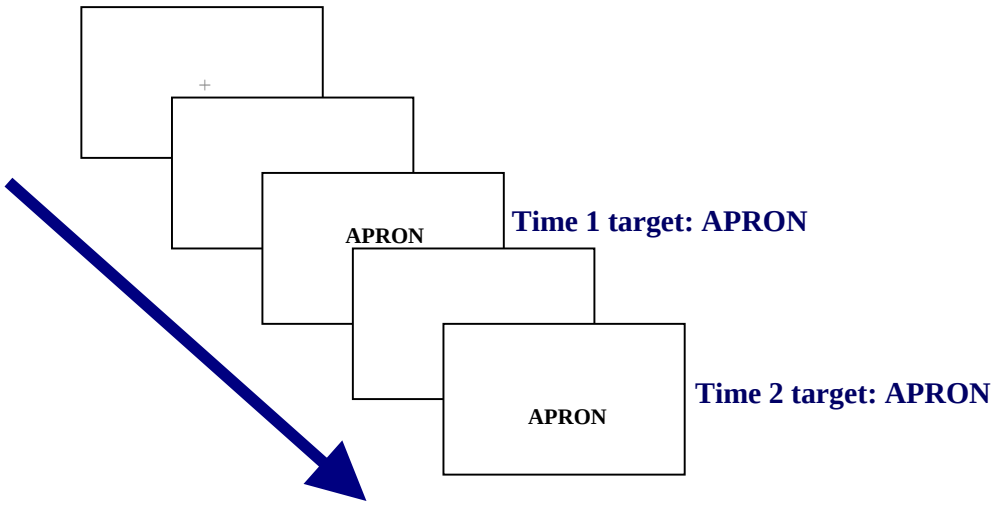


***Figure 4.2*. Example of a positive priming trial in the modified priming task.**

Inhibition Task: At Time 1 the probability of Word A being presented was 60%, compared to 20% for Words B and C. At Time 2 Words A and B were presented randomly with equal probability. Participants were asked to press the mouse button as quickly and accurately as they could each time the target word (Word A) appeared at Time 1 and Word B appeared at Time 2. There were two key trials of interest in the inhibition task: (i) *control trials*: Word B was presented as target at Time 2 and a distractor Word C was presented at Time 1; (ii) *negative priming condition*: Word B was presented as target at Time 2 but also as a distractor at Time 1 (see Figure 4.3). Hence, participants must overcome the previous response suppression to Word B at Time 1 to respond to Word B at Time 2. The inhibition differential was calculated by subtracting the mean RT at Time 2 in the negative priming condition from the control condition. The inhibition task consisted of 120 trials plus a short practice block of 24 trials that participants were required to pass (perform at ≥ 80% accuracy) before progressing to the actual experiment task. Trials resulting in incorrect or outlier responses (+/- 1.5 *SD*) were excluded before the mean RT was calculated.

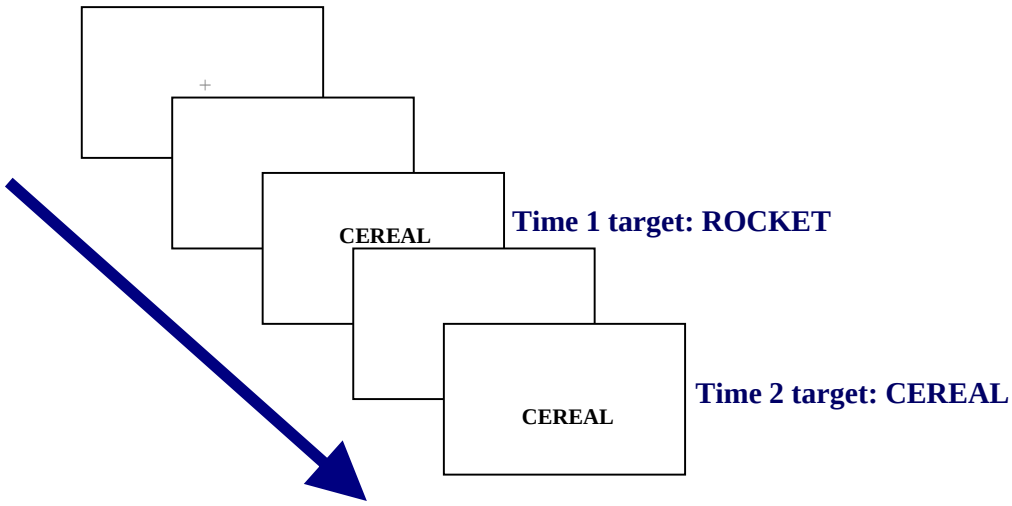


***Figure 4.3*. Example of a negative priming trial in the modified priming task.**

4.2.1.3 Procedure. The study was approved by the University of Wollongong Ethics committee. Written informed consent was obtained from each participant before commencement of the experiment. Participants were instructed to respond to the 'target' word by pressing the mouse button using the index finger of their dominant hand. Computer tasks took approximately 40 minutes to compete. Presentation of facilitation and inhibition tasks were counterbalanced and randomly assigned to control for order or fatigue effects.

4.2.2 Results

4.2.2.1 Facilitation. The mean response times, standard deviations, and facilitation differentials are presented in Table 4.1. Mean response times for control and positive priming conditions are plotted in Figure 4.4.

Table 4.1. *Mean Response Times (ms) for Facilitation Data Across Interstimulus Interval Conditions (ISI) in the Modified Priming Paradigm*

		Condition		
Task	**ISI (ms)**	**Control**	**PP**	**Facilitation**
Modified Priming	600	461.65 (52.04)	440.12 (45.13)	21.53 (21.80)
	1000	474.06 (62.02)	453.03 (47.79)	21.03 (24.64)

Note. Standard deviations in parentheses; ms = milliseconds; PP = positive priming; Facilitation = control RT – positive priming RT, higher positive values indicates more facilitation.

Paired *t*-tests were employed to examine mean differences between control and positive priming trials, and to determine whether the interstimulus interval condition (ISI; 600ms or 1000ms) had a significant effect on mean facilitation differentials. The positive priming trials produced a significantly faster mean RT compared to control trials at an ISI of 600ms, $t(14) = -3.83$, $p < 0.01$, and 1000ms, $t(14) = -3.31$, $p < 0.01$. There was no difference between the mean facilitation differentials at 600ms and 1000ms, $t(14) = 0.06$, $p = 0.95$. The percentage of response errors was < 2% for the facilitation task, at both ISIs, and was not submitted to further analysis.

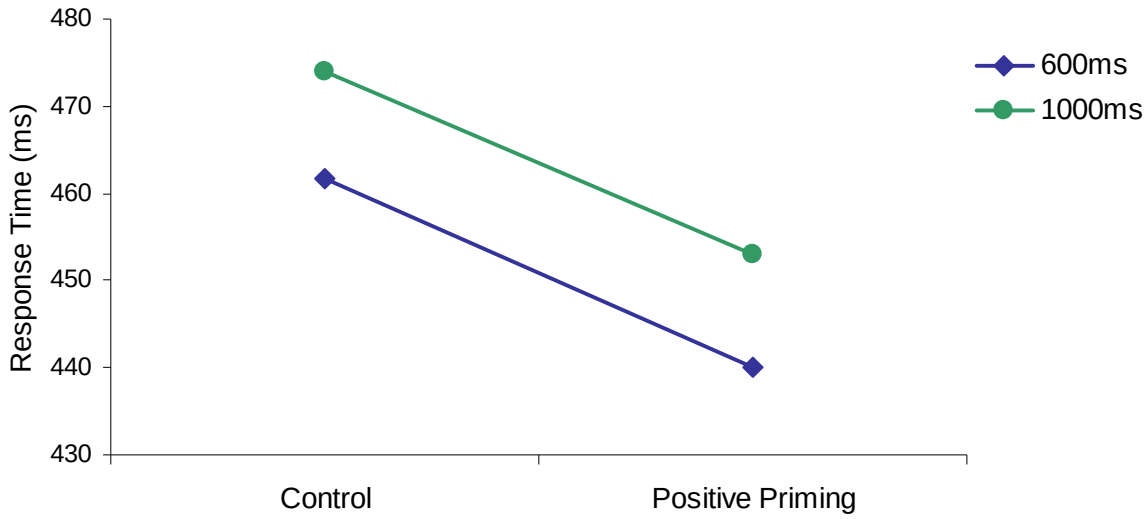


Figure 4.4. **Mean response times (milliseconds) on control and positive priming conditions of the facilitation task.**

4.2.2.2 Inhibition. The mean response times, standard deviations, and inhibition differentials are presented in Table 4.2. Paired *t*-tests were employed to examine mean differences between control and negative priming trials, and to determine whether the interstimulus interval condition (ISI; 600ms or 1000ms) had a significant effect on mean inhibition differentials. There was no significant difference between mean RTs for negative priming and control trials at an ISI of 600ms, $t(14)= -1.40$, $p = 0.18$ or 1000ms, $t(14)= 1.75$, $p = 0.10$. There was a significant difference between the mean inhibition differentials at 600ms and 1000ms, $t(14) = 2.68$, $p = 0.02$. The percentage of response errors was < 5% for the inhibition task, at both ISIs, and was not submitted to further analysis.

Table 4.2. *Mean Response Times (ms) for Inhibition Data Across Interstimulus Interval (ISI) Conditions in the Modified Priming Paradigm*

		Condition		
Task	**ISI (ms)**	**Control**	**NP**	**Inhibition**
Modified Priming	600	462.80 (52.58)	452.85 (48.64)	9.93 (27.56)
	1000	475.59 (57.44)	487.58 (49.02)	-11.96 (26.50)

Note. Standard deviations in parentheses; ms = milliseconds; NP = negative priming; Inhibition = control RT – negative priming RT (higher negative values indicates stronger inhibition, positive values indicate facilitation).

4.2.3 Discussion

The key difference between Bannon et al.'s (2008) paradigm and traditional priming paradigms was the sequential and singular presentation of target and distractor stimuli, instead of the simultaneous presentation of target and distractor stimuli. These tasks were designed to independently examine facilitation and inhibition processes and their time courses.

4.2.3.1 Facilitation. As predicted, target stimulus repetition produced positive priming effects for both ISIs (600ms and 1000ms). In addition, no difference was found between facilitation differentials for 600ms and 1000ms ISIs, suggesting stable and reliable facilitation

effects over this temporal course. These results replicate the results of Bannon et al.'s (2008) study that was restricted to an ISI of 1000ms. Such findings suggest this paradigm can be used to examine facilitation processes independently without inhibition processes confounding the results.

4.2.3.2 Inhibition. If the internal representation of the distractor stimulus is suppressed in this paradigm response times would be delayed when a distractor is re-presented as a target. In contrast to the study's hypothesis, significantly increased response latencies for the negative priming condition compared to the control condition were not observed at an ISI of 600ms or 1000ms. Although the response time cost at the longer ISI (1000ms) was not large enough to be significant, this inhibition differential was significantly different from the inhibition differential at the short ISI (600ms), which produced a response time advantage (instead of cost). Overall, these results failed to support the active-suppression theory for the distractor stimulus and failed to replicate the previous findings of Bannon et al.'s study for the ISI of 1000ms (2008).

As explored in Chapter 2, inconsistent findings throughout the negative priming literature suggests that negative priming effects could be elusive and could depend on subtle variations in the design of the experiment (Lowe, 1998; Malley & Strayer, 1995; Moore, 1994; Neill & Valdes, 1992; Neill & Westberry, 1987). The absence of a simultaneous distractor on the probe trial in this paradigm may have contributed to the lack of negative priming effects. That is, negative priming is said to depend on high-conflict probes, in which distractor stimuli interfere directly with the correct response (Moore, 1994; Tipper & Cranston, 1985). Although this suggestion may be one way of explaining discrepant findings, other studies have found negative priming effects without the presentation of a probe distractor (Bannon et al., 2008; Milliken et al., 1998, Experiment 4; Neill, Terry, & Valdes, 1994). It is also possible that the small sample

size, and thus lack of power, in the current study may have influenced results. In general, the difficulty across several experiments and researchers to capture these effects consistently and to replicate results raises important questions: does the phenomenon only occur in a narrow range of circumstances? If so, what is the mechanism underlying these effects previously attributed to distractor suppression or retrieval processes?

4.2.3.3 Summary. While this modified priming paradigm was effective at measuring facilitation processes independently, it was not successful at demonstrating significant distractor suppression or other inhibitory mechanisms. Hence, we subsequently modified a paradigm commonly used to measure inhibition (Go/Nogo paradigm) to determine if we could capture both facilitation and inhibition processes independently.

4.3 Experiment 2

The aim of Study 1 was to find paradigms that could measure facilitation and inhibition separately in OCD. Given the modified priming task did not capture significant inhibition effects, Experiment 2 investigated whether a paradigm commonly used to measure inhibition (Go/Nogo task) could capture facilitation and inhibition independently. Like the traditional priming paradigm, the Go/Nogo task has also been criticised for confounding inhibition and facilitation processes (Bannon et al., 2008; Thomas et al., 2009). That is, although commission errors to Nogo stimuli are typically interpreted as reflecting failure of inhibition, it is possible that more frequent occurrences of Go stimuli compared to Nogo stimuli may prime Go stimuli leading to a bias to respond, making commission errors more likely. If this is the case, then commission errors could be argued to reflect facilitation effects rather than a failure of inhibition processes. Hence, an alternative measure of inhibition was used in Experiment 2.

The Go/Nogo task was modified in Experiment 2 in an attempt to measure the temporal course of facilitation and inhibition independently. As is typical of a Go/Nogo task, stimuli were presented singly, in a continuous fashion, so that a stimulus was both prime to the following stimuli and probe to the preceding stimuli. In the conventional paradigm, RTs to all Go-stimuli were averaged across sequence types (e.g., Go1 and Go2). If facilitation was a central mechanism, it was posited that RTs would capture a robust linear trend of increasing facilitation (i.e., decreasing RT) with repetitions of the Go stimuli (Hypothesis 1). In other words, facilitation effects for GGGG>GGG>GG>G (G = Go stimuli), with the proviso that these effects would plateau at some level of stimulus repetition. Further, consistent with theoretical postulations for facilitation and underlying neurophysiological mechanisms believed to underpin these processes, it would be expected that facilitation would be stronger for shorter ISIs (Hypothesis 2).

Instead of using commission errors as an index of inhibition processes, an X-Go stimulus was added to examine inhibition. Similar to the priming procedure, participants were required to withhold a response to the first presentation of the Nogo1 (diamond) stimulus but respond to it if it was repeated. It was theorised that if the response to the Nogo-Go stimulus (distractor) was actively suppressed, re-presenting it as a Go-stimulus (X-Go) on the immediately following trial would result in a response cost (inhibition; Hypothesis 3). Because this active suppression process could be time-bound, three ISI conditions (600, 800, and 1000ms) were employed. As far as the author is aware, no other studies have modified the Go/Nogo task to examine inhibition (active suppression) in this manner. A valuable aspect of such an experiment is that a response-cost (unlike commission errors), if demonstrated, could be more confidently attributed to effects involving inhibitory mechanisms such as distractor suppression.

Sequence effects on commission error rate were also assessed using this paradigm. It was hypothesised that the commission error rate to Nogo1 stimuli may increase as a function of the number of preceding Go stimulus repetitions and possibly reflect facilitation effects (Hypothesis 4).

4.3.1 Method

4.3.1.1 Participants. Thirty-three undergraduate psychology students (26 females) aged between 18 and 52 years (M = 23.67, SD = 9.40) participated in Experiment 2 for partial course credit. These data were collected by Louise Turner and written up as part of her Honours Thesis (Turner, 2009). For the current study, additional data analyses were conducted on this data set.

4.3.1.2 Task - Modified Go/Nogo Paradigm. In this modified version of the Go/Nogo task 300 trials (plus 50 practice trials) were presented centrally, one at a time, on a laptop computer using Presentation® software (Version 12.2, Neurobehavioral Systems, www.neurobs.com). Four stimuli were used: Go stimuli were ovals (Go1) or circles (Go2); Nogo stimuli included a square (Nogo2) and the first presentation of a diamond (Nogo1); and the combined Nogo-Go stimulus required participants to withhold their response to the initial presentation of a diamond but then respond to two or more consecutive repetitions of a diamond (X-Go). The outline of stimuli were presented in blue on a grey background (see Figure 4.5 for example). Participants were required to press the mouse button with the index finger of their dominant hand to the Go and X-Go stimuli, and withhold a response to Nogo stimuli. Mean RTs were calculated for all correct responses to Go stimuli following correctly withheld responses to the subsequent Nogo stimuli. Trials resulting in incorrect or outlier responses (+/- 1.5 SD) were

excluded before the mean RT was calculated. Stimuli were presented for 200ms each. Three ISI conditions were administered: 600ms, 800ms, and 1000ms.

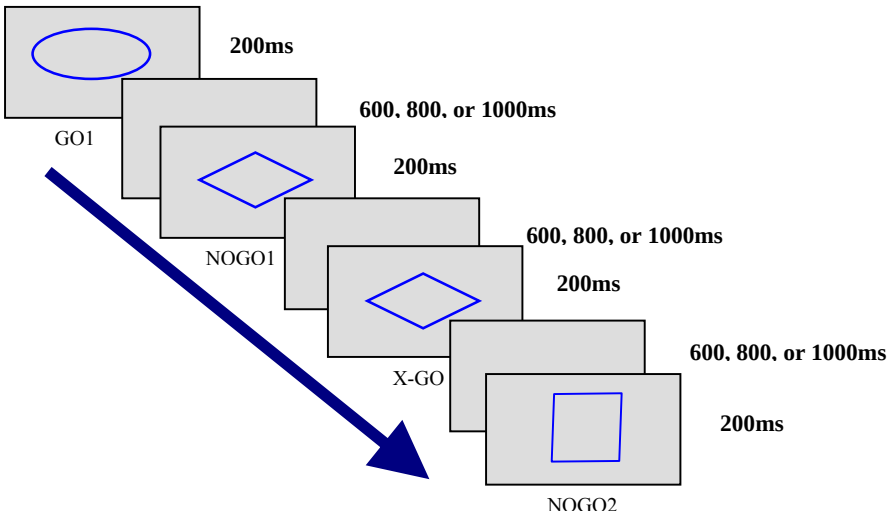


Figure 4.5. **Schematic representation of the timeline of events for the modified Go/Nogo task.**

The sequence of stimuli presentation was determined by a randomisation program that adhered to the following probability rules. Go1 stimuli were twice as probable as Nogo1 (to increase Go-stimulus repetitions and provide a measure of facilitation). Single occurrences of Nogo1 stimuli (diamond) were followed by one of three equi-probable stimuli: Nogo1 (diamond, X-Go), Go2 (circle, X-Go control), or Nogo2 (square). Go2 and Nogo2 stimuli were always followed by either a Go1 or Nogo1 stimulus, with probabilities of 0.67 and 0.33, respectively. Once the randomised program was determined, the same program was employed for all participants. The probability of the Go stimulus in the experiment block used was 0.68.

4.3.1.3 Procedure. The study was approved by the University of Wollongong Ethics committee. Written informed consent was obtained from each participant before commencement

of the experiment. Prior to commencement of the task, participants completed a one minute practice run on which they were required to attain ≥ 80% accuracy in order to progress to the actual experiment. The computer tasks took approximately 30-40 minutes to complete. Presentation of ISI conditions were counterbalanced and randomly assigned to control for order or fatigue effects.

4.3.2 Results

4.3.2.1. Facilitation. Mean response times and standard deviations for Go1 stimulus repetitions on the Go/Nogo task are displayed in Table 4.3. Mean response times for Go1 stimulus repetitions are plotted in Figure 4.6.

Table 4.3. *Mean Response Times (milliseconds) for Go Stimulus Repetitions Across Interstimulus Interval Conditions in the Modified Go/Nogo Paradigm*

	Interstimulus Interval (ms)		
Go stimulus	**600**	**800**	**1000**
G	372.67 (61.30)	376.41 (67.74)	383.68 (60.62)
GG	316. 67 (42.06)	321.79 (48.65)	335.37 (53.41)
GGG	307.68 (43.40)	309.09 (45.61)	318.20 (46.79)
GGGG	302.18 (42.12)	309.90 (48.70)	321.29 (47.33)
GGGGG	301.86 (54.28)	310.00 (53.92)	324.84 (56.87)

Note. Standard deviations in parentheses. G = Go1; RT values are for the highlighted stimulus, preceding stimuli are reported to explicate facilitation effects as a function of stimulus repetitions.

To examine facilitation effects Go1 (G) data were subjected to a 5 Stimulus (G, GG, GGG, GGGG, GGGGG) X 3 ISI (600, 800, 1000ms) repeated measures ANOVA. For the stimulus and ISI factors, two planned contrasts were employed: the linear contrast determined whether repetitions of Go1 stimuli and variations of ISI produced linear changes to response times, while the quadratic contrast assessed non-linear changes such as X- or U-shaped functions.

Go1 stimulus repetitions produced a decrease in response time, as indicated by a significant linear effect, $F(1, 32) = 88.42$, $p < 0.001$. The reduction in response time was larger after shorter (G, GG) sequence repetitions compared with longer sequence repetitions (GGGG, GGGGG), resulting in a significant quadratic contrast $F(1, 32) = 155.72$, $p < 0.001$ (see Figure 4.6). A significant linear effect was also found for ISI condition, with response time increasing as ISI increased, $F(1, 32) = 6.84$, $p < 0.05$. However, other effects including the quadratic trend for ISI condition were not significant.

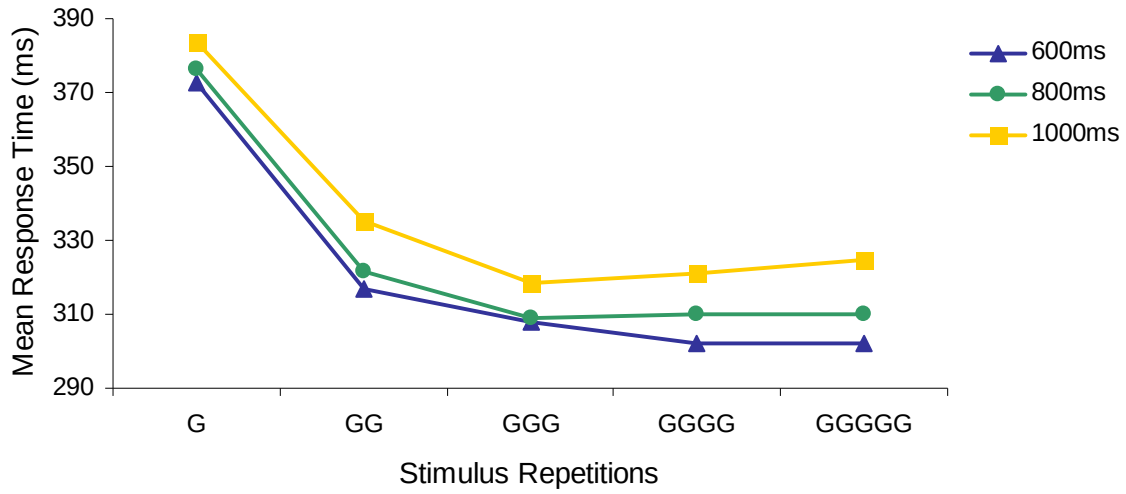


***Figure 4.6*. Mean response times (milliseconds) to Go1 stimulus repetitions (G is the first Go stimulus in a sequence, and GGGGG is the fifth) across 600ms, 800ms and 1000ms interstimulus interval conditions in the modified Go/Nogo task.**

The commission error rate to Nogo1 stimuli following Go1 stimulus repetitions is presented in Table 4.4. Due to a small number of trials in stimulus types GGGGN and GGGGGN, such data were combined into one group. To examine the effect of the number of preceding Go1 (G) stimuli upon accuracy to Nogo1 (N) stimuli, the commission error rate to Nogo1 stimuli were analysed using a 4 Stimulus (GN, GGN, GGGN, GGGGN) X 3 ISI (600, 800, 1000ms) repeated measures ANOVA. Planned contrasts assessed linear and quadratic

effects for commission errors to Nogo1 stimuli based on the number of preceding Go1 stimuli and variations in ISI. Because error data were not normally distributed, commission error data were submitted to a square root transformation. The skewness was acceptable after square root transformations. The aforementioned mixed ANOVA was performed on transformed data. Statistics for the transformed data are reported in text, whereas raw data are presented in Table 4.4 to facilitate better understanding.

A significant linear effect was found for ISI condition, $F(1, 32) = 6.75$, $p = 0.01$, with the means indicating that commission error rate decreased with increase in ISI. The linear effect was approaching significance for Stimulus, $F(1, 32) = 3.81$, $p = 0.06$. However, no significant linear trend was observed for the Stimulus X ISI interaction, $F(1, 32) = 1.99$, $p = 0.17$. Similarly, no significant quadratic trend was observed for ISI, Stimulus, or Stimulus X ISI interaction.

Table 4.4. *Mean Commission Error Percentage to Nogo1 Stimuli as a Function of Preceding Go1 Stimulus Repetitions Across Interstimulus Intervals*

	Interstimulus Interval (ms)		
Stimulus	**600**	**800**	**1000**
GN	26.03 (17.87)	15.88 (12.20)	15.27 (14.30)
GGN	23.24 (16.33)	18.39 (15.17)	17.12 (17.15)
GGGN	20.03 (14.49)	20.36 (14.66)	16.42 (17.21)
GGGGN	17.64 (14.81)	17.18 (18.16)	16.67 (16.33)

Note. Standard deviations in parentheses; ms = milliseconds; G = Go1; N = Nogo1; Commission error percentage are reported for the highlighted stimulus.

4.3.2.2. Inhibition. In relation to the modified Go/Nogo paradigm it was predicted X-Go stimuli would produce a delayed mean RT compared to the mean RT of a non-inhibited (equi-probable control) stimulus (i.e., Nogo1 then Go2). Only X-Go and Go2 trials after which participants correctly suppressed a response to the preceding Nogo1 stimulus were analysed. These data were subjected to a 2 Stimulus type (X-Go control, X-Go) X 3 ISI (600, 800,

1000ms) repeated measures ANOVA. Planned linear and quadratic contrasts were employed for the ISI condition.

Mean response times, standard deviations, and inhibition differentials are presented in Table 4.5. Planned contrasts revealed a significant effect for Stimulus type $F(1, 32) = 21.94$, $p < 0.001$, with X-Go stimuli producing a faster mean RT compared to X-Go control stimuli. A significant linear effect was also found for ISI, $F(1, 32) = 13.60$, $p = 0.001$, with mean RTs increasing as ISI increased. No significant quadratic trend for ISI condition was observed.

Table 4.5. *Mean Response Times (ms) for Inhibition Data Across Interstimulus Interval (ISI) Conditions in the Modified Go/Nogo Paradigm*

		Condition		
Task	**ISI (ms)**	**X-Go Control**	**X-Go**	**Inhibition**
Modified Go/Nogo	600	390.23 (62.46)	342.26 (90.44)	47.97 (67.82)
	800	413.04 (57.31)	359.99 (94.64)	53.05 (80.07)
	1000	413.16 (64.95)	369.23 (86.10)	43.93 (52.68)

Note. Standard deviations in parentheses; ms = milliseconds; Inhibition = X-Go control RT – X-Go RT (higher negative values indicates stronger inhibition, positive values indicate facilitation).

Omission errors to X-Go and X-Go control stimuli were analysed using a 2 Stimulus type (X-Go control, X-Go) X 3 ISI (600, 800, 1000ms) repeated measures ANOVA. Because error data were not normally distributed, omission error data were submitted to a square root transformation. The skewness was acceptable after square root transformations. The aforementioned mixed ANOVA was performed on transformed data. Statistics for transformed data are reported in text, whereas raw data are presented in Table 4.6 to facilitate better understanding.

Table 4.6. *Mean Omission Error Percentage to X-Go and X-Go Control Stimuli Across Interstimulus Intervals*

		Stimulus Type	
Task	**ISI (ms)**	**X-Go Control**	**X-Go**
Modified Go/Nogo	600	9.09 (11.09)	15.15 (12.86)
	800	7.55 (10.88)	15.55 (9.50)
	1000	5.88 (9.47)	15.15 (10.58)

Note. Standard deviations in parentheses; ms = milliseconds.

A significant effect was found for Stimulus type, $F(1, 32) = 34.92$, $p < 0.001$, with X-Go stimuli producing significantly more omission errors ($M = 15\%$) compared to the X-Go control ($M = 7.5\%$). The Stimulus X ISI interaction was also significant, $F(1, 32) = 4.01$, $p = 0.05$, with the number of omission errors to X-Go stimuli staying constant across ISIs, but decreasing with ISI increase for the X-Go control stimulus. No other significant linear or quadratic trends were observed.

4.3.3. Discussion

Study 1 aimed to design a paradigm that could capture facilitation and inhibition effects separately so this could be applied to an OCD group in Study 2. Consistent with this goal, the current experiment modified a Go/Nogo task to try and capture facilitation and inhibition effects separately. Specifically, the current experiment modified the conventional Go/Nogo task by increasing stimulus repetitions to examine facilitation, and added the X-Go stimuli to examine whether Nogo stimuli are actively suppressed (inhibition processes). Multiple ISIs were used to examine the temporal course of facilitation and inhibition and to enhance the chances of capturing the elusive inhibitory effects. The sequence effect of Go stimulus repetitions on commission error rate was also assessed.

4.3.3.1. Facilitation. It was hypothesised that by increasing Go stimulus repetitions in the Go/Nogo task it would provide an effective measure of facilitation. The significant reduction in response times to Go stimulus repetitions confirmed this. More specifically, larger reductions in response times were produced to initial Go stimulus repetitions compared with latter sequence repetitions. The finding that facilitation reached a plateau after several stimulus presentations was not surprising and is likely to represent floor effects. Similar findings have been reported in neuropsychological investigations (e.g., Grill-Spector, Henson & Martin, 2006) whereby initial increases in responding and reductions in neuronal activity are followed by plateau effects. The reliable demonstration of facilitation effects in this task reinforces previous criticisms of the Go/Nogo paradigm for confounding constructs of inhibition and facilitation (Bannon et al., 2008; Thomas et al., 2009). That is, previous Go/Nogo findings attributed to inhibition processes may actually have been due to facilitation or a combination of facilitation and inhibition processes.

Further, response times decreased as ISI decreased, indicating that facilitation processes were sensitive to temporal manipulations with stronger facilitation observed at shorter ISIs (see Figure 4.6). The facilitation effects observed across ISIs of 600-1000ms are consistent with Salo et al.'s (2002) findings of facilitation at both 500ms and 2000ms RSIs, and Waechter et al.'s (2010) replication of positive priming across ISIs ranging from 50ms to 650ms. Thus, the current study suggests the facilitation effect is stable and robust over a temporal course of 600-1000ms.

An added advantage of using the modified Go/Nogo task to measure facilitation is that it enables the examination of facilitation effects across multiple stimulus repetitions, as opposed to the sole stimulus repetition in priming tasks. Furthermore, the modified Go/Nogo task provides additional measures compared to the conventional Go/Nogo task. That is, the conventional Go/Nogo tasks yielded only 2 measures: overall RT to Go stimuli and errors. In contrast, the new

task provides the ability to measure errors, stimulus and response facilitation (i.e., repetition of same stimulus and response - Go1 repetitions), response facilitation (i.e., repetition of Go response with presentation of different Go stimuli, Go1 and Go2), and RTs to X-Go stimuli that have recently been suppressed. Each of these can also be analysed as a function of sequence.

It was hypothesised that the commission error rate to Nogo1 stimuli may increase as a function of the number of preceding Go stimulus repetitions and possibly reflect facilitation effects. In contrast to the study's predictions, a linear trend approaching significance in the opposite direction to that hypothesised was observed. This suggests that factors other than facilitation or inhibition may influence commission error rate. For example, strategic expectancies may explain the decrease in commission error percentage as the number of Go1 stimulus repetitions preceding the presentation of Nogo1 stimuli increased. That is, participants may have been more likely to anticipate a change to a Nogo stimulus by the fourth and fifth Go1 stimulus repetitions, and thus were more cautious about their responses at latter Go1 stimulus repetitions. While such findings contrast with Durston et al.'s (2002) results, they are consistent with Thomas et al.'s (2009) findings. The significant linear effect observed for ISI condition suggests that control strategies may also influence commission error rate. That is, when participants had more time to think about their responses in between stimulus presentations they made less errors. In summary, such findings suggest that various factors including strategic expectancies, control strategies and inhibition or facilitation may influence commission error rate in Go/Nogo tasks.

4.3.3.2. Inhibition. This experiment failed to yield evidence for the active suppression of the distractor concept. In contrast to the study's hypothesis, a significant response time advantage (instead of cost) was found for X-Go stimuli compared to X-Go control stimuli at all

three ISIs examined (600, 800, and 1000ms). Because only correct trials were included (correct omissions to Nogo1 followed by correct responses to X-Go), these results are particularly difficult to explain by the active suppression or the episodic-retrieval hypothesis. If the distractor (Nogo stimuli) is suppressed in a systematic manner, why would a re-presentation of the distractor be associated with shorter rather than longer RTs on most of the trials? Interestingly, the X-Go stimulus was also found to produce significantly more omission errors compared to the X-Go control stimulus. The gap between omission errors produced to X-Go and X-Go control stimuli was most pronounced at 1000ms ISI. This pattern of results is perhaps best accommodated by a more complex, response-competition model that suggests the implication of several factors that may include but are not restricted to response-conflict. This model is further discussed later.

4.3.3.3. Summary. Although this paradigm provided an effective measure of facilitation, it did not yield evidence for active suppression of the distractor stimulus (inhibition). The results reinforce the notion that evidence for Houghton and Tipper's (1994) active-suppression hypothesis is elusive and, at best, may only be obtained in a narrow set of circumstances, such as with the simultaneous presentation of a probe distractor. That is, both modified paradigms in Experiment 1 and 2 were different from conventional tasks because they used a sequential presentation of target and distractor stimuli instead of simultaneous presentation, and both failed to find evidence for the active suppression of the distractor stimulus. Hence, in an attempt to capture hitherto elusive evidence for distractor suppression, Experiment 3 utilised a conventional priming task in which targets and distractors were presented simultaneously. Paradoxically, whilst traditional Go/Nogo tasks were alleged to be sensitive to inhibition, the new modified Go/Nogo task demonstrated that facilitation effects could be reliably captured, as evidenced by

linear and quadratic trends and ISI effects. This paradigm shows promise for use in clinical groups.

4.4 Experiment 3

The design of the conventional priming paradigm was based in part on the priming procedure used by Neumann and DeSchepper (1991). Several modifications were made for the potential future use of this paradigm with clinical populations. This included the use of words in place of letters. In addition, an immediate response to the target word was required on both prime and probe trials, instead of just the probe trial. Furthermore, in order to maintain continuity between Experiments 1 and 2 participants were required to respond to targets via a manual keypress response, as an alternative to vocal naming of the target word (as some clinical participants might not have been willing to name threatening words). Positive and negative priming effects have still been reported in studies utilising manual keypress responses instead of vocal naming responses (Neill & Westberry, 1987; Stadler & Hogan, 1996; Tipper et al., 1991). Given the argument that a probe distractor is needed to produce negative priming effects (Moore, 1994; Tipper & Cranston, 1985), it was predicted that the simultaneous presentation of target and distractor on prime and probe trials in this paradigm would create conflict on the probe trial and produce a slower response time on negative priming trials (Hypothesis 1) and faster response time on positive priming trials (Hypothesis 2) compared to control trials.

4.4.1. Method

4.4.1.1. Participants. Twenty-seven undergraduate psychology students (16 females) aged between18 and 59 years (M = 29.15, SD = 11.08) participated in Experiment 3 for partial course credit.

4.4.1.2. Task -Traditional Priming Paradigm. In this paradigm words were presented on a laptop computer using Presentation® software. Each trial consisted of a warning stimulus (**!**), a fixation cross (**+**), the presentation of a word pair at Time 1, a fixation cross (**+**), and the presentation of a second word pair at Time 2 (see Figure 4.7). These words were presented in capital letters in the centre of the screen, and were separated from left to right by four spaces. One word was presented in green and the other in blue. Using a keypress response participants were instructed to indicate which side the green target word was presented "as quickly and accurately as possible."

This paradigm involved the presentation of 240 trials (plus 24 practice trials) that were presented in 3 blocks of 80 trials with 40-second breaks between blocks. The 240 trials were randomly presented and consisted of the following breakdown: 120 control trials (target and distractor word at Time 2 did not match the target or distractor word from Time 1); 60 positive priming trials (target word at Time 2 was the same as the target word at Time 1); and 60 negative priming trials (target word at Time 2 was the same as the distractor word at Time 1).

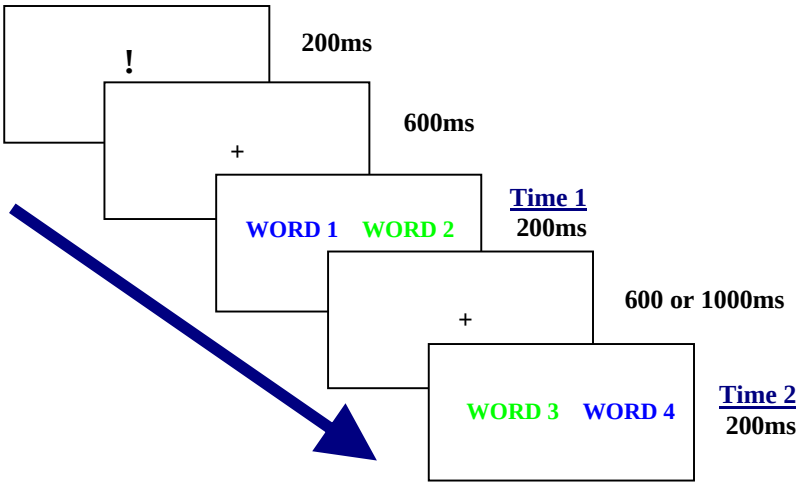


Figure 4.7. **Schematic representation of the timeline of events of a control trial in the traditional priming paradigm.**

Twelve neutral words were used throughout the 240 trials (see Appendix E). These words were acquired via the Western Australian psycholinguistics database and did not differ in word length (5 letters) or frequency (40-60 Kucera-Francis written frequency). Each word was presented as a target and distractor an equal number of times, and the degree to which the target words were presented on the left and right hand side at Time 1 and 2 was counterbalanced. Participants completed the priming paradigm twice, at an ISI of 600ms and 1000ms, using two different lists of twelve words. To ensure that participants were attending to the identity of the word (and not just responding to colour), participants were informed that they would need to complete a recognition test of the target words on completion of the computer task.

Mean RTs were calculated for all correct responses. Trials resulting in incorrect or outlier responses (+/- 1.5 *SD*) were excluded before the mean RT was calculated. The facilitation differential was calculated by subtracting the mean RT at Time 2 in the positive priming trials from the controls trials. The inhibition differential was calculated by subtracting the mean RT at Time 2 in the negative priming trials from the control trials.

4.4.1.3. Procedure. The study was approved by the University of Wollongong Ethics committee. Written informed consent was obtained from each participant before commencement of the experiment. Participants were instructed to respond to the 'target' word using their index or ring finger of their dominant hand. Right handed participants used the (←) and (→) keys to indicate left and right, respectively. Left handed participants used the (z) and (c) keys on the keyboard to indicate left and right, respectively. The computer tasks took approximately 40 minutes to complete. Presentation of ISI conditions were counterbalanced and randomly assigned to control for order and fatigue effects. Participants also completed a recognition test of target words at the conclusion of the computer task.

4.4.2. Results

4.4.2.1. Facilitation. Mean response times, standard deviations, and facilitation differentials for the priming paradigm are presented in Table 4.7. As the assumption of normality was not met Wilcoxon Signed-Ranks non-parametric tests were utilised to examine mean differences between control and positive priming trials, and to determine whether this paradigm produced a significant facilitation effect. As is evident from the mean facilitation differentials in Table 4.7, differences between control and positive priming conditions were marginal. Consistent with this, no significant differences were found between control and positive priming conditions at an ISI of 600ms, $Z = -0.22$, $p = 0.83$, $r = -0.04$ or 1000ms, $Z = -0.96$, $p = 0.34$, $r = -0.18$, suggesting this paradigm was not effective at capturing a facilitation effect.

Table 4.7. *Mean Response Times (ms) for Facilitation Data Across Interstimulus Interval (ISI) Conditions in the Traditional Priming Paradigm*

		Condition		
Task	**ISI (ms)**	**Control**	**PP**	**Facilitation**
Traditional Priming	600	341.89 (44.76)	341.24 (44.11)	0.64 (7.94)
	1000	345.83 (51.72)	345.09 (51.99)	0.74 (5.99)

Note. Standard deviations in parentheses; ms = milliseconds; PP = positive priming; Facilitation = control RT – positive priming RT.

4.4.2.2. Inhibition. Mean response times, standard deviations, and inhibition differentials for the priming paradigm are presented in Table 4.8. As the assumption of normality was not met Wilcoxon Signed-Ranks tests were employed. It is apparent from the mean inhibition differentials in Table 4.8 that differences between the control and negative priming conditions were marginal. Consistent with this, no significant differences were found between control and negative priming conditions at an ISI of 600ms, $Z = -0.41$, $p = 0.68$, $r = -0.08$ or 1000ms, $Z = -1.78$, $p = 0.08$, $r = -0.34$. Similar to the facilitation data, these results suggest this paradigm was not effective at capturing an inhibition effect. In regards to errors, the percentage of response errors was < 3% over all trial types (control, positive priming, and negative priming) for both ISIs, and was not submitted to further analysis.

Table 4.8. *Mean Response Times (ms) for Inhibition Data Across Interstimulus Interval Conditions (ISI) in the Traditional Priming Paradigm*

		Condition		
Task	**ISI (ms)**	**Control**	**NP**	**Inhibition**
Traditional Priming	600	341.88 (44.76)	342.34 (44.63)	1.30 (10.76)
	1000	345.83 (51.72)	343.26 (51.57)	3.17 (6.70)

Note. Standard deviations in parentheses; ms = milliseconds; NP = negative priming; Inhibition = control RT – negative priming RT (higher negative values indicates stronger inhibition, positive values indicate facilitation).

4.4.3. Discussion

In contrast with the study's hypotheses, the traditional priming paradigm did not produce significant negative or positive priming effects at an ISI of 600ms or 1000ms. These results contrast with studies that have found positive and negative priming using a key-press response (Neill & Westberry, 1987; Stadler & Hogan, 1996).

The target and distractor words in our paradigm were presented further apart (spatially) from each other compared to other identity priming studies which have presented them overlapping each other (Neumann & DeSchepper, 1991; Stadler & Hogan, 1996; Tipper, 1985) or within the same stimulus using Stroop conflict or Stroop nonconflict stimuli (Neill & Westberry, 1987). It is possible that the distractors were so easily distinguished from targets by both their colour and spatial separation that a selection state did not need to be maintained in this paradigm and thus, negative priming effects were not produced. Similar explanations were theorised to explain other findings that failed to capture inhibition (Neill et al., 1995; Tipper & Cranston, 1985). If this is indeed the case, the study reiterates the elusiveness of the inhibition effects. However, it is also important to acknowledge that null results do not disprove theories of negative priming.

4.5 General Discussion

The current study makes several important contributions to the selective attention literature. Firstly, it is one of few studies attempting to examine facilitation and inhibition effects separately by modifying commonly used paradigms that have been criticised for confounding facilitation and inhibition effects. This resulted in the contribution of a new Go/Nogo task that provides not only a measure of facilitation, but also plots its progression across stimulus

sequence and can disentangle stimulus-response and response facilitation. Such additional measures of facilitation will be valuable when assessing facilitation in clinical populations. Secondly, it investigated the temporal course of facilitation and inhibition, which prior to this study has received limited attention. Thirdly, it is one of few studies investigating the influence of Go stimulus sequence effects on commission errors in a Go/Nogo task, to determine whether commission errors really reflect a failure of inhibition, or facilitation effects. It is also important to note that this series of experiments was conducted to determine which experimental paradigms will provide the best information when measuring facilitation and inhibition in OCD in Study 2.

In regards to facilitation, both the modified priming (Experiment 1) and modified Go/Nogo tasks (Experiment 2) were found to be effective at measuring facilitation processes independently. These results are consistent with previous findings from the identity priming literature that suggest facilitation is a reliable finding across tasks (Gibbons et al., Stahl, 2006; Groh-Bordin & Frings, 2009; Koshino et al., 2000; Lowe, 1979; Neumann & DeSchepper, 1991; Salo et al., 2002; Stadler & Hogan, 1996; Tipper, 1985; Troche et al., 2008; Waechter et al., 2010). In addition, significant facilitation effects were observed at all temporal intervals from 600ms-1000ms in modified paradigms. The robustness and replication of facilitation effects across ISIs of 600ms-1000ms is consistent with previous research on facilitation (Salo et al., 2002; Troche et al., 2008; Waechter et al., 2010). Hence, it appears facilitation effects are stable across time suggesting this aspect of selective attention is always in operation.

In regards to commission errors, findings from the Go/Nogo task suggest various factors may influence commission error rates in addition to inhibition or facilitation. Such factors may include strategic expectancies or control strategies. For example, when people have more time to think about their response in between stimulus presentation (i.e., at longer ISIs) they may be

more cautious about their responses, resulting in fewer commission errors. Given commission errors are commonly used as a measure of inhibition in Go/Nogo tasks, this study highlights the need for further research investigating what factors influence commission errors and thus what mechanisms underlie commission errors.

In regards to inhibition, Tipper and colleagues (Houghton & Tipper, 1994; Tipper, 1985; Tipper et al., 1994) used negative priming effects as evidence for the theory that internal representations of distractor stimuli are actively suppressed in order to select and respond to simultaneously present target stimuli. Two previous findings (Bannon et al., 2008; Milliken et al., 1998) yielded evidence that the active suppression hypothesis could be captured in priming paradigms using a sequential presentation of target and distractor stimuli. In contrast to these earlier findings, no significant inhibition effects were found in Experiments 1 or 2 when the distractor was presented sequentially, or in Experiment 3 when the target and distractor stimuli were presented simultaneously. In effect, no evidence for active suppression of the distractor was found in the three experiments. There are at least two possible explanations: (a) inhibition is a state- and context-dependent mechanism; and (b) the hypothesised process of active suppression of the distractor stimuli does not occur and the delayed RT observed in negative priming experiments are associated with alternative mechanisms.

(a) Inhibition is a state- and context dependent mechanism: Other studies using priming paradigms have also failed to produce negative priming effects (e.g. Allport et al. 1985; Lowe, 1979; Malley & Strayer, 1995; Neill, 1977; Tipper & Cranston, 1985). Previous research suggests that experimental design factors (experimenter instruction, difficult probe distractor, temporal manipulations, number of stimuli, repetition of stimuli, contextual similarity, and response modality) all need to be "just right" to produce negative priming effects (Lowe, 1998;

MacLeod et al., 2002; Malley & Strayer, 1995; Moore, 1994; Neill, 1977; Neill et al., 1992; Neill & Westberry, 1987; Tipper & Cranston, 1985). Tipper and colleagues suggested priming paradigms may not always produce the predicted negative priming effects because only goal-dependent properties of the distractor are suppressed while others remain active (Tipper et al., 1994). However, this explanation does not appear to explain our results. Specifically, the goal for our first two paradigms was target identification via a keypress response, but no identity negative priming effects were found despite the target identification goal. In Experiment 3, target identification via a keypress response to its location was the objective. Once again no identity priming results were found. If it is the case that inhibition is a state-and context-dependent mechanism, greater specification regarding the factors that influence negative priming effects is required.

(b) Active suppression does not occur and alternative explanations may underpin response costs previously attributed to negative priming: Several alternative theories for negative priming have been proposed.

(i) Feature mismatch theory (Park & Kanwisher, 1994) states that negative priming effects are a result of stimulus conflict, and facilitation results should be observed when identical stimuli are repeated. This theory predicts that facilitation effects will be produced when the distractor stimulus in the prime trial is re-presented in the exactly the same manner (e.g., same colour) as a target stimulus in the probe trial in Experiments 1 and 2. Whilst significant facilitation was found in Experiment 2, it was not in Experiment 1. Furthermore, the stimulus colour mismatch on prime and probe trials in Experiment 3, in which the word is presented in blue (distractor) and then green (target), is expected to produce negative priming effects, but none were found. Hence, this theory does not appear to be able to account for these results.

(ii) The episodic retrieval theory (Neill & Valdes, 1992; Neill et al., 1992) holds that negative priming effects are the result of conflict between a "do not respond" tag for the distractor stimulus on the prime trial and a "respond" tag for the same stimulus when it is re-presented as a target on the probe trial. The delay in responding is said to reflect the time needed to resolve this conflict. The episodic retrieval theory would predict negative priming effects in all three of our experiments as they all involve the re-presentation of the distractor stimulus ("do not respond" tag) in the prime trial as a target stimulus ("respond" tag) in the probe trial. However, no negative priming effects were found for any of our experiments, indicating the episodic retrieval theory also cannot account for these results.

(iii) The pattern of results observed in this series of studies is best accommodated by a more complex, response-competition model that suggests the implication of several factors including, (1) strength of stimulus activation, (2) strength of the required response, and (3) strength of competing responses including response conflict that may occur in certain circumstances. The response gain observed in Experiments 1 and 2 for repeated target and Go-stimuli implicate the combined effects of the first two factors. Responses to X-Go stimuli involve a response advantage associated with stimulus repetition and a response cost associated with conflicting response requirements, while responses to Go2 (X-Go control) stimuli involve response competition but no response advantage associated with stimulus repetition. Such an explanation does not implicate the notion of active suppression.

In effect, the facilitation differential observed for the X-Go stimulus is due to the greater delay to the control Go2 stimulus brought about by the low-probability of the Go2 stimulus. In other words, the lower probability of the Go2 stimulus produces a slower response time relative to response times to X-Go stimulus which are affected by both stimulus activation gains and

response conflict costs. Furthermore, the larger number of omission errors to X-Go compared to Go2 stimuli may be the consequence of confusion and/or conflict and be explained by the fact that two different responses (Nogo and Go) are called for to the X-stimulus (diamond) at different times whereas only Go responses are required for the Go2 stimulus. However, research needs to examine whether such factors can explain the presence or absence of negative priming effects. Only when negative priming effects are able to be predicted and produced in a reliable fashion will researchers be close to determining the mechanisms or factors underlying negative priming.

4.5.1 Limitations and Future Research

This study contributes new findings to the selective attention literature. Despite this, potential limitations must be considered in interpreting these results. Suggestions for future research will also be discussed

A limitation of the current research was that none of the modified paradigms were able to adequately capture negative priming effects in normal controls. Although null results do not disprove theories of negative priming, it challenges them to become more specific about their predictions for confirmation and corroboration. That is, future research needs to determine the specific experimental factors that produce negative priming effects. This will also provide insight into the mechanisms underlying negative priming effects (e.g., inhibition, episodic retrieval). Furthermore, if facilitation processes are always in operation further research needs to establish if there an experimental paradigm that can tease apart inhibition processes and measure it independently without being confounded by facilitation processes.

Given the effectiveness of the modified priming and Go/Nogo paradigms at measuring facilitation processes, future research could utilise such paradigms to further examine the time course of facilitation processes. That is, studies could employ longer ISIs (> 1000ms) to determine the duration to which facilitation effects persist. Such research would clarify the duration of facilitation effects instead of relying on Houghton and Tipper's (1994) estimate of "some time."

The modified priming and modified Go/Nogo tasks also have useful clinical applications. Using the same modified priming paradigm Bannon and colleagues (2008) found reduced inhibition and excessive facilitation in people with OCD compared to a normal control group. Further research needs to examine whether these results can be replicated. Study 2 aims to address this. Although there has been a heavy focus on inhibition processes in OCD throughout the literature, only a few studies have examined facilitation processes in OCD. Given the importance of facilitation processes in selective attention, it would be valuable to examine the facilitation processes in those with OCD using the modified priming and Go/Nogo tasks. The advantage of demonstrating facilitation abnormalities across several tasks is that it indicates it is a reliable and stable finding. Study 2 aims to directly address this issue.

4.5.2 Summary and Conclusions

In summary, this study contributed to the current literature in the following ways: First, it demonstrated that the modified priming and modified Go/Nogo task are effective at measuring facilitation effects. Second, facilitation effects were produced at multiple temporal intervals from 600ms-1000ms, indicating such effects are stable and robust over this temporal interval. Third, findings in relation to commission errors suggest that they are influenced by various factors such

as inhibition, facilitation, strategic expectancies and control strategies. Fourth, all three experimental paradigms failed to yield evidence for active suppression of distractor stimuli, raising questions as to the nature of the mechanisms underlying reported inhibition effects. Because such paradigms did not produce significant negative priming effects, whether inhibition rapidly decays over time is still unclear. A manuscript regarding Study 1 will be submitted to *Journal of Experimental Psychology: Learning, Memory and Cognition* after the submission of this thesis.

As stated previously, this series of experiments was conducted to determine which paradigms would be the most appropriate to use in Study 2 when investigating facilitation and inhibition in OCD. Development of the experimental paradigms and conducting the three studies to evaluate these paradigms was done over a period of two years (2007-2009). Given the results from Study 1 and the time needed to collect data from clinical populations for Study 2, it was decided that the modified priming and Go/Nogo paradigms would be used to examine facilitation and inhibition in OCD in Study 2. That is, as the conventional priming paradigm was not found to produce significant facilitation or inhibition effects it will not be used in Study 2.

In regards to the modified priming paradigm, it was shown to be an effective measure of facilitation. Although the negative priming results in the modified priming paradigm at 1000ms ISI were not significantly different from the control condition, a response time cost was observed. Hence, given the response cost at 1000ms with normal controls and that Bannon et al. (2008) found OCD participants showed less negative priming compared to normal control and panic disorder patients using this same paradigm, it is valuable to examine facilitation and inhibition in OCD using this paradigm. An added benefit of this approach is the replication of Bannon et al.'s (2008) study. The modified Go/Nogo paradigm was also found to be an effective

measure of facilitation. The added advantage of using the modified Go/Nogo task in addition to the modified priming task is that it enables the examination of facilitation effects across multiple Go stimulus repetitions, as opposed to the sole stimulus repetition in the priming task and can disentangle stimulus-response and response priming. In summary, the modified priming and Go/Nogo tasks will be used in Study 2 to investigate facilitation and inhibition in OCD.

CHAPTER 5

STUDY 2: FACILITATION AND INHIBITION ABNORMALITIES IN OBSESSIVE-COMPULSIVE DISORDER

5.1 Rationale and Aims of Study 2

Recent research suggests that enhanced facilitation and impaired inhibition may contribute to the repetitive nature of obsessions and compulsions in OCD (Bannon et al., 2008). However, few studies have examined facilitation in OCD, and findings are conflicting regarding inhibition in OCD. Hence, the primary aim of Study 2 was to address this gap in the OCD literature by examining both facilitation and inhibition in OCD. Because traditional priming and Go/Nogo paradigms have been criticised for confounding facilitation and inhibition, Study 2 utilised the modified priming and Go/Nogo paradigms from Study 1 to measure such processes independently. An added benefit of such an approach is the replication of Bannon et al.'s (2008) seminal study that was one of first to argue that OCD may be characterised by a combination of both deficits. Furthermore, the inclusion of the Go/Nogo task enables the examination of facilitation effects across multiple Go stimulus repetitions and the differentiation between stimulus-response facilitation and response facilitation, which has not yet been examined in OCD.

It is conceivable that differences between OCD and controls in previous studies may not be due to differences in the strength of facilitation and/or inhibition in OCD, but instead due to the result of differences in the temporal course of facilitation and/or inhibition in OCD. In other words it is useful to determine if activation levels for salient stimuli/responses is both stronger and/or lasts longer. These differences may have pharmacokinetic implications because of

differential properties of the many pharmacological agents available today. As limited research has investigated the temporal course of inhibition and facilitation in OCD, Study 2 aimed to address this gap in the literature. Specifically, participants completed the modified Go/Nogo at two ISI conditions (600ms and 1000ms).

In addition, the present study aimed to test the hypothesis that such selective attention abnormalities are specific to OCD by including a normal control group and a clinical control group that shared similar clinical features with OCD, namely, GAD.

5.2 Hypotheses and Objectives of Study 2

Based on the literature reviewed in Chapter 3, hypotheses for Study 2 were generated:

1. OCD, GAD and normal control participants will not differ on baseline response time measures in the priming task (i.e., mean RT for facilitation and inhibition control conditions) or the Go/Nogo task (i.e., mean RT for initial Go1 and Go2 stimuli in sequence).
2. OCD participants will display stronger facilitation in the priming task (i.e., larger facilitation differential) and Go/Nogo task (i.e., larger response time reductions to repeated Go stimuli) compared to normal control and GAD participants (Bannon et al., 2008).
3. OCD participants will display weaker inhibition in the priming task (i.e., smaller inhibition differential) and Go/Nogo task (i.e., smaller differential between X-Go and X-Go control stimuli) compared to normal control and GAD participants (Bannon et al., 2008).

We also wanted to determine whether:

4. Temporal determinants (e.g., ISI) will influence facilitation and inhibition across groups and will differentially affect the 3 groups.
5. Facilitation and inhibition differentials on different cognitive tasks will correlate with each other
6. Facilitation and inhibition will correlate with the severity of OCD or GAD symptoms.
7. Facilitation and inhibition will correlate with checking and washing subtypes of OCD.

5.3 Method

5.3.1 Participants

Sixty participants took part in this study. Participants in the two clinical groups were allocated to their respective groups based on their primary diagnosis according to the Anxiety Disorders Interview Schedule-IV (DiNardo, Brown, & Barlow, 1994), which was administered by a qualified clinical psychologist or intern clinical psychologist (under supervision) from Westmead Hospital's Anxiety Treatment and Research Unit. Twenty-one participants were diagnosed with OCD, 18 were diagnosed with GAD, and 21 had no current or past mental health diagnosis and were allocated to the normal control group. Initially clinical participants were just recruited from Westmead Hospital's Anxiety Treatment and Research Unit (Sydney, Australia). However, due to difficulties recruiting clinical participants, recruitment had to target multiple locations. Hence, clinical participants were also recruited from OCD Support Groups (Mental Health Association NSW, Australia), and the University of Wollongong (via a media release). The total duration of data collection spanned 2 years. Normal controls were recruited via a media release in the Illawarra region (NSW, Australia) and internet advertisement (Sydney Exchange).

Exclusion criteria for all participants included any history of substance abuse or dependence, brain damage or neurological disease (e.g., Huntington's disease, Parkinson's disease, or epilepsy), bipolar or psychotic disorder, and mental retardation. Such conditions were excluded in the current study because of their potential influence on dependent measures. Nine participants with OCD had secondary diagnoses (5 major depression, 3 dysthymic disorder, 2 social phobia, and 1 panic disorder), and 14 participants with GAD had secondary diagnoses (6 major depression, 3 dysthymic disorder, 5 social phobia, 4 panic disorder, and 5 specific phobia). Although these secondary diagnoses may potentially confound the study, the comorbidity of OCD and GAD with other anxiety disorders and depression is high (as discussed in Chapters 1 and 3), making recruitment of a 'pure' OCD and GAD sample difficult. Participants with comorbid OCD and GAD diagnoses were not included in the study.

Twenty-two participants were medicated at the time of testing (10 OCD, 12 GAD): 5 persons with OCD and 4 persons with GAD were taking selective serotonin reuptake inhibitors; 2 persons with OCD and 4 persons with GAD were taking serotonin-norepinephrine reuptake inhibitors; 2 persons with OCD and 6 persons with GAD were taking benzodiazepines; 2 persons with OCD and 1 person with GAD were taking antipsychotics; and 2 persons with OCD and 1 person with OCD were taking tricyclic antidepressants and monoamine oxidase inhibitor, respectively.

5.3.2 Questionnaires

5.3.2.1 Anxiety Disorders Interview Schedule-IV (ADIS-IV; DiNardo et al., 1994). The ADIS-IV is a clinician administered, semi-structured diagnostic interview developed specifically to diagnose anxiety disorders according to the DSM-IV (American Psychiatric Association,

1994). Other disorders frequently associated with anxiety, such as depression and dysthymia, are also assessed. The ADIS-IV has good to excellent reliability for most DSM-IV diagnostic categories, with the majority of kappas between 0.60 and 0.86 (Brown, Di Nardo, Lehman, & Campbell, 2001).

The ADIS-IV was administered to confirm clinical diagnoses of OCD and GAD and assess for other anxiety disorders, depression, substance abuse, bipolar, and psychotic disorders.

5.3.2.2 Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989). The Y-BOCS is a 10-item semi-structured clinician administered interview designed to assess the severity of obsessions and compulsions. The Y-BOCS has demonstrated good psychometric properties, such as excellent interrater reliability (r_s = 0.93 to 0.98) and good internal consistency (α = 0.69 to 0.91; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989; Woody, Steketee, & Chambless, 1995). In regards to validity, the Y-BOCS has demonstrated convergent validity with other measures of OCD symptomatology, such as the Clinical Global Impression-Obsessive Compulsive scale (r = 0.74; Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989). The Y-BOCS has also shown to be sensitive to changes in OCD symptomatology following pharmacological and cognitive-behavioural treatment (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; van Oppen, Emmelkamp, van Balkom, & van Dyck, 1995).

The Y-BOCS was administered to OCD patients to obtain a clinician rated score for OCD severity. The Y-BOCS was administered by a qualified clinical psychologist or intern clinical psychologist (under supervision) from Westmead Hospital's Anxiety Treatment and Research

Unit. The overall total on the Y-BOCS scale was used in the current study as the dependent measure for OCD severity.

5.3.2.3 Padua Inventory – Washington State University Revision (PI-WSUR; Burns, Keortge, Formea, & Sternberger, 1996). The PI-WSUR is a 39-item self-report questionnaire that measures the degree of distress caused by obsessions and compulsions. The five subscales of the PI-WSUR measure: (i) contamination obsessions and washing compulsions; (ii) dressing/grooming compulsions; (iii) checking compulsions; (iv) obsessional thoughts of harm to self/others; and (v) obsessional impulses to harm self/others (Burns et al., 1996). The items on the PI-WSUR are a subset of items selected from the original Padua Inventory (PI; Sanavio, 1988). The PI-WSUR has improved psychometric properties compared to the original PI. For example, Burns et al. (1996) found it demonstrated an improved ability to discriminate between obsessions and worry (as measured by the Penn State Worry Questionniare; PSWQ) than the original PI. Specifically, they found the PI-WSUR shares only 12% of its variance with the PSWQ compared to 34% for the original PI. In addition, although the total and subscale scores on the PI-WSUR are significantly correlated with scores on the PSWQ (r_s = 0.08 to 0.37), items on the PI-WSUR are more strongly correlated with the PI-WSUR total or its own subscale than with the PSWQ (Burns et al., 1996). In regards to reliability, the PI-WSUR total and subscales have demonstrated good internal consistency (α = 0.77 to 0.92) and test-retest reliability (r_s = 0.61 to 0.84; Burns et al., 1996).

The PI-WSUR was administered to OCD patients to obtain a self-report measure of OCD severity, and a dimensional score for contamination/washing and checking subtypes. Total scores

on the contamination/washing and checking subscales were used as the dependent measures for OCD 'washing' and 'checking' subtypes in this study.

5.3.2.4 Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report questionnaire designed to measure the presence and severity of symptoms of depression in adults and adolescents. Research has shown the BDI-II has good reliability and validity. It has been reported to have high internal consistency, with coefficient alphas ranging from 0.89 to 0.93 (Beck, Steer, Ball, & Ranieri, 1996; Beck, Steer, & Brown, 1996), and a high test-retest reliability of 0.93 at 1-week intervals (Beck, Steer, & Brown, 1996). The BDI-II has also demonstrated good convergent validity, with high correlations on other measures of depression such as the Hamilton Psychiatric Rating Scale for Depression (r = 0.71; Beck, Steer, & Brown, 1996) and the Mental Health subscale of the SF-20 (r = 0.65; Arnau, Meagher, Norris, & Bramson, 2001).

The BDI-II was administered in the current study to assess levels of depression in all participants and determine whether different scores between clinical groups could account for differences observed on facilitation and/or inhibition tasks. The BDI-II total score was used as the dependent measure in this study.

5.3.2.5 State-Trait Anxiety Inventory - State Form (Speilberger, 1983). The state form of the State-Trait Anxiety Inventory (STAI-S) contains 20 items that assess feelings of apprehension, nervousness, worry and tension 'at this moment.' The STAI-S has demonstrated high internal consistency, with a median alpha coefficient of 0.93 reported for independent samples of students, military recruits, and working adults (Speilberger, 1983). In contrast, test-

retest coefficients at intervals of 1 hour, 20 days and 104 days are low, ranging from 0.16 to 0.62. This lack of stability across time is to be expected, as a valid measure of state anxiety should reflect the influence of circumstances present at the time of testing (Speilberger, 1983). The STAI-S scale has also demonstrated evidence for construct validity in studies that have found higher STAI-S scores in high stress conditions (e.g., exam, stressful movie) compared to normal conditions (Speilberger, 1983).

The STAI-S was administered to all participants in the current study to rule out the possibility that facilitation and/or inhibition deficits observed were related to levels of anxiety during the task rather than due to clinical group status. The total score on the STAI-S scale was used as the dependent measure for this study.

5.3.2.6 General Health Questionnaire - 12 (GHQ-12; Goldberg & Williams, 1988). The GHQ-12 is a 12-item self-report scale that measures short term change in mental health and levels of psychological functioning. The GHQ-12 has demonstrated good reliability and convergent validity. For example, a study investigating the use of the GHQ-12 with Australian adolescents reported high internal consistency ($\alpha = 0.88$) and good correlations with the Rosenberg Self-Esteem Scale ($r_s = 0.61$ to 0.62) and Depression Anxiety Stress Scale ($r_s = 0.54$ to 0.60), showing evidence for its construct validity (Tait, French, & Hulse, 2003). Similarly, a study in which England's National Health Service administered psychiatric interviews (Clinical Interview Schedule-Revised; CIS-R) and the GHQ-12 found the GHQ-12 had good internal consistency ($\alpha = 0.89$) and test-retest reliability at a two-week interval ($r = 0.73$; Hardy, Shapiro, Haynes, & Rick, 1999). A significant correlation between the GHQ-12 total and the CIS-R total ($r = 0.70$) was also found, demonstrating its convergent validity (Hardy et al., 1999).

The total score on the GHQ-12 was used as the dependent measure for this study. This score was utilised in this study as a screening inventory to exclude persons with psychological problems from the control group. The threshold cut-off score used for the GHQ-12 was 24, as this threshold demonstrated the best conservative estimate of minor psychiatric morbidity in Hardy et al.'s (1999) study. It is important to note that no control participants had to be excluded from the present study based on their scores on the GHQ-12, BDI-II or STAI-S. This was probably because advertisements used to recruit normal control participants specified that the current study was recruiting individuals without a history of anxiety, depression or other psychological illness to act as a control group.

5.3.2.7 Obsessive Beliefs Questionnaire - 44 (OBQ-44; Obsessive Compulsive Cognition Working Group {OCCWG}, 2005). The OBQ-44 is a 44-item self-report questionnaire that measures belief domains associated with OCD. Three factors are assessed: (i) responsibility for harm and threat estimation (assesses one's beliefs that they are able and obligated to prevent subjective negative events and one's beliefs about likelihood and severity of harm occurring), (ii) perfectionism and intolerance of uncertainty (assesses one's beliefs that imperfection cannot be tolerated and that it is necessary to be certain), and (iii) importance/control of thoughts (assesses one's beliefs that the thoughts are meaningful and dangerous and that it is necessary to control thoughts; OCCWG, 2005). All three factors have demonstrated good internal consistency with Chronbach's alpha's of 0.93, 0.93 and 0.90, respectively (Tolin, Worhunsky, & Maltby, 2006). Significant correlations between the OBQ-44 total and subscale scores of the Interpretation of Intrusions Inventory in an OCD sample has provided evidence for its convergent validity (r_s = 0.59 to 0.88; OCCWG, 2005). Furthermore, correlations between the OBQ-44 and another

measure of obsessions and compulsions (Padua Inventory-Revised) have been shown to be higher than its correlations with other measures of depression and anxiety (Beck Depression Inventory and Beck Anxiety Inventory), providing some support for the discriminate validity of the OBQ-44 (OCCWG, 2005). However, some beliefs on the OBQ-44 have also been shown to correlate with worry (as measured by the PSWQ; Myers, Fisher, & Wells, 2008).

The original OBQ was developed to measure enduring predisposing beliefs that may increase one's risk for OCD (OCCWG, 2003). Hence, the OBQ-44 was administered in the current study to determine whether abnormalities in facilitation and inhibition in OCD correlate with scores indicating proneness to OCD. If such abnormalities correlate with OBQ-44 scores it would suggest that such abnormalities may be trait-markers (instead of state-markers) of the disorder. Further, a preliminary report found levels of increased facilitation correlated with higher OBQ-44 total scores (Parker, 2007). The total score on the OBQ-44 score was used as the dependent measure for this study.

5.3.2.8 Penn-State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item self-report questionnaire that measures the intensity and excessiveness of worry, without reference to the content of the worries. The PSWQ has demonstrated good internal consistency, with alpha coefficients ranging from 0.86 to 0.93 across clinical and college samples (Molina & Borkovec, 1994), and good test-retest reliability across intervals of 2-10 weeks (r_s = 0.74 to 0.93; Molina & Borkovec, 1994). In regards to convergent validity, the PSWQ is moderately correlated with other measures of worry, such as the Worry Domains Questionnaire (r = 0.67) and the Student Worry Scale (r = 0.59; Davey, 1993). Furthermore, PSWQ scores have been reported to be higher in individuals with GAD compared

to individuals with other anxiety disorders (Brown, Antony, & Barlow, 1992), and are sensitive to changes in GAD symptomatology following cognitive-behavioural therapy (Borkovec & Costello, 1993).

The PSWQ was administered to participants in the current study to assess the severity of their worry, especially in GAD patients. The total score on the PSWQ was used as the dependent measure for this study.

5.3.2.9 Word List – In order to determine the neutral words that could be used in the modified priming tasks, all participants completed a predetermined word list (see Appendix D). This list comprised of 43 words and included neutral, general-threat (e.g., harm, tumour), panic-threat (e.g., faint, dizzy), and OCD-threat (e.g., germs, blood) words. This list was previously used by Bannon et al. (2008). Participants were instructed to rate each word on a 7-point Likert Scale, ranging from 1 (neutral) to 7 (extremely threatening). Three words rated as neutral by each participant were randomly selected for use in the facilitation and inhibition tasks of the modified priming paradigm.

5.3.3 Measures of Facilitation and Inhibition (Computer Tasks)

5.3.3.1 Modified Priming Paradigm. The modified priming paradigm used in the current study was also used in Study 1 (see chapter 4.2.1.2). Three words rated as neutral (and not personally threatening) from Bannon et al.'s (2008) previously trialed word list were randomly selected and presented in the facilitation and inhibition task. Participants completed the facilitation and inhibition task at an ISI of 1000ms.

5.3.3.2 Modified Go/Nogo Paradigm. The modified Go/Nogo paradigm used in the current study was also administered in Study 1 (see chapter 4.3.1.2). Participants completed the Go/Nogo paradigm twice, at two ISI conditions: 600ms and 1000ms.

5.3.4 Procedure

The study was approved by the Sydney West Area Health Service and University of Wollongong Ethics' committees. Written informed consent was obtained from all participants prior to their participation. In regards to clinical participants, the ADIS-IV was initially administered to confirm clinical diagnoses of OCD and GAD. The Y-BOCS was also completed with those who had an OCD diagnosis. Excluding those participants who were patients at Westmead Hospital's Anxiety Treatment and Research Unit, participants were compensated for their time and travel expenses by a token reimbursement of $30.

All participants completed the BDI-II, STAI-S, GHQ, OBQ, PSWQ and Bannon et al.'s (2008) word list. In addition, OCD participants completed the PI-WSUR (see Table 5.1). Four computer tasks were administered to measure facilitation and inhibition. Participants were seated approximately 60cm from the laptop computer screen. The modified facilitation and inhibition priming tasks and two modified Go/Nogo tasks were completed within a single session. Presentation of these computer tasks were randomised and counterbalanced to control for order and fatigue effects. Completion of the four tasks took approximately 45 minutes.

Table 5.1. *Questionnaires Completed by Normal Controls, OCD, and GAD groups*

Questionnaire	Normal Controls	OCD	GAD
ADIS-IV	✗	✓	✓
Y-BOCS	✗	✓	✗
PI-WSUR	✗	✓	✗
BDI-II	✓	✓	✓
STAI-S	✓	✓	✓
GHQ-12	✓	✓	✓
OBQ-44	✓	✓	✓
PSWQ	✓	✓	✓
Word List	✓	✓	✓

Note. ADIS-IV = Anxiety Disorders Interview Schedule-IV; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; PI-WSUR = Padua Inventory-Washington State University Revision; BDI-II = Beck Depression Inventory-II; STAI-S = State Trait Anxiety Inventory – State Form; GHQ-12 = General Health Questionnaire-12; OBQ-44 = Obsessive Beliefs Questionnaire-44; PSWQ = Penn State Worry Questionnaire.

5.3.5 Statistical Analyses

Because statistical analyses for the current study are complex and vary for hypotheses and paradigms, analyses and results are best presented together. Two-tailed tests were employed to determine statistical significance.

5.4 Results

5.4.1 Demographic and Clinical Characteristics

To determine whether groups differed on demographic and clinical characteristics one-way analysis of variance (ANOVA) tests were conducted. This involved dependent variables: (i) demographics (age, sex, total years of education, handedness); and (ii) clinical characteristics (BDI-II, STAI-S, GHQ, OBQ, PSWQ). The independent variable was group status (OCD, GAD,

and normal control). Planned comparisons were used to identify group differences. The group data for participants' demographic and clinical characteristics are presented in Table 5.2.

Demographic variables: The group means did not differ significantly for any of the demographic variables: sex, $F(2, 57) = 0.23$, $p = 0.80$; age, $F(2, 57) = 0.39$, $p = 0.68$; years of education, $F(2, 57) = 0.14$, $p = 0.87$; and handedness, $F(2, 57) = 0.91$, $p = 0.41$. **Clinical variables:** In regards to clinical characteristics, a significant group difference was observed for levels of depression, $F(2, 57) = 17.30$, $p < 0.001$, state anxiety, $F(2, 57) = 26.97$, $p < 0.001$, worry, $F(2, 57) = 48.44$, $p < 0.001$, obsessive beliefs, $F(2, 57) = 16.07$, $p < 0.001$, and psychological functioning, $F(2, 57) = 11.29$, $p < 0.001$.

Table 5.2. *Group Demographic and Clinical Characteristics*

Characteristics	**Normal Controls**	**OCD**	**GAD**
	***M* (SD)**	***M* (SD)**	***M* (SD)**
Female, Male	11, 10	13, 8	11, 7
Age	32.52 (14.29)	34.10 (10.47)	36.28 (15.28)
Years of Education	15.05 (2.22)	14.62 (3.11)	14.78 (2.39)
Handedness (Right, Left)	19, 2	20, 1	18
Y-BOCS	NA	23.38 (8.15)	NA
PI-WSUR	NA	30.71 (17.77)	NA
BDI-II	04.05 (3.73)	17.38 (11.00)	18.00 (9.33)
STAI-S	27.81 (7.19)	43.05 (11.01)	48.78 (9.36)
GHQ-12	08.62 (3.12)	15.10 (6.43)	16.61 (6.90)
OBQ-44	110.19 (26.93)	166.52 (64.64)	190.28 (35.39)
PSWQ	36.10 (9.58)	56.29 (9.97)	66.11 (9.82)

Note. Y-BOCS = Yale-Brown Obsessive Compulsive Scale; PI-WSUR = Padua Inventory-Washington State University Revision; BDI-II = Beck Depression Inventory-II; STAI-S = State Trait Anxiety Inventory – State Form; GHQ-12 = General Health Questionnaire-12; OBQ-44 = Obsessive Beliefs Questionnaire-44; PSWQ = Penn State Worry Questionnaire.

Clinical groups vs. Normal Controls: As expected, planned comparisons revealed the OCD group displayed higher levels of depression ($p < 0.001$), state anxiety ($p < 0.001$) and worry ($p < 0.001$), a significantly greater number of obsessive beliefs ($p = 0.001$), and lower levels of psychological functioning ($p = 0.001$) than the normal control group. Similarly, GAD participants reported higher levels of depression ($p < 0.001$), state anxiety ($p < 0.001$) and worry ($p < 0.001$), a greater number of obsessive beliefs ($p < 0.001$), and lower levels of psychological functioning ($p < 0.001$) compared to normal controls.

OCD vs. GAD: While the OCD group reported significantly lower levels of worry than the GAD group ($p = 0.008$), no other group differences were found between the OCD and GAD groups (depression, $p = 1.00$; state anxiety, $p = 0.18$; obsessive beliefs, $p = 0.34$; and psychological functioning, $p = 1.00$).

5.4.2 Modified Priming Paradigm

5.4.2.1 Speed of Processing

To determine if there was a significant difference in baseline speed of processing between the groups, two one-way ANOVAs were conducted. The dependent variables were the facilitation control response time and inhibition control response time. The independent variable was group status (OCD, GAD, and normal control).

Group differences for baseline speed of processing within the facilitation task were not significant, for facilitation control RT $F(2, 57) = 0.43$, $p = 0.66$. Similarly, no significant group differences were found for baseline speed of processing within the inhibition task, for inhibition control RT $F(2, 57) = 0.26$, $p = 0.78$.

5.4.2.2 Facilitation

To assess whether OCD participants exhibited stronger facilitation effects compared to normal control and GAD groups, the mean facilitation differential and mean number of errors were subjected to Kruskal-Wallis tests. Such facilitation data were analysed with non-parametric tests because these variables were not normally distributed and the facilitation differential displayed a large amount of variance in all groups. With regard to groups, two planned comparisons using Mann-Whitney U-Tests (OCD vs. NC, and OCD vs. GAD) were employed to examine whether deficits occurred in and were specific to OCD. As contrasts were ***planned*** and based on a single degree of freedom (for effect), adjustment for alpha was neither required nor made (Tabachnick & Fidell, 1989).

The group data for the facilitation task are presented in Table 5.3, and plotted in Figure 5.1. It is evident from Figure 5.1 that all groups displayed a faster mean response time to the positive priming condition compared to the control condition. A significant group difference was found between the mean ranks for the facilitation differential (OCD = 38.52; normal control = 27.33; GAD = 24.83; χ^2 (2) = 7.02, p = 0.03) and commission errors (OCD = 37.90; normal control = 25.12; GAD = 28.14; χ^2 (2) = 6.89, p = 0.03). However, no group differences were observed in regards to omission errors (OCD = 33.69; normal control = 28.93; GAD = 28.61; χ^2 (2) = 1.99, p = 0.37).

Table 5.3. *Response Times (milliseconds) and Number of Errors on the Facilitation Task*

Condition	Normal Controls *M* (SD)	OCD *M* (SD)	GAD *M* (SD)
Control	551.85 (76.12)	537.64 (65.64)	557.66 (68.85)
Positive Priming	540.99 (83.63)	507.51 (67.83)	551.00 (64.07)
Facilitation	10.85 (38.27)	30.13 (24.77)	6.66 (27.70)
Omission Errors	0.19 (0.40)	0.47 (0.81)	0.22 (0.55)
Commission Errors	0.81 (1.36)	1.90 (1.89)	1.22 (2.37)

Note. Facilitation = control RT – positive priming RT (higher positive values indicate stronger facilitation).

The OCD group displayed significantly stronger facilitation than normal control (OCD = 25.14; normal control = 17.86; $Z = -1.92$, $p = 0.05$) and GAD groups (OCD = 24.38; GAD = 14.89; $Z = -2.59$, $p = 0.01$). Similarly, the OCD group produced significantly more commission errors than normal controls (OCD = 25.81; normal control = 17.19; $Z = -2.41$, $p = 0.02$), and had values that approached significance when compared to the GAD group (OCD = 23.10; GAD = 16.39; $Z = -1.90$, $p = 0.07$)

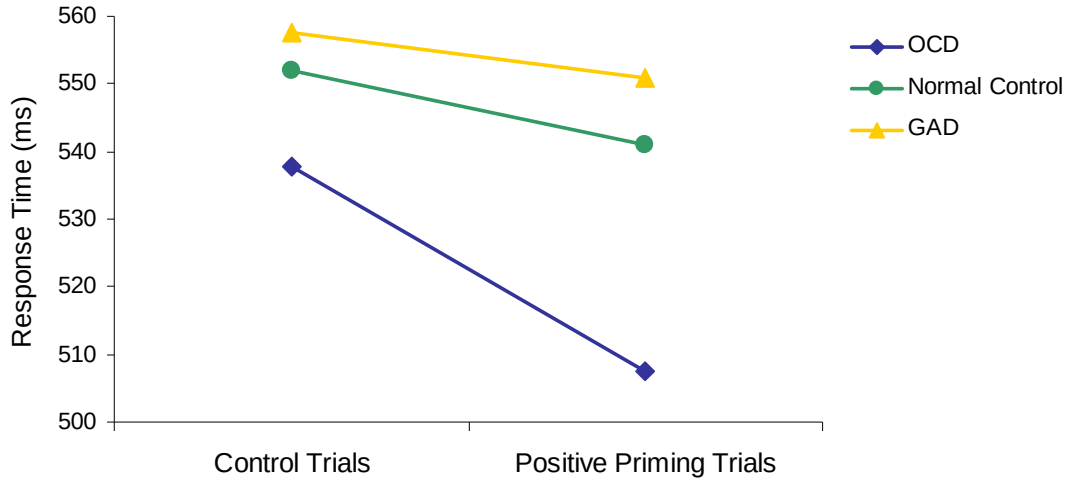


Figure 5.1. **Mean response times (milliseconds) on the facilitation task for OCD, normal control and GAD groups.**

5.4.2.3 Inhibition

To determine whether OCD participants displayed inhibition deficits compared to normal control and GAD groups, the mean inhibition differential and mean number of errors were subjected to Kruskal-Wallis tests. The data were analysed with non-parametric tests because error data were not normally distributed and the inhibition differential displayed a large amount of variance in all groups. The group data for the inhibition task are presented in Table 5.4, and plotted in Figure 5.2.

Table 5.4. *Response Times (milliseconds) and Number of Errors on the Inhibition Task*

Condition	**Normal Controls**	**OCD**	**GAD**
	***M* (SD)**	***M* (SD)**	***M* (SD)**
Control	606.65 (111.92)	627.92 (122.82)	604.18 (112.74)
Negative Priming	593.68 (112.16)	597.77 (88.96)	611.58 (121.29)
Inhibition	12.97 (68.34)	30.15 (86.27)	-7.40 (51.94)
Omission Errors	1.00 (1.87)	2.38 (4.14)	0.56 (1.20)
Commission Errors	4.95 (4.39)	7.62 (6.03)	4.78 (5.23)

Note. Inhibition = control RT – negative priming RT (higher negative values indicates stronger inhibition, positive values indicate facilitation).

It is evident from Figure 5.2 that only the GAD group displayed a mean response cost in the negative priming condition relative to the control condition. As the mean inhibition differential is positive (instead of negative) for OCD and normal control groups, it suggests they were *faster* to respond in the negative priming condition relative to the control condition.

No significant group differences were observed between the mean ranks for the inhibition differential (OCD = 33.14; normal control = 31.98; GAD = 25.69; χ^2 (2) = 1.99, p = 0.37), commission errors (OCD = 36.07; normal control = 28.81; GAD = 25.97; χ^2 (2) = 3.59, p =

0.17), or omission errors (OCD = 36.24; normal control = 29.14; GAD = 25.39; χ^2 (2) = 4.75, p = 0.09).

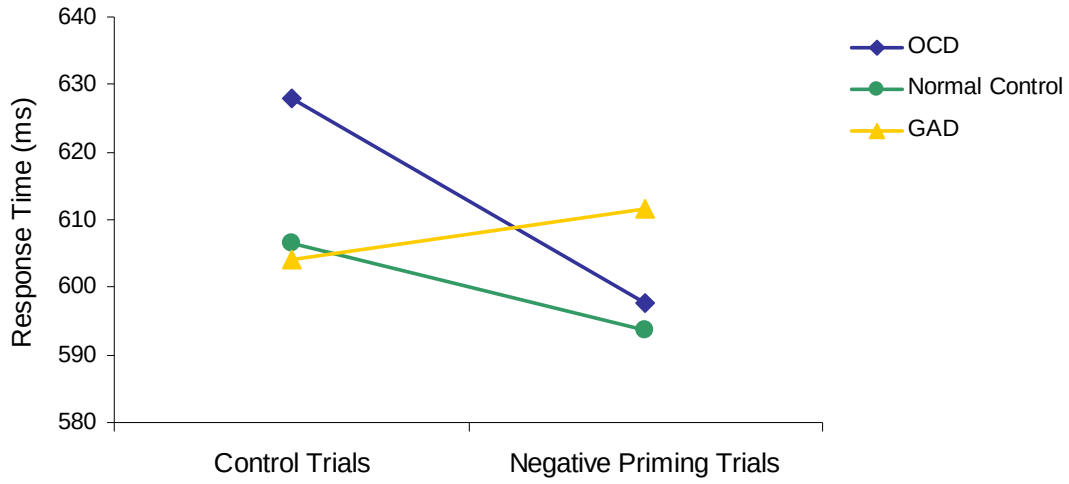


Figure 5.2. **Mean response times (milliseconds) on the inhibition task for OCD, normal control and GAD groups.**

5.4.3 Modified Go/Nogo Paradigm

5.4.3.1 Speed of Processing

To determine if there was a significant difference in baseline speed of processing between the groups, baseline response times to initial (i) Go1 and (ii) Go2 stimuli in a sequence of Go stimuli were each subjected to 3 Group (OCD, normal control, GAD) X 2 ISI (600ms, 1000ms) mixed ANOVAs, with repeated measures for ISI. No significant group difference was found for baseline response times to the initial Go1 stimulus, $F(2, 57) = 0.07$, $p = 0.94$, or the initial Go2 stimulus, $F(2, 57) = 0.42$, $p = 0.66$

5.4.3.2 Facilitation

Go1 stimulus repetitions:

In the modified Go/Nogo paradigm, facilitation effects are accrued through stimulus-response repetitions (i.e., when repetitions of identical stimuli require the same response). Hence, sequence analyses related to facilitation accrued through Go1 stimulus repetitions was initially conducted. The group data for Go1 stimulus repetitions are presented in Table 5.5.

Table 5.5. *Response Times (milliseconds) to Repetitions of Go Stimuli Following Nogo2 Stimuli*

Stimulus Sequence	Normal Controls *M* (SD)	OCD *M* (SD)	GAD *M* (SD)
		ISI = 600ms	
N2G	417.82 (44.12)	426.06 (64.19)	437.54 (63.99)
N2GG	408.16 (54.73)	391.52 (48.90)	421.66 (49.46)
N2GGG	398.25 (49.65)	374.76 (44.76)	391.77 (42.77)
		ISI = 1000ms	
N2G	463.58 (54.71)	452.36 (72.33)	454.09 (64.38)
N2GG	430.75 (55.86)	415.52 (48.01)	440.35 (61.46)
N2GGG	412.01 (48.93)	401.08 (50.15)	423.10 (40.90)

Note. N2 = Nogo2; G = Go1; ms = milliseconds; ISI = interstimulus interval; RT values are for the highlighted stimulus, preceding stimuli are reported to explicate facilitation effects as a function of stimulus repetitions.

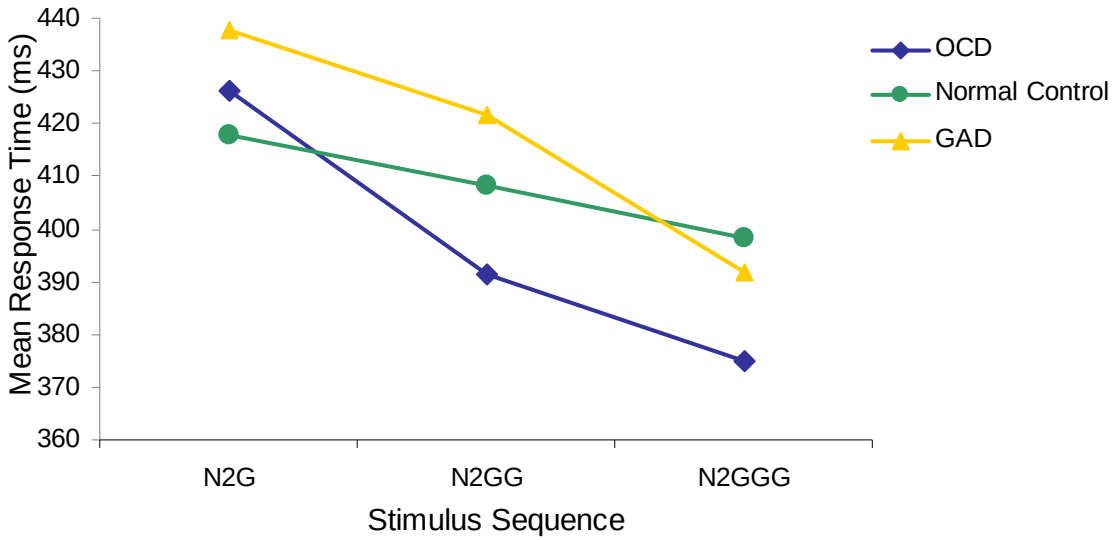
To assess whether the OCD group exhibited stronger facilitation effects compared to normal control and GAD groups, Go1 stimulus repetitions following Nogo2 stimuli were subjected to a 3 Group (OCD, normal control, GAD) X 3 Stimulus (N2G, N2GG, N2GGG) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. Due to a small number of trials in stimulus types N2GGGG and N2GGGGG ($n \leq 3$), only three Stimulus levels were included in this analyses. For the Stimulus factor two planned contrasts were employed: the linear contrast determined whether repetitions of Go1 stimuli produced linear changes to response times, while the quadratic contrast assessed non-linear changes in

Stimulus, such as X- or U-shaped functions. For the group factor, 2 planned contrasts (OCD vs. NC; OCD vs. GAD) were conducted. As contrasts were ***planned*** and based on a single degree of freedom (for effect), adjustment for alpha was neither required nor made (Tabachnick & Fidell, 1989).

The group data for mean RTs to Go1 stimulus repetitions sequenced after the presentation of Nogo2 stimuli are plotted in Figure 5.3. Go1 stimulus repetitions produced a decrease in response time, as indicated by a significant linear effect, $F(1, 57) = 46.82$, $p < 0.001$. A significant effect was also found for ISI condition, with longer response times produced at an ISI of 1000ms compared to 600ms, $F(1, 57) = 45.22$, $p < 0.001$. In addition, the Stimulus X ISI X Group interaction was significant, $F(2, 57) = 5.79$, $p < 0.01$, suggesting that between-group effects were influenced by Stimulus and ISI factors. No other significant effects were observed.

To clarify between-group differences observed in the 3-way interaction, two separate group comparisons (OCD vs. NC; OCD vs. GAD) were conducted for 3 Stimulus (N2G, N2GG, N2GGG) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. **OCD vs. normal controls**: The Stimulus X ISI X Group interaction was significant, $F(1, 40) = 4.92$, $p = 0.03$. The results indicate that facilitation gains were larger for OCD participants (vs. normal controls) with this group differential being more pronounced at the shorter ISI (OCD vs. normal controls, 34.54ms vs. 9.66ms) as compared with the difference at the longer ISI (OCD vs. normal control, 36.84ms vs. 32.83ms; see Figure 5.4). The overall between groups effect, $F(1, 40) = 0.66$, $p = 0.42$, Stimulus X Group, $F(1, 40) = 1.65$, $p = 0.21$, and ISI X Group effects, $F(1, 40) = 0.04$, $p = 0.84$, were not significant.

(A)



(B)

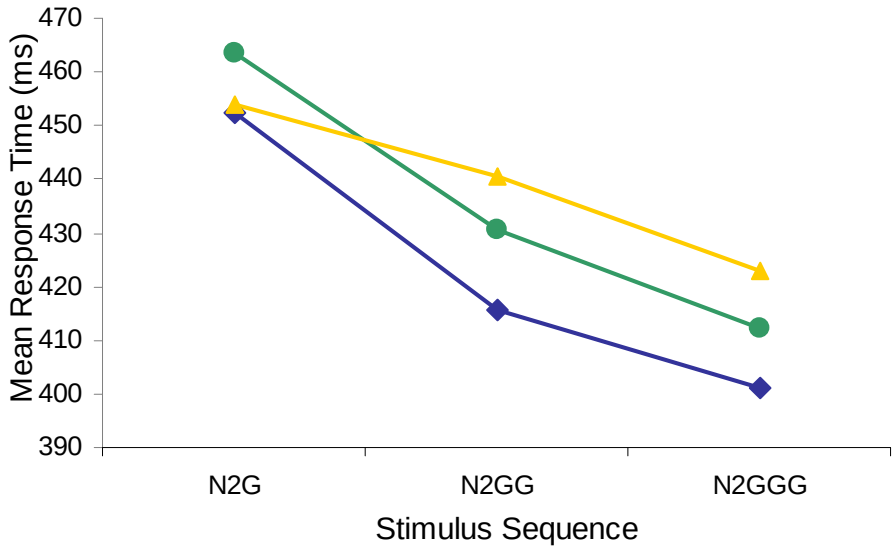
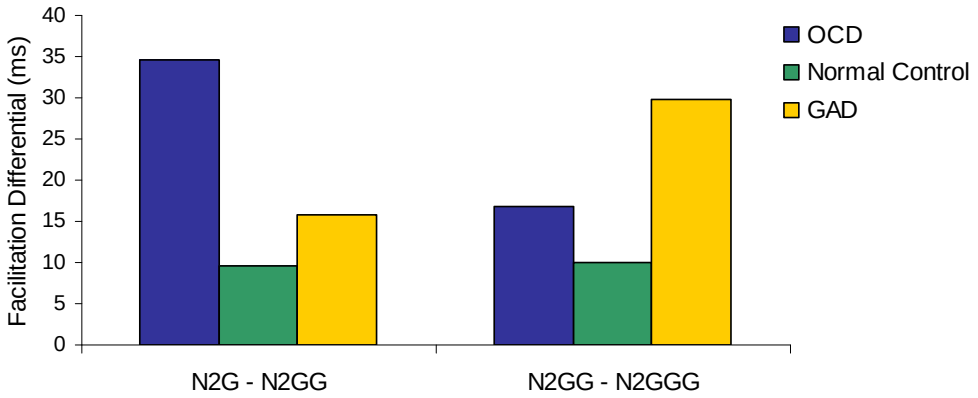


***Figure 5.3*. Mean response times (milliseconds) to Go1 (G) stimulus repetitions following a Nogo2 (N2) stimulus (N2G is the first Go1 stimulus in a sequence, and N2GGG is the third) for OCD, normal control, and GAD groups at (A) 600ms and (B) 1000ms interstimulus intervals.**

(A)



(B)

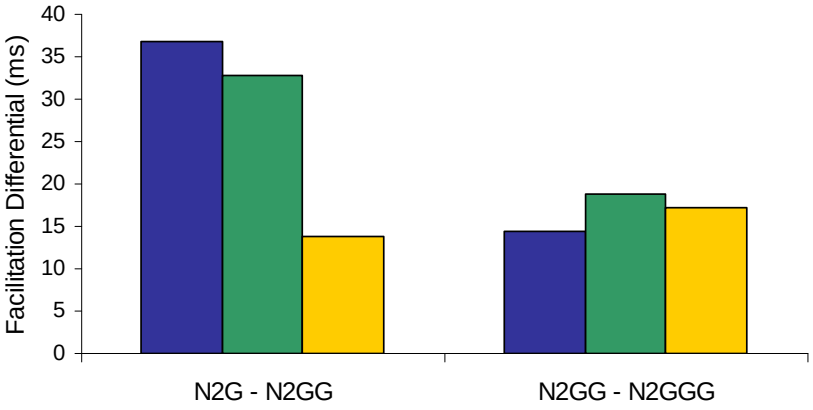


***Figure 5.4*. Mean facilitation differential (milliseconds) for initial and latter Go1 stimulus repetitions (i.e., N2G – N2GG and N2GG – N2GGG) for OCD, normal control, and GAD groups at (A) 600ms and (B) 1000ms interstimulus intervals.**

OCD vs. GAD: Facilitation differences between the OCD and GAD group was examined by a 2 Group (OCD, GAD) X 3 Stimulus (N2G, N2GG, N2GGG) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. The quadratic effect for the Stimulus X Group interaction was approaching significance, $F(1, 37) = 2.68$, $p = 0.11$, suggesting stronger facilitation for the OCD group at initial Go1 stimulus repetitions than for

latter Go1 stimulus repetitions, as indicated in Figure 5.4. The between groups effect, $F(1, 37) = 1.52$, $p = 0.23$, Stimulus X Group, $F(1, 37) = 0.59$, $p = 0.45$, ISI X Group, $F(1, 37) = 0.12$, $p = 0.73$, and Stimulus X ISI X Group effects, $F(1, 37) = 0.93$, $p = 0.34$, were not significant.

Go2 then Go1 stimulus repetitions

Whilst facilitation is accrued through stimulus–response repetitions, if both the stimulus *and* response create a template after their initial presentation, then the enhanced facilitation observed in OCD could be because of a stronger response-template or a stronger stimulus-template. Hence, to assess whether the enhanced facilitation observed in OCD is the result of stimulus repetition or response repetition, analyses were conducted on repetitions of different Go stimuli (i.e., Go2 then Go1). That is, analyses were conducted on Go1 repetitions following the presentation of a different Go stimulus (i.e.Go2) to determine whether the enhanced facilitation gain observed in OCD is due to response repetition (e.g., Go2, Go1) or stimulus repetition (e.g., Go1, Go1). This analyses involved a 3 Group (OCD, normal control, GAD) X 6 Stimulus (G2, G2G, G2GG, G2GGG, G2GGGG, G2GGGGG) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. The group data for Go2 stimuli and subsequent Go1 stimulus repetitions are presented in Table 5.6 and Figure 5.5.

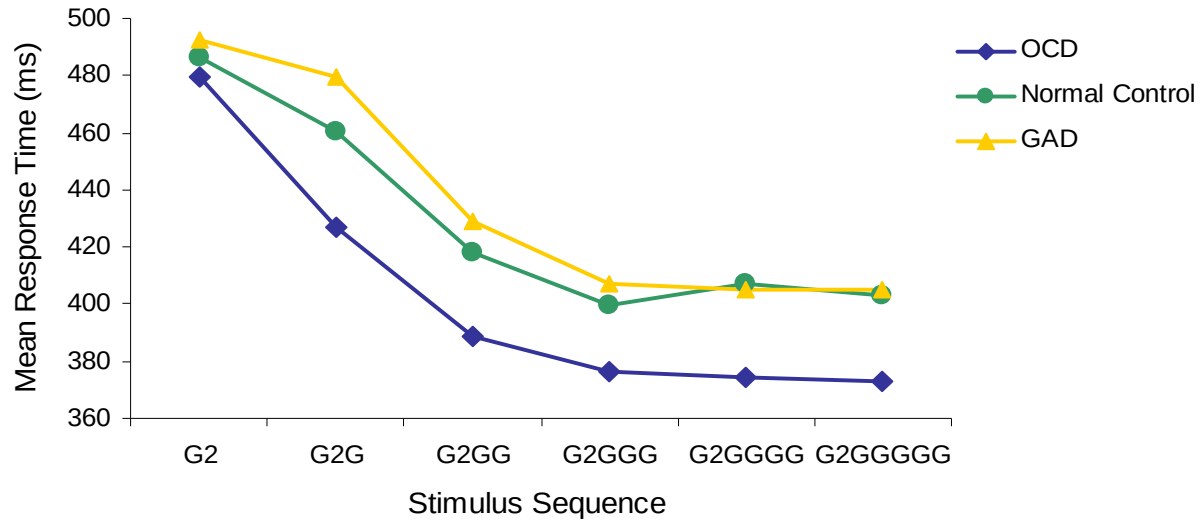
Table 5.6. *Response Times (milliseconds) to Repetitions of Go Stimuli*

Stimulus Sequence	Normal Controls *M* (SD)	OCD *M* (SD)	GAD *M* (SD)
		ISI = 600ms	
G2	486. 18 (45.24)	479.48 (55.38)	492.51 (43.00)
G2G	460. 49 (65.41)	426.90 (48.65)	479.52 (61.98)
G2GG	417.84 (62.29)	388.43 (40.05)	428.72 (61.09)
G2GGG	399.51 (48.40)	376.33 (33.84)	407.43 (52.97)
G2GGGG	406.82 (48.85)	374.65 (35.89)	404.91 (54.11)
G2GGGGG	403.12 (48.11)	372.67 (40.31)	405.32 (50.26)
		ISI = 1000ms	
G2	521.62 (59.29)	523.68 (67.46)	539.48 (49.10)
G2G	498.35 (67.15)	462.55 (52.89)	499.81 (63.03)
G2GG	443.91 (53.16)	421.59 (49.28)	432.55 (50.39)
G2GGG	428.20 (50.58)	400.76 (47.32)	427.77 (58.92)
G2GGGG	427.91 (55.21)	410.28 (60.28)	437.14 (75.27)
G2GGGGG	423.06 (54.66)	401.19 (49.71)	432.46 (69.58)

Note. G = Go1; G2 = Go2; ms = milliseconds; ISI = interstimulus interval; RT values are for the highlighted stimulus, preceding stimuli are reported to explicate facilitation effects as a function of stimulus repetitions.

Go stimulus repetitions produced a decrease in response time, as indicated by a significant linear effect, $F(1, 57) = 306.31$, $p < 0.001$. The reduction in response time was larger after shorter (e.g., G2, G2G) sequence repetitions compared with longer sequence repetitions (e.g., G2GGGG, G2GGGGG), resulting in a significant quadratic contrast $F(1, 57) = 211.60$, $p < 0.001$ (see Figure 5.5). A significant effect was also found for ISI condition, with longer response times produced at an ISI of 1000ms compared to 600ms, $F(1, 57) = 50.34$, $p < 0.001$. In addition, the quadratic effects for Stimulus X ISI, $F(1, 57) = 7.76$, $p < 0.01$, and Stimulus X ISI X Group, $F(2, 57) = 3.69$, $p = 0.03$, were significant. Such results and Figure 5.5 indicates that facilitation is stronger earlier in the sequence, to initial Go repetitions, and plateaus off at latter repetitions. This pattern is modulated by group and ISI conditions.

(A)



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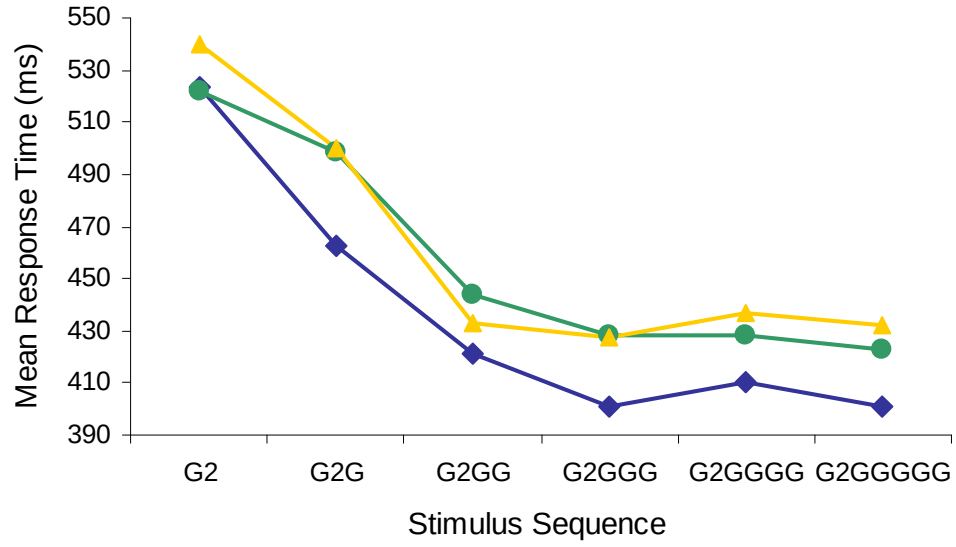

***Figure 5.5*. Mean response times (milliseconds) to Go1 (G) stimulus repetitions following a Go2 (G2) stimulus (G2 is the first Go stimulus in a sequence, and G2GGGGG is the sixth) for OCD, normal control, and GAD groups at (A) 600ms and (B) 1000ms interstimulus intervals.**

As Figure 5.5 suggests group differences are more prominent between the first two Go repetitions and then plateaus off, to disentangle the 3-way interactions we conducted two separate 2 Group comparisons (OCD vs. NC; OCD vs. GAD) for 2 Stimulus (Go2, Go1) X 2 ISI

(600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. The results are presented in Figure 5.6.

OCD vs. normal controls: The Stimulus X Group effect was significant, $F(1, 40) = 6.39$, $p = 0.02$ (see Figure 5.6). As no between group differences were found for baseline speed of processing to initial Go2 stimuli, this finding indicates that OCD participants are responding significantly faster to the first Go response repetition (i.e., G2G), compared to normal controls. In contrast, the ISI X Group, $F(1, 40) = 0.20$, $p = 0.66$, and ISI X Stimulus X Group, $F(1, 40) = 0.72$, $p = 0.40$, effects were not significant.

OCD vs. GAD: The 2 Group (OCD, GAD) X 2 Stimulus (G2, G2G) X 2 ISI (600ms, 1000ms) mixed ANOVA, yielded a significant Stimulus X Group effect, $F(1, 37) = 5.94$, $p = 0.02$ (see Figure 5.6). As no between group differences were found for baseline speed of processing to initial Go2 stimuli, this finding indicates that OCD participants are responding significantly faster to the first Go response repetition (i.e., G2G), compared to GAD participants. In contrast, the ISI X Group, $F(1, 37) = 0.56$, $p = 0.46$, and ISI X Stimulus X Group, $F(1, 40) = 1.59$, $p = 0.22$, effects were not significant.

To determine whether these group differences were present later in the sequence, a 2 Group (OCD, normal controls or GAD) X 5 Stimulus (G2G, G2GG, G2GGG, G2GGGG, G2GGGGG) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors, was conducted for OCD vs. normal controls and OCD vs. GAD. None of the group interactions were significant for OCD vs. normal control or OCD vs. GAD.

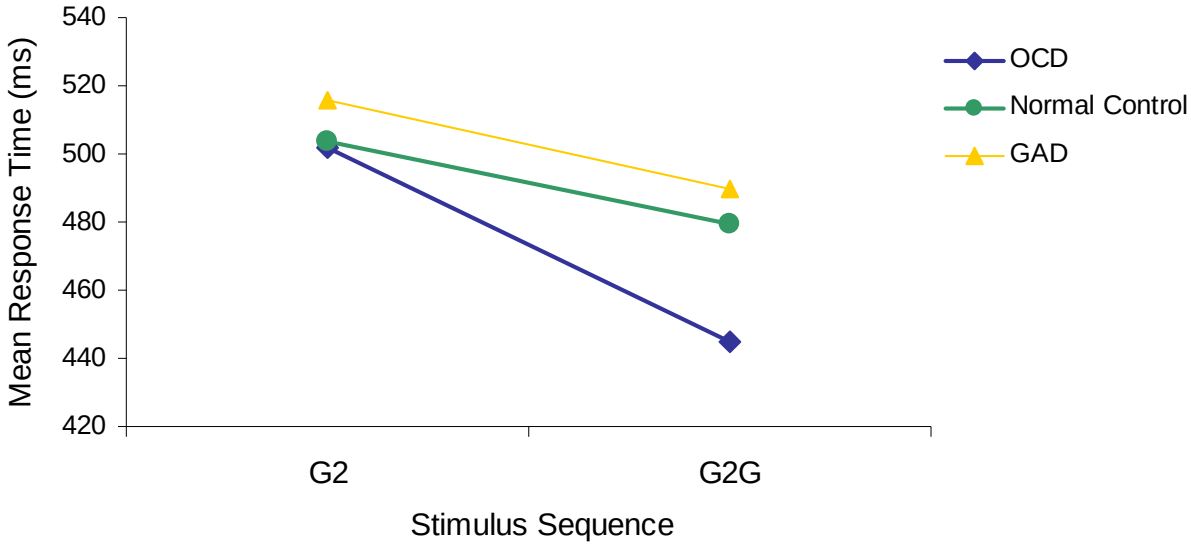

Figure 5.6. **Mean response times (milliseconds) to Go2 (G2) and subsequent Go1 (G2G) stimuli averaged across interstimulus interval conditions for OCD, normal control, and GAD groups.**

Summary: OCD participants demonstrate stronger facilitation gains to the first Go response repetition (even when Go stimuli are different), compared to both normal control and GAD groups (see Figure 5.6). Go stimulus repetitions produced a decrease in response time across all groups. This reduction was larger after initial (e.g., G2, G2G) sequence repetitions and plaetued at latter Go repetitions (e.g., G2GGGG, G2GGGGG). In addition, faster response times were produced at the shorter ISI (600ms) across all groups.

Commission errors during facilitation task:

To assess the effect of the number of preceding Go1 (G) stimuli upon accuracy to Nogo1 (N) stimuli, commission error percentage was analysed using a 3 Group (OCD, normal control, GAD) X 4 Stimulus (GN, GGN, GGGN, GGGGN) X 2 ISI (600, 1000ms) mixed ANOVA, with repeated measures for the Stimulus and ISI factors. Due to a small number of trials in stimulus

types GGGGN and GGGGGN, such data were combined into one group. Planned contrasts assessed linear and quadratic effects for commission errors to Nogo stimuli based on the number preceding Go1 stimulus repetitions. Because error data were not normally distributed, commission error data were submitted to a square root transformation. The skewness was acceptable after square root transformations. Planned contrasts assessed linear and quadratic effects for commission errors to Nogo stimuli based on the number preceding Go1 stimulus repetitions. The aforementioned mixed ANOVA was performed on both transformed and raw data. Statistical analyses on transformed and raw data were consistent with each other; statistics for transformed data are reported in text, whereas raw data are presented in tables and figures to facilitate better understanding of reported values.

The group data for commission error percentage to Nogo1 stimuli as a function of preceding Go1 stimulus repetitions are presented in Table 5.7. A significant effect was observed for ISI condition, with more commission errors produced at an ISI of 600ms compared to 1000ms, $F(1, 57) = 5.03$, $p = 0.03$. A significant linear effect was found for Stimulus, $F(1, 57) = 18.37$, $p < 0.001$, with the means indicating that the percentage of commission errors to Nogo1 stimuli decreased as the number of Go1 stimulus repetitions preceding the presentation of Nogo1 stimuli increased (see Figure 5.7). No significant quadratic effect was observed for Stimulus, $F(1, 57) = 0.82$, $p = 0.37$.

Table 5.7. *Commission Error Percentage to Nogo1 Stimuli as a Function of Preceding Go1 Stimulus Repetitions*

Stimulus	Normal Controls *M* (SD)	OCD *M* (SD)	GAD *M* (SD)
		ISI = 600ms	
GN	15.99 (16.27)	20.07 (14.22)	17.46 (15.34)
GGN	13.89 (17.15)	18.25 (15.28)	14.35 (9.82)
GGGN	14.81 (18.37)	17.99 (15.10)	12.96 (11.59)
GGGGN	11.64 (15.90)	16.40 (18.13)	14.81 (16.61)
		ISI = 1000ms	
GN	14.63 (11.84)	17.35 (12.07)	11.90 (10.10)
GGN	21.03 (17.99)	16.67 (13.44)	10.19 (11.27)
GGGN	14.81 (15.45)	13.76 (15.28)	9.88 (10.70)
GGGGN	10.58 (14.26)	10.58 (12.41)	7.41 (10.08)

Note. G = Go1; N = Nogo1; ms = milliseconds; ISI = interstimulus interval; commission error percentage are reported for the highlighted stimulus.

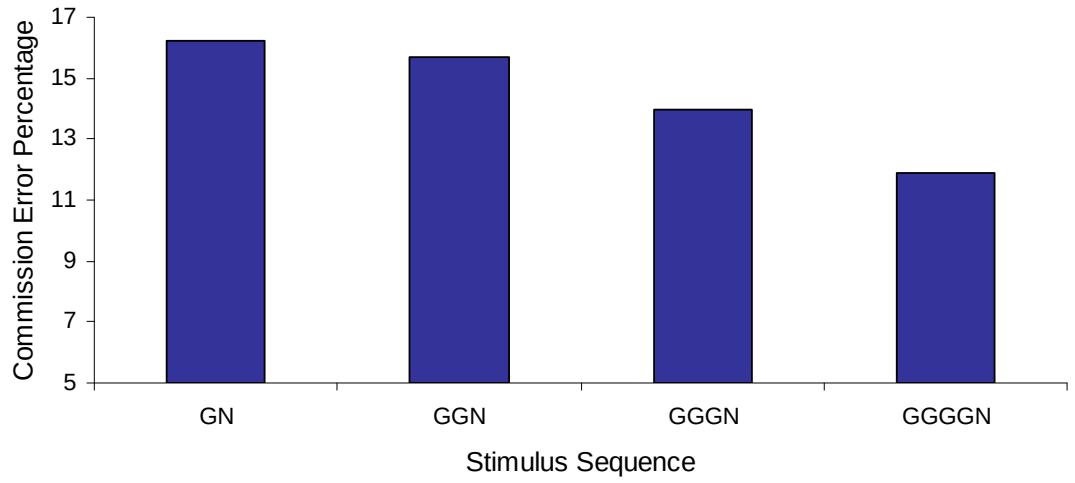

Figure 5.7. **Mean commission error percentage to Nogo1 stimuli as a function of the number of preceding Go1 stimuli averaged across interstimulus interval conditions.**

Group effects: Although there was no overall group differences, $F(2, 57) = 0.75$, $p = 0.48$, the ISI X Group effect was significant, $F(2, 57) = 5.00$, $p = 0.01$. Further analyses revealed that the OCD group produced more commission errors at an ISI of 600ms compared to 1000ms,

whereas the normal control group displayed the opposite effect (see Figure 5.8). No difference was observed between the OCD and GAD groups, and no other effects were significant.

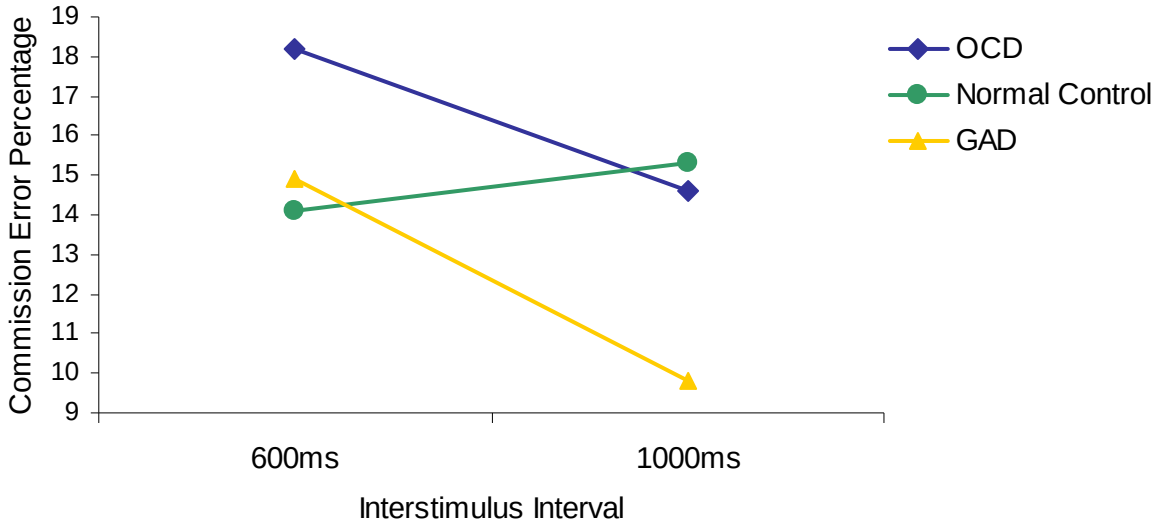

***Figure 5.8*. Mean commission error percentage to Nogo1 stimuli at 600ms and 1000ms interstimulus intervals for OCD, normal control, and GAD groups.**

5.4.3.3 Inhibition

Response time and omission error rate for X-Go and X-Go control stimuli

In the Go/Nogo task the X-Go stimulus was used as a measure of inhibition. That is, it was predicted X-Go stimuli would produce a delayed mean RT compared to the mean RT of a non-inhibited (equi-probable control) stimulus (i.e. Nogo1 then Go2). Only X-Go and Go2 trials after which participants correctly suppressed a response to the preceding Nogo1 stimulus were analysed. To assess whether the OCD group displayed inhibition deficits compared to the normal control and GAD groups, mean RTs and omission errors to X-Go and X-Go control stimuli were subjected to a 3 Group (OCD, normal control, GAD) X 2 Stimulus type (X-Go control, X-Go) X 2 ISI (600ms, 1000ms) mixed ANOVA, with repeated measures for the Stimulus type and ISI factors. Because error data were not normally distributed, omission error data were submitted to

a square root transformation that improved the distribution. The aforementioned mixed ANOVA was performed on both transformed and raw data. Statistical analyses on transformed and raw data were consistent with each other; statistics for transformed data are reported in text, whereas raw data are presented in tables to facilitate better understanding of reported values.

Mean RTs for X-Go and X-Go control stimuli are presented in Table 5.8. Significant effects were found for Stimulus type, $F(1, 57) = 172.75$, $p < 0.001$, ISI, $F(1, 57) = 81.12$, $p < 0.001$, and Stimulus type X ISI, $F(1, 57) = 51.43$, $p < 0.001$. The results together indicate that overall faster response times are produced at the shorter ISI (600ms) and to X-Go stimuli compared to the control, and that longer response times were produced for X-Go control stimuli at 1000ms ISI.

Groups effects: The between groups effect was not significant, $F(2, 57) = 0.14$, $p = 0.87$, and interaction effects with group were also not significant.

Table 5.8. *Response Times (milliseconds) for X-Go and X-Go Control Stimuli*

Stimulus Type	**Normal Controls** ***M*** **(SD)**	**OCD** ***M*** **(SD)**	**GAD** ***M*** **(SD)**
		ISI = 600ms	
X-Go Control	476.41 (44.85)	471.57 (64.80)	482.76 (47.99)
X-Go	412.98 (67.28)	409.06 (56.56)	425.15 (58.03)
		ISI = 1000ms	
X-Go Control	523.09 (59.79)	524.60 (62.14)	532.31 (51.88)
X-Go	425.36 (69.49)	431.27 (62.34)	427.72 (57.76)

Note. ms = milliseconds; ISI = interstimulus interval.

The group data for the percentage of omission errors to X-Go and X-Go control stimuli are presented in Table 5.9. In regards to omission errors, a significant effect was found for Stimulus type $F(1, 57) = 13.85$, $p < 0.001$, with the means indicating that participants produced significantly more omission errors to X-Go stimuli compared to X-Go control stimuli. A

significant effect for ISI was also observed, with omission error percentage lower at 1000ms compared to 600ms, $F(1, 57) = 13.50$, $p = 0.001$.

Group effects: Group differences were not significant, $F(2, 57) = 1.62$, $p = 0.21$, and no other significant effects were observed.

Table 5.9. *Omission Error Percentage for X-Go and X-Go Control Stimuli*

Stimulus Type	Normal Controls *M* (SD)	OCD *M* (SD)	GAD *M* (SD)
		ISI = 600ms	
X-Go Control	2.20 (3.41)	6.50 (7.82)	3.30 (5.79)
X-Go	3.90 (6.31)	8.00 (11.40)	6.30 (8.12)
		ISI = 1000ms	
X-Go Control	0.90 (3.09)	2.40 (4.46)	1.80 (3.86)
X-Go	3.20 (3.56)	3.70 (6.20)	3.30 (4.07)

Note. ms = milliseconds; ISI = interstimulus interval.

5.4.4 Relationship between Cognitive Tasks

To determine whether there was a relationship between the facilitation differentials for the modified Go/Nogo task at 600ms and 1000ms ISIs, Spearman's rho correlations were calculated. Such data were analysed with non-parametric tests because these variables were not normally distributed. The facilitation variables included the facilitation differential for: (i) stimulus-response priming (i.e., NG – NGG) at 600ms and 1000ms ISIs; and (ii) response priming (i.e., G2 – G2G) at 600ms and 1000ms ISIs. The inhibition tasks were not successful in producing the expected inhibition effects in the groups and were therefore not analysed in any subsequent correlations.

The correlation between the stimulus-response priming differentials at 600ms and 1000ms ISIs was significant, $r_s(60) = 0.64$, $p < 0.001$. Similarly, the correlation between the response-priming differentials at 600ms and 1000ms was significant, $r_s(60) = 0.54$, $p < 0.001$.

To determine whether there was a relationship between the facilitation differentials for the modified priming task and the Go/Nogo task at 600ms (where facilitation was demonstrated to be more enhanced in OCD), Spearman's rho correlations were calculated. The facilitation variables included: (i) the mean facilitation differential for the modified priming task (i.e., control RT – positive priming RT); and (ii) the mean facilitation differential for response priming in the modified Go/Nogo task (i.e., G2 – G2G) at 600ms ISI. Because measures of facilitation on the modified priming and modified Go/Nogo tasks were hypothesised to correlate, a one-tailed test was employed. As expected, a significant correlation was found between measures of facilitation on both tasks, $r_s(60) = 0.23$, $p = 0.04$.

5.4.5 Relationship between Performance and Clinical Characteristics

To determine if there were any relationships between cognitive measures of facilitation and OCD or GAD clinical characteristics, Spearman's rho correlations were calculated. Such data were analysed with non-parametric tests because the facilitation differentials were not normally distributed. The variables for the cognitive measures included: (i) the mean facilitation from the modified priming paradigm (i.e., control RT – positive priming RT), and (ii) the mean facilitation differential for response priming in the modified Go/Nogo task (i.e., G2 – G2G) at 600ms ISI. The clinical characteristics were: (i) Y-BOCS total, (ii) contamination/washing and checking subscales of the PI-WSUR, (iii) OBQ total, and (iv) PSWQ total. The inhibition tasks

were not successful in producing the expected inhibition effects in the groups and were therefore not analysed. The correlations are presented in Table 5.10.

Table 5.10. *Spearman's Rho Correlations for Facilitation Differentials on Modified Priming and Modified Go/Nogo Paradigms with Measures of OCD and Worry Severity and OCD proneness*

Task	Y-BOCS	PI-WSUR washing	PI-WSUR checking	OBQ-44	PSWQ
Modified Priming	0.38	0.23	0.49*	-0.01	0.03
Modified Go/Nogo (ISI: 600ms)	-0.09	-0.33	0.02	0.03	0.08

Note. ISI = interstimulus interval; Y-BOCS = Yale-Brown Obsessive Compulsive Scale total score; PI-WSUR = Padua Inventory-Washington State University Revision, washing and checking subscale scores; OBQ-44 = Obsessive Beliefs Questionnaire-44, total score; PSWQ = Penn State Worry Questionnaire, total score; * = $p < 0.05$.

As is evident from Table 5.10, the correlation between the facilitation differential on the modified priming paradigm and the PI-WSUR checking subscale was significant, $r_s(21) = 0.49$, $p = 0.03$. No other significant correlations were found between cognitive measures of facilitation and OCD or GAD clinical characteristics. Interestingly, a significant correlation was observed between the total scores on the OBQ and PSWQ, $r_s(60) = 0.71$, $p < 0.001$.

5.5 Discussion

The current study makes several important contributions to the OCD literature. Firstly, it is one of few studies investigating both facilitation and inhibition in OCD using paradigms that have been modified to measure them independently. An added benefit of such an approach is the replication of Bannon et al.'s (2008) seminal study. Secondly, the employment of the modified Go/Nogo paradigm enabled the current study to investigate stimulus-response vs. response priming in OCD, which has not been studied before. Thirdly, it examined the temporal course of inhibition and facilitation in OCD, which has received little attention prior to this study.

Fourthly, it is one of few studies investigating facilitation and/or inhibition in OCD that has included both a healthy control and anxious control group. The inclusion of the GAD group enables this study to determine whether facilitation and inhibition abnormalities are specific to OCD or anxiety disorders in general.

In general, the present study independently examined facilitation and inhibition processes in OCD using the modified priming and modified Go/Nogo tasks. Specifically, this study investigated whether people with OCD exhibit greater facilitation, reduced inhibition, or both compared to healthy and anxious control groups, and whether the temporal course of such facilitation and inhibition would be different in OCD. The current study also examined whether facilitation and inhibition are related to OCD or GAD severity and checking or washing subtypes of OCD.

5.5.1 Demographic and Clinical Characteristics

There were no significant group differences on demographic variables. Therefore any differences found between the groups on facilitation or inhibition cannot be attributed to differences on demographic variables such as sex, age, or education. As expected the OCD and GAD groups reported significantly higher levels of psychopathology on all clinical variables compared to normal controls. In contrast, the OCD and GAD showed similar levels of psychopathology for all clinical variables, except worry, for which GAD patients reported significantly higher scores. This indicates that any differences found between the clinical groups on facilitation or inhibition are unlikely to be related to differences in level of psychopathology.

5.5.2 Modified Priming Paradigm

In accordance with the study's predictions, no difference was found between OCD, normal control, and GAD groups' general speed of processing. In regards to facilitation, the OCD group exhibited greater facilitation effects to repeated target stimuli compared to normal control and GAD groups. This is consistent with the larger facilitation effects in OCD reported by Hartston & Swerdlow (1999), and it replicates Bannon et al.'s (2008) finding of stronger facilitation effects in OCD using the modified priming paradigm. Hence, these findings suggest that people with OCD exhibit stronger facilitation effects, which cannot be accounted for by a general speed of processing abnormality.

OCD participants made more commission errors on the facilitation task than normal controls and GAD participants. This difference was significant when compared to normal controls and it was marginally significant when compared to GAD participants. The faster response times observed to repetitions of target stimuli and an increased number of commission errors in OCD are consistent with Bannon et al.'s (2002) findings using a Go/Nogo task, and the interpretation that commission errors may reflect facilitation effects.

In regards to inhibition, it is important to note that this task was not able to capture inhibition effects across groups, and so the inhibition differential used as a measure of inhibition may be questionable. The current findings using the inhibition differential contrast with the study's hypotheses of reduced inhibition in OCD. That is, although the OCD group exhibited reduced negative priming on the modified priming task, as they produced a faster (instead of slower) response time to target stimuli that were distractors on the preceding trial, no significant group differences were found. Hence, this study failed to replicate Bannon et al.'s (2008) findings of reduced negative priming in OCD compared to controls. This is also inconsistent

with other studies that have found evidence of less or no negative priming in OCD using stimulus presentation durations of 100ms (Enright & Beech, 1990; Enright & Beech, 1993a, 1993b). However, as the current study employed a stimulus presentation duration of 200ms, this result is consistent with previous negative priming studies that have utilised stimulus presentation durations of longer than 100ms and failed to find a difference between OCD and anxious or normal control groups (e.g., Enright et al., 1995; Hoenig et al., 2002; MacDonald et al., 1999; McNally et al., 2001). Thus, further research needs to investigate whether findings of negative priming deficits at stimulus presentations durations of 100ms are really a reflection of impaired distractor suppression in OCD or a reflection of some other process, such as visual attention deficits (MacDonald et al., 1999).

5.5.3 Modified Go/Nogo Paradigm

The current study also employed the modified Go/Nogo paradigm to examine the facilitation and inhibition effects in OCD. The added advantage of using the modified Go/Nogo task is that it enables the examination of facilitation effects across multiple Go stimulus repetitions, as opposed to the sole stimulus repetition in priming tasks. In addition, it enables the current study to disentangle stimulus-response priming and response priming to determine what mechanisms may underlie enhanced facilitation results in OCD. Furthermore, unlike traditional Go/Nogo tasks which use commission errors as an index of inhibition deficits, the addition of the X-Go stimulus in the modified Go/Nogo task allowed the current study to examine whether Nogo stimuli are actively suppressed in OCD. To investigate the temporal course of facilitation and inhibition in OCD, participants completed the modified Go/Nogo task twice at two different ISIs (600ms and 1000ms).

In accordance with the study's predictions, no difference was found between OCD, normal control, and GAD groups' general speed of processing. Hence, any differences found between groups on measures of facilitation cannot be attributed to differences in baseline speed of processing.

In regards to facilitation, a reduction in response times to Go1 stimulus repetitions was evident for all groups. Although such facilitation effects were demonstrated at both 600ms and 1000ms ISIs, faster response times were produced at an ISI of 600ms compared to 1000ms. Facilitation gains were more pronounced for OCD compared to normal controls at the 600ms ISI. In regards to the GAD group, a quadratic trend suggested the OCD group displayed stronger facilitation than the GAD group. Such results suggest that OCD participants exhibit stronger facilitation effects to Go1 stimulus repetitions compared to normal controls and GAD groups, with earlier repetitions capturing this between-group difference better than later repetitions (quadratic effect).

To assess whether the enhanced facilitation observed in OCD is the result of stimulus repetition or response repetition, further analyses were conducted on repetitions of different Go stimuli (i.e., Go2 then Go1). A reduction in response times to Go stimulus repetitions was evident for all groups. Specifically, larger reductions in response times were produced to initial Go stimulus repetitions compared with latter sequence repetitions. Although such facilitation effects were demonstrated at both 600ms and 1000ms ISIs, faster response times were produced at the shorter ISIs (600ms compared to 1000ms). These findings are in accordance with those from Study 1 (Chapter 4).

Facilitation gains were stronger earlier in the sequence, to initial Go repetitions, and this response gain plateaued at latter repetitions. The OCD group demonstrated stronger facilitation

gains to the first Go response repetition (even when Go stimuli were different) compared to normal control and GAD groups. No other group differences were found for Go repetitions latter in the stimulus sequence. The stronger facilitation effects observed in OCD is consistent with the study's hypotheses and Bannon et al.'s (2002) finding of faster response times to Go stimuli in OCD.

In summary, OCD participants exhibited greater facilitation effects to both Go1 stimulus repetitions (stimulus and response priming), and Go response repetitions (response priming) which cannot be accounted for by a general speed of processing abnormality. Such findings suggest that response priming may be the mechanism underlying enhanced facilitation effects for OCD. Sequence analyses suggest that a stronger facilitation gain for OCD is demonstrated at the first response repetition and this difference plateaus at latter repetitions. In regards to temporal course, the results suggest the enhanced facilitation gain for OCD participants may be more pronounced at the 600ms ISI.

The study's findings regarding commission errors deserve mention because they were in a direction that was not expected. In effect, longer strings of Go stimuli resulted in a linear decrease in errors to the subsequent Nogo stimulus. This finding is intriguing because response time results clearly indicate a build up of facilitation with repeated Go stimuli, so the opposite effect would be expected. Such findings also fail to support the pattern of commission errors predicted based on inhibition theory. Consequently, this pattern of results is particularly problematic for research that has interpreted commission errors as indicative of inhibition failure.

These results suggest that various factors may influence commission error rate. For example, strategic expectancies may explain the decrease in commission error percentage as the number of Go1 stimulus repetitions preceding the presentation of Nogo1 stimuli increased. That

is, participants may have been more likely to anticipate a change to a Nogo stimulus by the fourth and fifth Go1 stimulus repetitions, and thus were more cautious about their responses at latter Go1 stimulus repetitions. This result and interpretation is also consistent with Thomas et al.'s (2009) findings. However, it contrasts with Durston et al.'s (2002) findings and Study 1's hypothesis that commission errors to Nogo1 stimuli would increase as a function of the number of preceding Go stimulus repetitions, thus reflecting facilitation effects.

Results also indicate that overall participants produced more commission errors to Nogo1 stimuli at an ISI of 600ms compared to 1000ms. However, at a group level the normal control group demonstrated a different pattern of results compared to OCD and GAD groups. That is, the normal control group made more commission errors at an ISI of 1000ms compared to 600ms, whereas OCD and GAD groups displayed the opposite pattern. These results are difficult to reconcile and interpretations can, at this stage, be no more than speculative and at best hypotheses requiring corroboration. It is possible that OCD and GAD groups were more cautious about their responses than normal controls, with this strategy being more useful and having an impact at the longer ISIs (1000ms). In summary, although it is clear that commission errors were not related to facilitation or inhibition processes in this paradigm, the specific factors influencing commission errors is unclear, and requires further investigation.

All groups failed to show evidence for the active suppression of X-Go stimuli. That is, instead of a response time cost, a response time advantage was demonstrated for X-Go stimuli compared to X-Go control stimuli. Once again ISI had an impact on overall response times, with faster response times produced at an ISI of 600ms compared to 1000ms. In contrast to the study's predictions no group differences were found for response times to X-Go stimuli. However, as both the current study and Study 1 (Chapter 4) failed to produce evidence for the

active suppression of X-Go stimuli, response times to X-Go stimuli do not appear to reflect inhibition. Hence, one cannot interpret the absence of group differences on mean response times to X-Go stimuli as evidence for comparable inhibition effects across groups.

Omission errors to X-Go stimuli may better reflect inhibition effects than response times. That is, more omission errors were produced to X-Go stimuli compared to X-Go control stimuli across all groups, suggesting that response suppression to the Nogo1 stimulus (diamond) was not overcome quickly enough to respond when the same stimulus was re-presented as a Go stimulus. Further support for this contention is the finding that less omission errors were produced at an ISI of 1000ms compared to 600ms, when participants had more time in between stimulus presentations to overcome this suppression and respond to the now Go stimulus. However, no group differences were found for the omission error rate produced to X-Go stimuli, suggesting that group performances on this measure of inhibition were comparable. This contrasts with previous reports of inhibition deficits in OCD on Go/Nogo tasks (e.g., Bannon et al., 2002; Penades et al., 2007), although different dependent variables (e.g., commission errors, percentage of successful inhibition trials) were used to measure inhibition in such studies.

In summary, OCD participants showed a comparable performance to normal control and GAD groups on measures of inhibition in the modified Go/Nogo paradigm. Furthermore, no evidence was found to suggest that the temporal course of inhibition in OCD is different to that of normal control or GAD groups.

5.5.4 Relationship between Cognitive Tasks

The current study investigated whether participants' facilitation differentials on the modified Go/Nogo tasks were consistent from the short (600ms) to the long (1000ms) ISI. The

significant correlations found between both the stimulus-response priming and response priming differentials at 600ms and 1000ms indicates participant's facilitation differentials were consistent across ISIs.

The present study also examined whether there was a relationship between facilitation differentials across paradigms. A significant correlation between measures of facilitation on the modified priming and modified Go/Nogo task suggests these findings are also consistent across tasks.

5.5.5 Relationship between Performance and Clinical Characteristics

Given that facilitation abnormalities may contribute to OCD symptoms, the current study also investigated whether facilitation effects were related to OCD or GAD severity. However, no significant correlation was found between facilitation effects and overall OCD severity, as measured by the Y-BOCS, or GAD severity, as measured by the PSWQ.

As several studies have reported that checking subtypes of OCD demonstrate different patterns of facilitation (Hartston & Swerdlow, 1999) or inhibition (Hoenig et al., 2002), the present study also examined whether washing and checking subtypes of OCD correlated with facilitation. Subtype analyses revealed that higher scores on the checking subscale of the PI-WSUR were related to larger facilitation effects on the modified priming paradigm. This is consistent with Hartston and Swerdlow's (1999) findings of increased facilitation in OCD subgroups with a history of checking compulsions. Such preliminary findings indicate that enhanced facilitation may contribute to the repetitive quality of OCD symptoms, especially checking compulsions. For example, once a checking compulsion has been performed it is more highly primed, thus facilitating the recurrence of such behaviour. However, further research on

the relationship between checking compulsions and facilitation is needed, as no significant relationship was found between the facilitation differential on the modified Go/Nogo task and scores on the PI-WSUR checking subscale.

In contrast to OCD checkers, no significant correlations were found between scores on the PI-WSUR washing subscale and facilitation effects in the modified paradigms. In regards to the OBQ, a significant relationship was found between total OBQ and PSWQ scores. This finding is consistent with another study that reported a significant positive relationship between worry and obsessive beliefs (Meyers et al., 2008). However, OBQ scores were not related to facilitation effects in either of the modified paradigms. This contrasts with Parker's (2007) finding that higher OBQ scores correlated with greater facilitation effects in OCD and university control groups. This may have been because GAD participants displayed similar scores to the OCD group on the OBQ, but did not produce the enhanced facilitation effects seen in the OCD group.

In summary, the severity of OCD or GAD was not related to facilitation effects on either of the modified paradigms. However, OCD checkers displayed greater facilitation effects on the modified priming paradigm. The relationship between facilitation and checking compulsions requires further investigation.

5.5.6 Implications

The findings from the present study have important implications for the understanding of information processing and neurobiological mechanisms of OCD. In particular, given the robust finding of facilitation abnormalities in OCD, it is possible that enhanced facilitation may contribute to the repetitive quality of obsessions and compulsions. That is, enhanced facilitation

to primed stimuli and responses may result in a continued focus on and the perpetuation of primed thoughts and behaviours. For example, once an individual has completed a checking compulsion, this behaviour is more highly primed increasing the likelihood that the individual will continue to focus on and subsequently repeat this behaviour. These results are consistent with previous suggestions that obsessions and compulsions in OCD may act as self-primers that facilitate their own recurrence (Bannon et al., 2008; Hartston & Swerdlow, 1999).

However, given non-threatening stimuli were used in the current study and that obsessions and compulsions are limited to the individual's idiographic OCD-related concerns, it is possible that enhanced facilitation in OCD is unrelated to anxiety symptoms and is instead a trait abnormality of OCD. To clarify this, future research needs to examine whether enhanced facilitation is present prior to the onset of OCD and when OCD has remitted. Furthermore, given that people with OCD don't repeat neutral thoughts or behaviours persistently, it seems likely that facilitation abnormalities interact with other specific vulnerabilities to developing OCD to result in a narrow range of obsessions and compulsions. Such specific factors may include cognitive threat schemas (Bannon et al., 2008) or thought suppression. Within this perspective, pre-existing cognitive schemas would selectively bring certain thoughts to attention, and stronger facilitation mechanisms would carve them into consciousness. If thought suppression is subsequently attempted as a way of coping with these hyperaccessible distressing thoughts, a paradoxical increase in the frequency of the thought would be expected, resulting in a repeating cycle (Tolin et al., 2002).

Thus, enhanced facilitation may contribute to the chronic and relapsing pattern of OCD. In regards to implications for treatment, the current findings suggest that reducing enhanced facilitation effects may also improve OCD symptoms. As treatments for OCD often result in

only partial improvement of symptoms, treatment approaches that attempt to reduce enhanced facilitation may supplement and improve the effectiveness of current treatments for OCD. Amir and colleagues (2009) recently found that an attention training intervention aimed at reducing an attention bias for threat-relevant information was effective at reducing anxiety in GAD patients (Amir, Beard, Burns, & Bomyea, 2009). The development of a similar intervention aimed at reducing facilitation effects would be valuable for the treatment of OCD.

5.5.7 Limitations and Future Research

A limitation of the current research was that none of the modified paradigms were able to adequately capture negative priming effects in OCD or normal controls. While priming and Go/Nogo tasks have commonly been used to measure inhibition, the current study used priming and Go/Nogo paradigms that had been modified to disentangle facilitation and inhibition. The current study's results are consistent with findings from Study 1, which highlighted the elusiveness of inhibition when it was unable to capture inhibition in three different paradigms across multiple temporal intervals. Although the modified paradigms used in the current study are not valid and reliable measures of inhibition, it is possible that other tasks exist which are valid and reliable measures of inhibition within a clinical context. Hence, future research on inhibition in OCD needs to ensure they employ paradigms with adequate psychometric properties.

The absence of evidence for the active suppression hypothesis raises questions about its reliability and validity. That is, if negative priming is a state-and context-dependent effect then greater specification about the specific factors needed to produce negative priming is required. Furthermore, because the only negative priming studies that have reported negative priming

deficits in OCD are those that have employed a stimulus presentation duration of 100ms, future research needs to investigate whether such deficits are really a reflection of impaired distractor suppression in OCD or some other process, such as visual attention deficits (MacDonald et al., 1999).

Although the temporal course of facilitation and inhibition effects was examined in the current study, the temporal intervals were limited to 600ms and 1000ms. As, Hoenig and colleagues (2002) found differing patterns of negative priming in OCD checkers and non-checkers using response-stimulus intervals of 500ms and 2000ms, future research should extend on the present findings by employing different temporal intervals that also include ISIs of longer than 1000ms to assess whether enhanced facilitation effects persist for longer in OCD. Furthermore, the reliability of facilitation and inhibition effects across time was not examined in the present study. Hence, to determine whether enhanced facilitation effects are stable trait-like effects in OCD, research needs to investigate the test-retest reliability of facilitation.

Another limitation of the current study was that it was cross-sectional and only examined people with OCD who were symptomatic. A cross-sectional design limits our ability to discern whether selective attention abnormalities in OCD are present before one develops OCD. Additionally, the use of only symptomatic OCD participants means we are unable to determine whether such abnormalities remain when OCD symptoms remit. Bannon et al.'s (2008) findings suggest that enhanced facilitation effects exist in both remitted and symptomatic OCD. However, it is evident that further research regarding the stability of selective attention abnormalities in OCD pre- and post-treatment using a longitudinal design would be of great value.

The current study employed neutral stimuli in modified paradigms (i.e., neutral words and shapes). While it is important to establish whether OCD patients have general abnormalities

in the selective attention, it is also important to determine whether they have specific selective attention abnormalities. That is, as OCD patients only experience obsessions and compulsions in relation to their specific concerns it is also important to examine selective attention in relation to their specific OCD-related concerns. Bannon and colleagues (2008) employed both neutral and OCD-threat words and observed that OCD-threat related words exaggerated OCD inhibitory deficits but had no impact on facilitation effects in OCD. Given this is the only study to investigate the impact of anxiety provoking stimuli on facilitation effects in OCD, further research is needed. Such research may consider using visual pictures of OCD-threat related stimuli instead of words, as Moritz et al. (2010) argue that worries of OCD patients are typically triggered by visual cues or images (e.g., dirt on table).

Although clinical participants with a history of psychosis, substance abuse, and comorbid OCD and GAD were excluded from this study, it is possible that the number of OCD and GAD participants with secondary diagnoses, such as depression, confounded the results. For example, depression has been suggested to influence performance on tests of executive functioning, such as measures of inhibition (Basso, Bornstein, Carona, & Morton, 2001). However, in this study depression appears unlikely to have influenced the enhanced facilitation effects observed in OCD compared to normal control and GAD groups, as the GAD group reported comparable levels of depression on self-report measures (i.e., BDI-II) to the OCD group, and had a comparable number of participants diagnosed with major depression and dysthymia as the OCD group.

As discussed in Chapter 1, the literature indicates that OCD is a heterogeneous disorder with some researchers positing distinct clinical subgroups exist, e.g. ‘washers’ and ‘checkers’ (Khanna et al., 1990; Summerfeldt et al., 1999). It is possible that subgroups of OCD display different patterns of selective attention abnormalities. For example, the present study found OCD

checkers were more likely to have enhanced facilitation. Research investigating facilitation and inhibition abnormalities in large homogenous subgroups of OCD would help identify whether each subgroup has its own profile of selective attention abnormalities.

Given the present study is only one of few studies examining facilitation in OCD it highlights the need for further investigation in this area. Such research could be included in the recent search for neurocognitive endophenotypes of OCD (Chamberlain et al., 2005; Menzies et al., 2007). Endophenotypes are a heritable quantitative trait associated with increased genetic risk for a disorder and are therefore present in both patients and their clinically unaffected relatives (Gottesman & Gould, 2003). Menzies et al. (2007) combined structural neuroimaging (magnetic resonance imaging) and cognitive testing (stop-signal task) to assess OCD patients, their unaffected first-degree relatives, and controls. The authors reported that variations in brain systems related to motor inhibitory control may mediate genetic risk for OCD, representing evidence for a neurocognitive endophenotype of OCD. While research on neurocognitive endophenotypes in OCD has focused on inhibition, future research could examine facilitation in OCD and their unaffected first-degree relatives.

5.5.8 Summary and Conclusions

. This is the most comprehensive study on facilitation in OCD to date, as it examined facilitation in OCD across several tasks, across sequences, across ISIs, and against a normal control and anxious control group. In addition to the contribution of a new paradigm that could reliably measure facilitation (modified Go/Nogo task), this study yielded three new pieces of information. First, OCD patients exhibited enhanced facilitation to primed stimuli and responses compared to normal control and GAD groups on two different measures of selective attention,

suggesting that stronger facilitation effects may be specific to OCD. Second, exaggerated facilitation was most evident in OCD patients with checking compulsions. Third, stronger facilitation effects in OCD were more pronounced at the 600ms ISIs, suggesting temporal determinants influence facilitation effects across groups. A manuscript regarding such findings has been submitted to *Behaviour Research and Therapy*.

These findings suggest selective attention abnormalities may contribute to the development and maintenance of OCD symptoms, especially for OCD checkers. Specifically, stronger facilitation processes that enhance the re-activation of primed stimuli and responses may underlie the recurring nature of obsessions and compulsions.

CHAPTER 6

FINAL COMMENTS AND CONCLUSIONS

This thesis addressed a gap in the OCD literature by systematically examining both facilitation and inhibition independently in OCD. In order to do this 2 studies were conducted. Available tasks to measure facilitation were inadequately researched and tasks to measure inhibition had major inadequacies. While ten studies have examined facilitation using the identity priming paradigm (Gibbons et al., 2006; Groh-Bordin & Frings, 2009; Koshino et al., 2000; Lowe, 1979; Neumann & DeSchepper, 1991; Salo et al., 2002; Stadler & Hogan, 1996; Tipper, 1985; Troche et al., 2008; Waechter et al., 2010), only one study to date has assessed facilitation using the Go/Nogo paradigm (Thomas et al., 2009). Further, tasks measuring inhibition (e.g., Go/Nogo task) have been criticised for confounding facilitation and inhibition (Bannon et al., 2008; Thomas et al., 2009), and found inconsistent results (e.g., Moore, 1994; Tipper, 1985; Tipper & Cranston, 1985). Hence, Study 1 aimed to develop priming and Go/Nogo tasks that disentangled facilitation and inhibition, so they could be utilised in Study 2 to assess facilitation and inhibition independently in OCD. Such aims were ambitious, and although not all of these objectives were realised, this thesis makes valuable observations and contributions that have theoretical and clinical implications. Although conclusions from Study 1 and 2 have been discussed more elaborately in Chapters 4 and 5, respectively, this chapter aims to highlight and integrate the major findings and contributions of the thesis taken as an entity.

A comprehensive and systematic investigation of facilitation in OCD: A valuable contribution of this thesis is the comprehensive investigation of facilitation in OCD. That is, this thesis examined facilitation in OCD across several tasks, across systematic manipulations of

sequences to track the development and plateauing of the facilitation process, across ISIs and against a normal control group and anxious control group. Such a comprehensive scrutiny of facilitation in OCD, as far as we are aware, has not been undertaken until now. This thesis found that OCD participants exhibited enhanced facilitation effects compared to an anxious and normal control group on both the modified priming and modified Go/Nogo tasks. This exaggerated facilitation was more pronounced at the 600ms ISI, suggesting that facilitation may not only be stronger in OCD but also that these abnormalities may best be detected at earlier ISIs. Within the context that previous OCD literature has focused on inhibition deficits and that currently there are no more than 2 studies on facilitation, the demonstration of stronger facilitation in OCD across two tasks and against both a normal and clinical control group is a compelling argument that it is a robust finding, and thereby constitutes an important contribution to the OCD literature.

When combined with previous findings, such results indicate that enhanced facilitation may be specific to OCD. That is, this thesis and other studies examining facilitation in OCD have found enhanced facilitation in symptomatic and remitted OCD participants when compared with normal controls, panic disorder patients and GAD patients (Bannon et al., 2008; Hartston & Swerdlow, 1999). However, further research is needed on this understudied area.

Enhanced facilitation may contribute to the repetitive quality of obsessions and compulsions. To borrow from Bannon and colleagues (2008) analogy, such findings suggest that when stimuli or responses are primed in people with OCD, it is primed more strongly in a more permanent type of ink. Because this results in a higher level of activation for such stimuli or responses, it means people with OCD are more likely to stay stuck on these primed stimuli or responses and thus are more likely to repeat the primed thought or behaviour. For example, once an individual has completed a checking compulsion, this behaviour is more highly primed

increasing the likelihood that the individual will continue to focus on and subsequently repeat this behaviour.

These results have implications for understanding potentially the aetiology and maintenance of OCD, treatment strategies and relapse patterns, highlighting the importance of further research into this understudied area. Given enhanced facilitation effects were produced to non-threatening stimuli in this study and that similar facilitation deficits were found in symptomatic and remitted OCD groups, the pattern of results are consistent with the possibility that enhanced facilitation in OCD is unrelated to anxiety symptoms and is instead a trait-like abnormality of OCD. Given that people with OCD don't repeat neutral thoughts or behaviours persistently, it seems likely that facilitation abnormalities interact with other specific vulnerabilities to developing OCD to result in a narrow range of obsessions and compulsions. Such specific factors may include cognitive threat schemas (Bannon et al., 2008). Within this perspective, pre-existing cognitive schemas could be responsible for bringing anxiety provoking thoughts into attention and abnormally strong facilitation mechanisms could underlie the recurrence of such thoughts. If thought suppression is subsequently attempted as a way of coping with these hyperaccessible distressing thoughts, a paradoxical increase in the frequency of the thought would be expected, resulting in a repeating cycle (Tolin et al., 2002).

The current findings may have important treatment implications. If enhanced facilitation effects perpetuate the recurrence of OCD symptoms, pharmacological or psychological treatment approaches that specifically reduce facilitation in OCD may supplement and improve the effectiveness of current treatments for OCD. Enhanced facilitation may also signal an increased risk for developing OCD. Thus, preventive programs may be trialled.

New facilitation task: Another key contribution of the thesis is to provide the experimental and clinical literature with a facilitation paradigm that is valid, reliable, and is sensitive enough to be used for clinical research. The reliability of the Go/Nogo paradigm is evident in its internal consistency between studies. In particular, the linear and quadratic trends for Go stimulus repetitions and ISI effects were consistent from Study 1 and 2. The Go/Nogo paradigm also demonstrated discriminant validity in Study 2 as it was able to show group differences between OCD and control groups. Additional advantages of this paradigm include: (i) its short duration (< 10 minutes), (ii) its ability to discriminate between stimulus-response priming and response priming, and (iii) it provides multiple dependent measures including response times and errors, and each of these can also be analysed as a function of sequence. The ability to analyse sequences enables researchers to identify poor motivation and compliance because the individual's data would not follow the linear and quadratic pattern. Thus, this paradigm will be valuable for the selective attention literature in both healthy control and clinical populations as it enables researchers to plot the progression of facilitation and disentangle stimulus-response and response facilitation. It's many advantages will likely result in its being used by other researchers.

Questions raised regarding phenomenon of inhibition: Despite the endeavour of this thesis to measure inhibition independent from facilitation using modified paradigms, this thesis was unable to capture inhibition across modified tasks and across temporal intervals. Such negative findings do not disprove the existence of postulated inhibition mechanisms. However, the consistent difficulty in detecting inhibition effects raises valid questions about the phenomenon of inhibition, its determinants, and underlying mechanisms, including the active-suppression theory of negative priming. If negative priming only occurs under a narrow set of

circumstances, greater specification regarding the factors that influence negative priming is required. Only when negative priming effects are able to be predicted and produced in a reliable fashion will researchers be close to determining the mechanisms or factors underlying negative priming. Further, because both modified paradigms were unable to capture inhibition effects, the absence of group differences on these paradigms in Study 2 cannot be interpreted as evidence for comparable inhibition across groups. Given the conflicting findings regarding inhibition in OCD literature and the uncertainty of the psychometric properties of paradigms used to measure inhibition, this thesis highlights the need for valid and reliable tasks measuring inhibition to be established before further investigating inhibition in OCD. In particular, research needs to investigate whether findings of negative priming deficits at stimulus presentations durations of 100ms are really a reflection of impaired distractor suppression in OCD or a reflection of some other process, such as visual attention deficits (MacDonald et al., 1999).

Other methodological contributions: This thesis also raised questions regarding what commission errors in the Go/Nogo task actually reflect. Commission errors in this paradigm have typically been interpreted as evidence for inhibition failure, and hence increased commission errors reflect impaired inhibition. However, the results from Study 1 and 2 are contrary to this assumption. Specifically, factors that were supposed to increase inhibition (e.g., several repetitions of the Go stimuli) served to produce fewer rather than more commission errors. The results strongly suggest that the commission errors on the Go/Nogo task are not the product of inhibition, but the consequence of several other factors. Such factors may include facilitation, strategic expectancies, or control strategies. Hence, this thesis highlights the need for further investigation of the factors influencing commission errors and thus the mechanisms underlying commission errors.

Overall, this thesis contributes new findings to both the selective attention and OCD literature, enhances the understanding of the specific aetiological factors that contribute to the development of OCD, and contributes a new and effective paradigm to measure facilitation in experimental and clinical research.

CHAPTER 7

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CHAPTER 8

APPENDICES

8.1 Appendix A – Participant information and consent form for clinical patients at Westmead Anxiety Treatment and Research Unit

8.2 Appendix B – Participant information and consent form for other research participants

8.3 Appendix C – Demographic questionnaire

8.4 Appendix D – Word list for modified priming paradigm in Study 1 and 2

8.5 Appendix E – Word list for traditional priming paradigm in Study 1

8.6 Appendix F – CD ROM of results for both studies

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8.1 Appendix A

PARTICIPANT INFORMATION

Study Title: Inhibition and Facilitation Abnormalities in Obsessive-Compulsive Disorder and Generalized Anxiety Disorder

Name of Researchers: Ms Juliette Drobny, A/Prof. Craig Gonsalvez, Ms Christen Elks

What is the purpose of the study?

This study is investigating the way in which people with obsessive-compulsive disorder and generalized anxiety disorder encode and respond to certain stimuli.
This study is being conducted as part of a joint project between SWAHS Anxiety Treatment and Research Unit and the University of Wollongong.
It is hoped the results of this study will contribute to our understanding of some of the causes of obsessive-compulsive disorder and aid in the development of effective treatments.

Who will be invited to enter the study?

You have been invited to enter the study because you have been diagnosed with either obsessive-compulsive disorder or generalized anxiety disorder or you do not have either of these disorders and your results will be used as a comparison.

What will happen on the study?

There are two parts to the study. In the first part, you will be asked to complete some questionnaires assessing your mood, anxiety and beliefs. You may or may not have already completed some of these questionnaires as part of routine clinical care at the clinic. You will also be asked to rate a list of words (on a scale from 1 – 7) according to how threatening you find them. This is done to ensure we do not use words in the computer task (in part 2) that you may find threatening. The questionnaires and word list can be completed at home.
***Estimated time commitment**:* 30 minutes

You will be asked to return to the clinic on a separate occasion to participate in the second part of the study. This second visit will occur at least 7 days after your initial assessment at a time convenient to you. You will be asked to complete 2 computer tasks. In these tasks you will be shown a series of stimuli (either words or shapes, e.g. circle, square) and asked to respond to some of these stimuli by pressing the mouse button on the keyboard.
Estimated time commitment: 40 minutes

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PARTICIPANT INFORMATION (cont.)

Are there any risks?

No. Participation in the study involves completing two computer tasks, questionnaires and providing your consent for the researchers to use the questionnaire data about your mood and anxiety that was collected as part of your treatment. The main inconvenience will be the time taken to complete additional questionnaires and the computer tasks.

Confidentiality / Privacy

It is important to be aware that all information obtained in connection with this study is held strictly confidential. In the reporting of results, no individual will be identified and only group averages will be reported. Information from questionnaires will be stored in locked filing cabinets. Your name will be removed from all questionnaires and computer tasks and replaced with an ID number. Information will be entered onto a computer database. No identifying information (e.g., name, address, date of birth) will be linked to your data. This database is password protected and is only accessible to the researchers involved in the study.

Do you have a choice?

Yes. Participation in this project is purely voluntary and you may choose to withdraw your consent at any time without penalty or prejudice. Your decision as to whether or not to participate in this project will not affect your treatment or relationship with your therapist, the SWAMHS Anxiety Treatment & Research Unit, Westmead Hospital or any affiliated institution.

Complaints

If you have any concerns about the conduct of the study, you may contact the Westmead Hospital Patient Representative, Ms Jillian Gwynne Lewis, Telephone No 9845 7014 or email jillian_lewis@wsahs.nsw.gov.au.

Contact details

If you have any problems while on the study, please contact:

Ms Juliette Drobny
Ph: 9840 4095 (work hours), 0401 426 080 (after hours)

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CONSENT TO PARTICIPATE IN RESEARCH

Study Title:
Inhibition and Facilitation Abnormalities in Obsessive-Compulsive Disorder and Generalized Anxiety Disorder

Name of Researchers:
Ms Juliette Drobny, A/Prof. Craig Gonsalvez, Ms Christen Elks

Name of Researcher: Christen Elks

1. I understand that the researcher will conduct this study in a manner conforming with ethical and scientific principles set out by the National Health and Medical Research Council of Australia and the Good Clinical Research Practice Guidelines of the Therapeutic Goods Administration.

2. I acknowledge that I have read, or have had read to me the Participant Information Sheet relating to this study. I acknowledge that I understand the Participant Information Sheet. I acknowledge that the general purposes, methods, demands and possible risks and inconveniences which may occur to me during the study have been explained to me by Christen Elks and I, being over the age of 16 years, acknowledge that I understand the general purposes, methods, demands and possible risks and inconveniences which may occur during the study.

3. I acknowledge that I have been given time to consider the information and to seek other advice.

4. I acknowledge that refusal to take part in this study will not affect the usual treatment of my condition.

5. I acknowledge that I am volunteering to take part in this study and I may withdraw at any time.

6. I acknowledge that this research has been approved by the Sydney West Area Health Service Human Research Ethics Committee.

7. I acknowledge that I have received a copy of this form and the Participant Information Sheet, which I have signed.

Name of participant _____ Date of Birth: _____

Address of participant _____

Signature of participant _____ **Date:** _____

Signature of researcher _____ Date: _____

Signature of witness _____ Date: _____

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INDEPENDENT WITNESS:

I, _____ (name of independent witness)

of _____ hereby certify as follows:

1. I was present when _____ ("the participant") appeared to read or had read to him/her a document entitled Participant Information Sheet; or I was told by _____ ("the participant") that he/she had read a document entitled Participant Information Sheet (*Delete as applicable)

2. I was present when _____ ("the researcher") explained the general purposes, methods, demands and the possible risks and inconveniences of participating in the study to the participant. I asked the participant whether he/she had understood the Participant Information Sheet and understood what he/she had been told and he/she told me that he/she did understand.

3. I observed the participant sign the consent to participate in research and he/she appeared to me to be signing the document freely and without duress.

4. The participant showed me a form of identification which satisfied me as to his/her identity.

5. I am not involved in any way as a researcher in this project.

Name of independent witness: _____

Address: _____

Signature of independent witness: _____ Date: _____

Relationship to participant of independent witness: _____

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DEPARTMENT OF PSYCHOLOGY

8.2 Appendix B

INFORMATION SHEET

Facilitation Abnormalities in Obsessive-Compulsive Disorder

Chief Researcher: Associate Professor Craig Gonsalvez (Ph: 4221 3674)
Email: craigg@uow.edu.au
Additional Researchers:
Ms Christen Elks (Ph: 0414 606 420) Email: cae27@uow.edu.au
Ms Juliette Drobny (Ph: 9840 4095) Email: Juliette_Drobny@wsahs.nsw.gov.au

What is the purpose of the study?
This study is investigating the way in which people with anxiety disorders encode and respond to certain stimuli. This study is being conducted as part of a joint project between SWAHS Anxiety Treatment and Research Unit and the University of Wollongong. It is hoped that the results of this study will contribute to our understanding of some of the causes of obsessive-compulsive and generalized anxiety disorder and aid in the development of effective treatments.

Who will be invited to enter the study?
You have been invited to enter the study because you have been diagnosed with either obsessive-compulsive disorder or generalized anxiety disorder or you do not have either of these disorders and your results will be used as a comparison.

What will happen on the study?
There are two parts to the study. In the first part, you will be asked to complete some questionnaires assessing your mood, anxiety and beliefs. You will also be asked to rate a list of words (on a scale from 1 – 7) according to how threatening you find them. This is done to ensure we do not use words in the computer task (in part 2) that you may find threatening.
Estimated time commitment: Approximately 30 minutes

In the second part of the study you will be asked to complete a diagnostic inventory and computer task with Christen Elks. The diagnostic inventory will be administered to confirm an anxiety diagnosis. In the computer tasks you will be shown a series of stimuli (either words or shapes, e.g. circle, square) and asked to respond to some of these stimuli by pressing the mouse button on the keyboard.
Estimated time commitment: Approximately 60 minutes for diagnostic inventory and 40 minutes for computer tasks

Are there any risks?
No. Participation in the study involves completing questionnaires, two computer tasks and diagnostic inventory. The main inconvenience will be the time taken to complete these tasks.

Confidentiality / Privacy
The results of this study will be used to write a journal article to be submitted as part of the requirements for the completion of Christen Elks's Doctor of Psychology (Clinical) Degree at the University of Wollongong. The results of the study may also be presented at national and international conferences.

It is important to be aware that all information obtained in connection with this study is held strictly confidential. In the reporting of results, no individual will be identified and only group averages will be reported. Information from questionnaires will be stored in locked filing cabinets. Participant's names will be removed from all questionnaires and computer tasks and replaced with an ID code. Only researchers will have access to questionnaire and computer task data.

Are there any benefits for participants?
Yes. You will be paid $30 for your participation in this study. You will also be provided with an opportunity to obtain information about the results and conclusion of the research.

Do you have a choice?
Your participation in this study is entirely voluntary. If you choose not to join the study, or you wish to withdraw from it at any time, your treatment with your psychologist/or participation in the support group will not be affected in any way.

Complaints
If you have any concerns about the conduct of the study, or your rights as a study participant, you may contact the University of Wollongong Ethics Officer on (02) 4221 4457.

Questions
If you have any questions about this research or the procedures used, please contact the researcher (see below for contact details).

Contact details
If you have any problems while on the study, please contact Christen Elks.
Phone – 0414 606 420 E-mail – cae27@uow.edu.au

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CONSENT FORM

Facilitation Abnormalities in Obsessive-Compulsive Disorder

Chief Researcher: Associate Professor Craig Gonsalvez (Ph: 4221 3674)
Email: craigg@uow.edu.au
Additional Researchers:
Ms Christen Elks (Ph: 0414 606 420) Email: cae27@uow.edu.au
Ms Juliette Drobny (Ph: 9840 4095) Email: Juliette_Drobny@wsahs.nsw.gov.au

I acknowledge that I have read and understand the Participant Information Sheet relating to the study *Facilitation Abnormalities in Obsessive-Compulsive Disorder*.
I have had the opportunity to ask any questions I may have about the research and my participation with Christen Elks (researcher). I have been advised to call the researchers at any time with any further questions or problems.

I acknowledge that the general purposes, methods, demands and possible risks and inconveniences which may occur to me during the study have been explained to me by Christen Elks (researcher) and I, being over the age of 18 years, acknowledge that I understand the general purposes, methods, demands and possible risks and inconveniences which may occur during the study.

I understand that if I consent to participate in this project I will be asked to:

- Complete questionnaires asking me about my mood, anxiety and beliefs, and rate a list of words (on a scale from 1 – 7) according to how threatening I find them.
- Complete a diagnostic inventory with Christen Elks to confirm an anxiety diagnosis
- Complete a computerised task wherein I will be shown a series stimuli (e.g. words or shapes) on a computer screen and asked to respond to some of these stimuli by pressing the mouse key on the keyboard.

I understand that all information I provide will be treated as strictly confidential and I will not be personally identifiable in any way. My name will not appear in any publication.

I acknowledge having been told that I am free to choose not to participate in this study. I understand that if I do consent to participate in this research, I am free to withdraw from this study at any time. If I decline to participate, or if I withdraw, it will not affect my psychological treatment/or participation in the support group in any way.

I understand that the results of the study will be used to write a journal article to be submitted as part of the requirements for the completion of Christen Elks's Doctor of Psychology (Clinical) degree at the University of Wollongong. I understand that the results may also be presented at national and international conferences. I consent to the data being used in this manner.

I am aware that I may contact the University of Wollongong Ethics Officer on (02) 4221 4457 if I have any concerns or complaints regarding the way in which the research is or has been conducted.

I acknowledge that I have read the above statement, which explains the nature and objectives of the investigation to my satisfaction. By signing below, I am indicating my consent to participate in this research as it has been described to me.

Signed:……. Date:…../…../……

Name……..….

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8.3 Appendix C

PARTICIPANT DEMOGRAPHIC INFORMATION

Study title: Inhibition and Facilitation Abnormalities in OCD and GAD

Please note all aspects of this study, including results, will be strictly confidential and only the researchers will have access to your personal information. Any publication of the results from this study will use only de-identified information, so no individual person will be identifiable.

1. ID Code: Your first initial, your mother's first initial, father's first initial and the day and month of your birth (e.g. cjg0308) _____

2. Age: _____

3. Sex (F/M): _____

4. Is English your first language? Yes No (please circle one)

5. Language spoken at home: _____

6. What is your highest level of education (e.g. Yr 10, 12, completing undergraduate degree, TAFE) & Total Years of Education?

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7. Handedness: Left Right (please circle one)

8. Have you ever received a head injury/ or blow to the head that resulted in loss of consciousness? (Y/N)_____

8.4 Appendix D

The word list completed by participants for use in the modified priming paradigm in study 1 and 2 is presented below.

Instructions: For numbers 1 to 43, circle the response that best represents how you feel about the word. Circle only one response for each word.

	Neutral		Slightly Threatening		Threatening		Extremely Threatening
	1	2	3	4	5	6	7
1. harm	1	2	3	4	5	6	7
2. plate	1	2	3	4	5	6	7
3. faint	1	2	3	4	5	6	7
4. nylon	1	2	3	4	5	6	7
5. pet	1	2	3	4	5	6	7
6. faeces	1	2	3	4	5	6	7
7. pasture	1	2	3	4	5	6	7
8. garbage	1	2	3	4	5	6	7
9. antique	1	2	3	4	5	6	7
10. germs	1	2	3	4	5	6	7
11. anxious	1	2	3	4	5	6	7
12. steel	1	2	3	4	5	6	7
13. coffin	1	2	3	4	5	6	7
14. button	1	2	3	4	5	6	7
15. panic	1	2	3	4	5	6	7
16. guilt	1	2	3	4	5	6	7
17. dirt	1	2	3	4	5	6	7
18. cereal	1	2	3	4	5	6	7
19. blood	1	2	3	4	5	6	7
20. tumour	1	2	3	4	5	6	7
21. crazy	1	2	3	4	5	6	7
22. ballot	1	2	3	4	5	6	7
23. pause	1	2	3	4	5	6	7
24. sex	1	2	3	4	5	6	7
25. sponge	1	2	3	4	5	6	7

26. fear	1	2	3	4	5	6	7
27. door	1	2	3	4	5	6	7
28. block	1	2	3	4	5	6	7
29. disease	1	2	3	4	5	6	7
30. toilet	1	2	3	4	5	6	7
31. death	1	2	3	4	5	6	7
32. fruit	1	2	3	4	5	6	7
33. number	1	2	3	4	5	6	7
34. chest	1	2	3	4	5	6	7
35. check	1	2	3	4	5	6	7
36. apron	1	2	3	4	5	6	7
37. cancer	1	2	3	4	5	6	7
38. rocket	1	2	3	4	5	6	7
39. locked	1	2	3	4	5	6	7
40. invite	1	2	3	4	5	6	7
41. dizzy	1	2	3	4	5	6	7
42. poison	1	2	3	4	5	6	7
43. outline	1	2	3	4	5	6	7

8.5 Appendix E

The word lists used in the traditional priming paradigm in study 1 are presented below.

WORD LIST 1

OPERA
AGENT
STONE
GUEST
SKILL
ROYAL
PORCH
SHEET
TRUCK
STEEL
RURAL
NOVEL

WORD LIST 2

BREAD
QUEEN
THEME
CLOTH
WHEEL
GRASS
DOZEN
METAL
PLAIN
PILOT
URBAN
WAGON

8.6 Appendix F

CD ROM of Results for Both Studies