An Electrophysiological Investigation of Emotional Attention and Memory Biases in Depression: The Role of Working Memory Inhibitory Control Deficits

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Abstract

The high recurrence rate in depression suggests specific cognitive factors increase an individual’s risk for developing repeated episodes of the disorder. A factor implicated in the literature is biased cognitive processing of negative information. This includes sustained attention, elaboration and autobiographical memory for negative versus positive events. Empirical evidence and contemporary models suggest impaired ability to utilise inhibitory control over the entry and removal of extraneous negative information in working memory mediates these emotional processing biases (see Beck, 2008; Joormann, Yoon, & Zetsche, 2007). In depression, inhibitory control deficits are linked to poor emotional regulation (Joormann & Vanderlind, 2014) and increased tendency to rumination (Joormann & Gotlib, 2008), which serve to perpetuate and exacerbate depressed affect (Beck, 2008; Joormann, Yoon, & Zetsche, 2007). Previous research has primarily focused on examining the relationship between depression and biased cognitive processes and cognitive control deficits in different studies. However, the predicted interrelations between these processes in depressed and remitted-depressed samples have received limited investigation. To advance insight into the functional relations among these emotional information processing biases and cognitive deficits, the aim of the dissertation was to investigate these processes in a single investigation.

The current work consisted of three event related potential (ERP) studies of information processing of emotional word stimuli in a sample of female university students. Participants underwent diagnostic assessment and subgroups were selected for subsequent studies if they met the criteria for one of the three
experimental groups: Current Major Depressive Disorder (MDD; \( n = 30 \)), remitted depression \( (n = 13) \), and a never-depressed healthy control \( (n = 30) \). The three ERP studies aimed to examine the impact that clinical status had on processing emotional stimuli under conditions of encoding and attention (Study 1), recent episodic memory (Study 2), and working memory inhibitory control (Study 3). Based on Joormann et al.’s (2007) impaired cognitive control model of depression, the dissertation also sought to investigate whether group status could be predicted based on the linear combination of participants’ results obtained in these preceding experiments (Study 4).

In the first study, participants completed a self-descriptive task of positive and negative adjectives to assess encoding processes and schema activation. A bias to negative adjectives was expected for the MDD group, a positive bias was expected for the control group, and similar processing (no bias) was anticipated for the remitted depression group. Further, due to their negative schemata, MDD participants were expected to show greater expectation of negative than positive stimuli (P3b), which would be coupled with compromised right-parietal hemisphere activation (Heller, 1990, 1993). The behavioural and ERP results supported these predictions. MDD participants exhibited reduced right hemisphere P3bs in response to negative adjectives. Both the P2 and late positive potential (LPP) data suggest that the susceptibility to depression may derive from a lack of an innate or learned bias of the perceptual system. Specifically, it appears that depressed and remitted-depressed individuals have difficulty automatically identifying potentially interpersonally threatening information (P2). This poor attention control may result in interpersonal difficulties, such that have been
typically observed in depression. They also appear to show impaired ability to turn attention towards positive stimuli in an attempt to reappraise mood in the face of stress (LPP).

In the second study, ERPs in response to correctly recognised items from Study 1 intermixed with new/distractor items (the Old/New task) were obtained to examine possible neural processes underlying recent episodic memory. It was hypothesised that control individuals should exhibit greater memory for positive words relative to negative words, while MDD participants would show a biased memory towards negative words relative to positive words. The results replicated previous behavioural evidence of mood-congruent memory effects in free-recall. Unexpectedly, all participants recognised more positive than negative words; with remitted participants also recognising more positive words than the MDD participants. This might reflect use of pleasant memories as an emotional regulation strategy to maintain positive moods (e.g. control, remitted participants) or to regulate or reverse unpleasant mood states (e.g. MDD participants). Control participants showed greater late parietal negativity (LPNs) for negative relative to positive words, suggestive of greater difficulty in recalling of negative information. No divergent group ERP data were found for the mid-frontal (FN400) or late parietal complex (LPC) old/new effects. These null electrophysiological findings likely indicate a Type II error due to methodological issues, such as reduced opportunity for memory decay or ease of the recognition task more so than evidence of unaffected neurological activation underlying mood-congruent memory. Alternatively, it is possible that the self-referential encoding strategy
(Study 1) may have resulted in more efficient retrieval, which in turn improved source memory accuracy in the MDD participants.

The third study employed a modified emotional Sternberg working memory task developed by Oberauer (2001, 2005a, 2005b) that required Go/Nogo responses. Study 3 predicted MDD participants to show significantly greater difficulties in cognitive inhibition of distractor interference and proactive interference stimuli compared to the control and remitted group. This effect was expected to be specifically enhanced for the negative stimuli. Dependent variables included error rates, and Nogo-N2, Nogo-P3 and Nogo-LPP components. The study found depression to be associated with prolonged activation of irrelevant negative information in working memory, as indicated by greater error rates for negative relative to positive distractor and proactive interference stimuli. Remitted participants exhibited greater Nogo-N2s during inhibition of negative distractor interference items, and enhanced Nogo-P3 during removal of negative proactive interference items from working memory. This may suggest evidence of recruitment of suppression-based processes in remitted depression in an effort to maintain euthymic mood when confronted with stressful stimuli. For the MDD group, the Study failed to support its ERP hypotheses. It is possible that the high task demand and participant fatigue served to suppress the manifestation of these hypothesised cognitive control deficits on the electrophysiological level.

A predictive discriminant function analysis (DA) was conducted in Study 4. It was predicted a strong significant relationship would be found between predictors outlined in Joormann et al.’s (2007) impaired cognitive control model and clinical
group status. Self-report results for rumination and emotional regulation, and behavioural measures of negative biases (difference score: negative stimuli minus positive stimuli) in memory recall and recognition (study 2) as well as working memory inhibitory failures (study 3) were examined. The discriminant analysis found two significant functions. The first function separated depressed from non-depressed status. Specifically, MDD participants were found to exhibit increased use of rumination, decreased application of emotional regulation, and greater negative memory and proactive inhibition errors compared to both the remitted and control participants. This cluster of predictors is consistent with those implicated in Joormann et al.’s model. The second function maximally separated the control from the remitted participants, wherein the remitted participants exhibited less self-reported suppression and greater distractor intrusions for positive relative to negative stimuli. Both these processes likely represent compensatory attempts to maintain their depression remission. Quadratic classification procedures revealed that 67.1% of participants were able to be correctly classified into their respective groups based on Joormann et al.’s model. This classification was significantly higher than that which would be predicted based on chance. The poorest sensitivity was observed for the remitted participants, which is understandable given the heterogeneous nature of the sample. For the MDD participants, the model showed high sensitivity (93.30%) and specificity (81.40%). However, it is important to note that the study results are limited in their applicability to older, male, adult-onset, and very severely depressed and remitted-depressed patients.

Overall, the dissertation’s findings suggest that interventions for depression should focus on repairing working memory inhibitory control deficits for negative stimuli.
This may be achieved with meta-cognitive therapy and mindfulness-based programs. Further, incorporating ERP measures into clinical interventions with depressed patients may facilitate assessment, diagnosis, and treatment evaluation.

The current work cannot attest to causation. It is acknowledged that although the results are consistent with theoretical expectations, no causal interpretations can be made. Future research should aim to examine these hypothesised associations using experiential manipulation of cognitive control within prospective longitudinal assessment or cross-lagged designs.

Keywords: depression, inhibition, working memory, cognitive bias, ERP
Statement of Originality

“This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself.”

Signed  …………………………………

Rachel Anne Gleave

Date  …………………………………
List of Work Published in the Course of the Research

Sections of the dissertation (primarily Section 2.1 to 2.5 and Section 7.4.) are published in the peer-reviewed edited book: Beyond the lab: applications of cognitive research in memory and learning (published under the PhD candidate’s maiden name, Dati). It is referenced below. This work was published in the course of the PhD research. The PhD candidate’s contribution to this published work includes the review of the literature and preparation of the manuscript for publication. The contribution of the second and third authors to this published work includes editing of the manuscript for publication.

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Abbreviations

Affective Decision Mechanism          ADM
Affective Norms for English Words     ANEW
American Psychiatric Association     APA
Anterior cingulate cortex             ACC
Beck Depression Inventory-Second Edition BDI-II
Blood level deoxygenation             BOLD
Cognitive Behaviour Therapy           CBT
Current source density                CSD
Diagnostic and Statistical Manual, fourth edition, text-revised DSM-IV-TR
Distractor Interference               DI
Electroconvulsive therapy             ECT
Electroencephalography                EEG
Error Related Negativity               ERN
Event-related Potential               ERP
Hypothalamic-pituitary-adrenal        HPA
International Classification of Disease-10th edition ICD-10
Late Positive Potential               LPP
Long-term memory                      LTM
Medial Frontal Negativity             MFN
Major Depressive Disorder             MDD
Mindfulness-Based Stress Reduction    MBSR
Negative Affective Priming            NAP
Positive psychotherapy                PPT
Prefrontal cortex                     PFC
Principal components analysis         PCA
Proactive Interference                PI
Randomised Control Trial              RCT
Resource Allocation Mechanism         RAM
Response Style Scale                  RRS
Selective Serotonin Reuptake Inhibitor SSRI
Short allele serotonin transporter gene 5-HTTLPR
Short-term memory                     STM
Slow Wave                             SW
To-be-forgotten                       TBF
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Chapter 1

Introduction to the Research Problem

Although sadness is a universal experience, clinical depression is not. The paradox of this mood disorder has fascinated philosophers, researchers, and clinicians since first described in 500BC by Hippocrates—what he termed melancholia. Today, a diagnosis of Major Depressive Disorder (MDD) includes the cardinal features of chronic despair and/or loss of interest or pleasure for at least two weeks. It includes a symptom profile often including memory and concentration impairments, a sense of worthlessness, fatigue, suicidal thoughts or behaviours, and marked indecisiveness (American Psychiatric Association, 2000, 2013). These symptoms significantly impact many aspects of the sufferer’s life, including interpersonal, occupational, behavioural, and physical functioning. Most perplexing, is that depression appears to violate the dogma of human nature, such as the self-preservation instinct, the maternal instinct, the sexual instinct, and the pleasure principle. Even vital biological functions like eating or sleeping are also usually attenuated.

Depression is the leading cause of disability worldwide, with an estimated 1.2 billion people suffering from the condition each year (World Health Organisation, 2001). It is a highly recurrent disorder, with over 75% of affected individuals expected to experience more than one depressive episode in their lifetime (Keller & Boland, 1998). The risk of depression relapse positively correlates with the number of previous episodes (Keller, 2003). Almost twice as many women than men met criteria for a depression disorder (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Gender differences have been explained in terms of hormonal effects (Nolen-Hoeksema & Hilt, 2009), and women’s stronger right Amygdala activation to emotional events (Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Hofer et al.,
2006; Wrase et al., 2003), emotional appraisal differences (Garcia-Garcia, Domínguez-Borràs, San Mingue, & Escera, 2008; LeDoux, 2000; Li & Lin, 2008), and to increased ruminative responses subsequent to negative life events (Nolen-Hoeksema & Jackson, 2001; Nolen-Hoeksema, Larsen, & Grayson, 1999).

Depression is projected to be the second leading cause of global death in less than a decade’s time, surpassed only by coronary heart disease (WHO, 2001). Therefore, the need for greater understanding of the causes, perpetuation, and relapse of depression has never been greater. This enhanced understanding will help to inform improved treatment interventions—especially vulnerable groups, such as remitted and female samples.

There are a number of validated factors implicated in the etiology and relapse of depressive disorders. These include, but are not limited to, cognitive, social, genetic and physiological factors. This dissertation concerns the examination of the cognitive processes implicated in the disorder. While the results of behavioural measures of cognition are extremely helpful, they typically produce data that are the outcome of a rapid sequence of a series of cognitive processes. Given its high temporal resolution—in the millisecond range—the event-related potential (ERP) of the electroencephalograph (EEG) can provide benefits over behavioural assays to reveal subtle activities at different stages in the information processing stream.

Cognitive models of emotion generation and regulation involve the interaction between two modes of information processing: (1) the automatic, reflexive, bottom-up processing, and (2), the slow, controlled and strategic top-down processing (Clark & Beck, 2010; Ochsner & Gross, 2005). Research finds little difference between depressed, remitted-depressed, and never-depressed individuals in their initial, bottom-up response to negative events (Rottenberg, 2007). However, depressed
individuals show greater problems in disengaging from processing no-longer-relevant negative information (Joormann & Gotlib, 2008). This deficit in top-down processing to update the contents of working memory, dependent on prefrontal neural systems (Nieuwenhuis, Yeung, Van Den Wildenbert, & Ridderinkhof, 2003), appears to result in rumination on adverse events (Joormann & Gotlib, 2008). In turn, the negative cognitive set negatively skews autobiographical memory (Shestyuk & Deldin, 2010), resulting in poorer problem solving strategies (Nolen-Hoekesema, Wisco, & Lyubomirsky, 2008). Together, it appears to perpetuate and exacerbate depressed affect (Beck, 2008; Joormann et al., 2007).

**Importance of the Dissertation**

To date, the research support for the above theoretical connections is inconsistent. This is, in part due to differing methodologies, test stimuli and sample characteristics between studies. For instance, despite known gender differences in emotional information processing (e.g., Lithari et al., 2010), many researchers analyse averaged data from mixed samples. Others seem to use neutral stimuli as a baseline to compare participants reactions to valence stimuli (e.g., Chiu, Holmes, & Pizzagalli, 2008; Ilardi, Atchley, Enloe, Kwasny, & Garratt, 2007). Others inappropriately generalise data from studies utilising emotional word stimuli with those using facial stimuli. As gender differences and stimulus characteristics are known to result in differing cognitive profiles, especially in ERP morphologies (see Section 2.2.2.), the amalgamation of these results will produce confusing and invalid conclusions. A further limitation of previous research has been the narrow focus on examining the relationship between depression and these different cognitive processes in independent investigations. This is a weakness of the literature because although it effectively informs us about the negative prognosis that each of these cognitive
patterns may have on depressed individuals, it does not describe how these processes are related to each other or interacts to maintain depression.

In light of the above, the main aim of this dissertation was to assess the validity of the cognitive model of depression proposed by Joormann et al.’s (2007) impaired cognitive control model in a sample of female participants (N = 73). More specifically, it sought to examine the behavioural and neural correlates of emotional biases in self-evaluation, attention, retrieval, and inhibitory control in individuals with MDD (n = 30), compared with those with previous histories of MDD (remitted; n = 13), and never-depressed healthy participants (controls, n = 30). To accomplish this, participants completed three cognitive tasks. Behavioural and ERP data was collected to evaluate encoding and attention (Study 1), explicit memory (Study 2), and working memory inhibitory control (Study 3) for positive and negative personality adjectives. Predictive discriminant analysis (DA; Study 4) was conducted in an attempt to determine whether group status could be predicted based on the linear combination of participants behavioural and self-report results obtained in these preceding experiments. It is hoped that the findings of the dissertation will increase understanding of the connections between the different facets of emotional information processing in depression, and help inform treatment paradigms.

Outline of the Dissertation

The next chapter of this dissertation provides a literature review of the theoretical and experimental data for emotional information processing biases and inhibitory control deficits in depression. It concludes with a justification for the selected research and statistical methodology. The following three chapters will each outline the aims and hypotheses, Method, Results and Discussions for Study 1, 2, and 3, respectively. The sixth chapter presents Study 4, which aims to integrate these
preceding results to investigate the associations proposed in Joormann et al.’s (2007) model of depression and its predictive validity. The thesis concludes with a general discussion of overall findings and limitations of the studies. The clinical implications of the obtained data are provided and recommendations for future research are made.
Chapter 2

Literature Review: Emotional Attention and Memory Biases in Depression and Working Memory Inhibitory Control Deficits

Unipolar depression has been linked to mood-congruent selective attention and memory biases (Beck, 1967; Matthews & MacLeod, 2005), which may be related to inhibitory deficits for negative information (Joormann et al., 2007). This proposed executive control deficit has been associated to one of the most imperative focuses in depression research, the question of vulnerability versus resiliency. That is, it may help explain why, when faced with similar life stressors, some individuals react with a vicious cycle of negative thoughts and attributions that can escalate into an episode of MDD, while others do not. This chapter aims to review existing support for the above predictions. It begins with an explanation of the major historical and contemporary models that implicate the role of cognition in depression. Given the dissertation’s focus on electrophysiological data, the chapter provides a concise summary of this technique. It then reviews recent behavioural, ERP, and neuroimaging research for attention, memory, and working memory inhibitory control processes in depression. From this, the rationale, aims and selected methodologies of the dissertation’s four studies are made.

Prior to discussing this literature, it is important to note that most operational definitions of depression in the empirical literature lack construct validity. Depression or dysthymia is a diagnosis made by appropriately qualified and trained professionals according to validated diagnostic protocols, such as the DSM-5 (APA, 2013) or International Classification of Disease-10th Edition (ICD-10; World Health Organization, 1993). However, time and funding limitations often require researchers
in this field to describe the construct based on specific cut-off scores on self-report measures reflective of depressive symptomatology, such as the Beck Depression Inventory-Second Edition (e.g., De Lissnyder, Koster, Derakshan & De Raedt, 2010; Koster, De Readt, Goeleven, Frank, & Crombex, 2005; Joormann, 2006; Shane & Peterson, 2007). Researchers using this approach typically justify their decisions based on empirical support for the dimensional qualities of the disorder (Ingram & Siegle, 2009). However, there is concern regarding the suitability of conceptualizing clinical depression using data derived from these samples (Ingram & Siegle, 2009). Specifically, research on the dimensional nature of depression is not conclusive, with data suggesting qualitative differences between subclinical and clinical depression and between different subtypes of depression (e.g., melancholic, atypical, psychotic features; see Bruder, Kayser, & Tenke, 2013 for a review). This suggests that generalisation of such subclinical findings to the practical implications in the treatment of depressive presentations may be inappropriate and misleading (Ingram & Siegle, 2009). Readers should be aware of this limitation when evaluating the value of the research presented in this chapter. To help this process, the term depression is reserved in this chapter for clinically (i.e., DSM or ICD) diagnosed samples, with the term dysphoric used to refer to research employing self-report sampling procedures. The current collection of dissertation studies employs a clinically diagnosed depressed, remitted depressed and never-depressed control sample. A glossary of key terms and cognitive tasks pertaining to the dissertation and the relevant literature is provided in Appendix A.

2.1. Cognitive Models of Depression

Many approaches have been taken by theorists in an attempt to understand the origin and maintenance of depression. Some of these theories involve genetics and
biological functioning, whereas other approaches focus on personal, social, and
cognitive characteristics. This thesis exclusively focused on cognitive underpinnings
of depressive disorder, in hope to improve psychological treatment interventions. It is
not the intention of this PhD candidate to disregard the significance of these other
facets of depression. However, discussion and analysis of these large bodies of work
is beyond the scope of the current work. The interested reader is encouraged to see
Gotlib and Hammen (2002) for a review of these other models of depression.

2.1.1. Traditional Cognitive models: Cognitive content and processing.

Interrelations among emotional information processing have been considered
since the earliest theoretical cognitive frameworks of depression. Much of the early
research into cognition in depression is guided by theories implicating negative
schemas (e.g., Beck, 1967) or associative networks in memory (e.g., Bower, 1981).
Both of these traditional models predict that depression is characterised by global
negatively skewed information processing. However, failure to observe such
universal biases in depression led to the development of alternative theories. The
most popular of these was that proposed by Williams, Watts, MacLeod, and Matthews
(1988), later revised by the authors in 1997.

**Beck’s schema theory.** Beck’s (1967) schema theory of depression
implicates that in repose to stressful life events, depression-vulnerable individuals
activate idiosyncratic negative memory representations, known as schemas. Once
activated, these schemas preferentially direct attention and retrieval to information
that is consistent with its negative content. The product of this pessimistic information
processing bias is the development of a *vicious cycle* of escalating negative automatic
thoughts about the self, world, and future (*negative cognitive triad*: Beck, Rush,
Shaw, & Emery, 1976). This is predicted to result in the manifestation and
perpetuation of the person’s depressive symptoms. With repeated activation, core
eegative schemas become hypersensitive and gain a more rigid, coherent, and
elaborated organisation (morph into depressive modes: Beck et al., 1996; Clark &
Beck, 1999). The negative cognitive schema, therefore, becomes more readily
accessible and entrenched, thereby decreasing the person’s ability to reappraise their
situation, and thus increasing vulnerability to repeated depressive episodes. Beck
(1967) argues that these negative schemas persist beyond the depressive episode itself
and into the period of recovery. That is, in remitted depression negative schemata are
dormant but remain potentially reactive in the face of relevant triggering events
(Segal, 1988).

**Bower’s associative network theory.** Bower’s (1981) associative network
theory is based on the belief that human memory encompasses a number of joint
associative networks, comprised of several distinct nodes. Nodes are individual
configurations of related life experiences, cognitions, and behavioural expressions that
become activated by unique situational triggers. Bower argued that each emotion—
such as joy, anxiety, or depression—has a specific innate node in memory. When an
appropriate evoking signal triggers an emotion node, it primes activation of its
uniquely associated memory structures. This produces activation of cognitive,
affective, and behavioural patterns assigned to that emotion, known as mood-
congruent processing (Bower, Monteiro, & Gilligan, 1978). Each emotion node is
said to reciprocally inhibit the activation of an emotion node of opposing quality, such
that a depressive node will hinder the joy node and its associated pleasant memories
and behaviours. Bower’s theory is a general model of the relationship between mood
and memory, rather than one that is concerned with cognitive aspects of emotional
psychopathology. However, it has the potential to explain how mood, negative
information processing biases, and emotional regulation deficits in depression might be mediated (Ingram, 1984; Teasdale, 1983, 1988).

**Williams et al.’s cognitive framework.** Williams et al.’s (1988; 1997) integrated cognitive model made three key proposals: (1) both encoding and retrieval processes involve priming and elaboration components. In this model, priming is conceptualised as an automatic, bottom-up processes involved in strengthening information schemata, making them more available. Elaboration refers to strategic, top-down processes which form or reinforce association between triggered schemata (Graf & Mandler, 1984). Priming processes are said to be dependent on affective decision mechanisms (ADM), and elaboration on the resource allocation mechanism (RAM). That is, initial interpretation of incoming emotional information as negative is a product of the ADM system; whereas succeeding augmented allocation of resources towards supplementary negative information, thus enhancing encoding and elaboration of this event, is the function of the RAM system. Unlike Beck and Bower’s models, William et al. argue that (2) a bias in one aspect of information processing does not imply a bias in the other. (3) Different emotional states affect different aspects of information processing. Specifically, anxious moods are said to involve a pattern of early vigilance to mood-congruent threat information, followed by avoidance of further elaboration of this material. Namely, anxiety is said to be associated with actions of the ADM system: hypervigilance, implicit memory biases, and non-memorial elaboration of such information, but not with the RAM system: explicit negative memory bias (Harvey et al., 2004; Mathews & MacLeod, 2005; Mitte, 2008). Williams et al. argue that depression does not manifest these mood-congruent hypervigilance biases dependent on the ADM. They interpret investigations that have found such implicit attention and memory preferences in
depressed samples (e.g., Gotlib & McCann, 1984) are the result of unaccounted anxiety levels. Rather, Williams et al. believes depression to be related to an overactive RAM system, which is said to result in their difficulty disengaging from negative information once it has been made the focus of explicit awareness (e.g., Bradley, Mogg, & Lee, 1997). This deficient process is said to result in greater elaboration and thus, deeper encoding of negative stimuli, thereby increasing depressed individual’s sensitivity to negative mnemonic cues.

In summary, Beck’s (1967) model is based on the diatheses-stress hypothesis in which latent negative belief systems become activated following the individual’s experience of adverse life experiences. It is said to function by negatively skewing cognitive processing. Bower’s (1981) theory concerns a more general model of the relationship between mood and memory that likewise argues that cognitive processing is influenced towards information consistent with an individual’s current mood state. It is further comparable to Beck’s model in its claim that negative thinking patterns are dependent on specific activating negative life events. Thus, the presence of such stressors is necessary for these maladaptive negatively-skewed cognitive profiles to be observed in remitted depressed samples. Experimental mood priming has been proposed as a protocol by which this may be assessed (Velten, 1968). Thus, based on this logic, in the absence of such stressors, remitted depression should demonstrate cognitive styles similar to never-depressed samples. Controversy surrounds these priming predictions, with some researchers arguing that latent cognitive vulnerability in remitted depression may not be falsifiable (e.g., Coyne, 1992; Coyne & Gotlib, 1983). Particularly, as distinctive negative cues are required to activate said dormant negative schemas, failure to observe expected cognitive biases in this sample could constantly be argued as a Type II error: in that mood-priming procedures were not
sufficiently idiosyncratic or salient. A further problem with this research is that remitted depression studies use post-morbid participants and, consequently cannot discriminate between these cognitive profiles as a latent predisposition or as a consequence of experiencing depression (Just, Abramson, & Alloy, 2001). Whereas Beck and Bower’s model predict biases at all levels and aspects of information processing, Williams et al.’s cognitive framework argues that the current depressed mood is associated with negative biases in elaboration and not in the priming process of attention and memory. They do not make any predictions concerning remitted depressed states. Similar to Beck and Bower’s model, Williams et al. does not specify the cognitive mechanisms that might be responsible for this recycling of negative cognitions; or how this process is effectively suppressed upon remission. Nolen-Hoeksema’s (1991) rumination theory and Wenzlaff and Bates (1998) ironic process theory—outlined next—make an attempt to explain these two questions, respectively.

**Nolen-Hoeksema’s rumination theory.** Rumination is the difficulty in disengaging from recurring thoughts about the causes and consequences of one’s situation (Johnson, Nolen-Hoeksema, Mitchell, & Levin, 2009; Nolen-Hoeksema, 1991). Rumination in the context of depressed affect is argued to enhance accessibility to negative memories, by increasing activation and attention to core negative self-schemas (Ciesla & Roberts, 2002; Ciesla & Roberts, 2007; Joormann, 2004; Joormann, 2005, 2009; Joormann, Eugene, & Gotlib, 2008; Joormann et al., 2007; Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1988; Lyubomirsky & Nolen-Hoeksema, 1995; Robinson & Alloy, 2003). Recent research into why people ruminate in the context of depression implicate malfunction in updating the contents of working memory from irrelevant negative information (Goeleven, De Readt, Barert, & Koster, 2006; Joormann 2004; Joormann & Gotlib, 2008; Lo & Allen, 2011). This
inhibitory deficit is thus said to result in enhanced elaboration on such material, thereby strengthening their storage and accessibility in memory (Hasher & Zacks, 1988).

**Wenzlaff and Bates ironic process theory of suppression.** Wenzlaff and Bates (1998) argue that biased attention and memory to negative stimuli may not only become dormant in remission from depression—as implied by Beck’s (1967) schema model—but are actively suppressed by these individuals. In this view, maintained euthymic mood in remitted depression is dependent on continual monitoring and controlling of incoming stimuli and thoughts. This continual monitoring system is argued to operate in the background of consciousness by ensuring that negative cognitions do not intrude into awareness and by focusing attention towards distracting positive or neutral events. Accordingly, cognitive suppression is likely to fail under conditions of overtaxed mental load or stress (Van der Does, 2005; Wegner, 1994; Wenzlaff & Bates, 1998). It is argued that this vigilance and positive distraction system will falter; leaving this accumulation of suppressed negative thoughts unrestricted to intrude into awareness. For this reason, suppression is frequently identified as a poor emotion regulation strategy (John & Gross, 2004) as it results in avoided events becoming more accessible than they would have been if cognitive control had never been attempted. This reactivation of negative cognition increases vulnerability to depressive episodes, possibility explaining the high relapse rate observed in the disorder. Like rumination, little is known about the cognitive processes associated with individual difference in the frequency of suppression or other emotional regulation strategies. One mechanism implicated in the research, is a person’s ability to control the contents of working memory (Joormann et al., 2007).
Working memory inhibitory control over negative stimuli is implicated in more contemporary models of depression cognition.

**2.1.2. Contemporary frameworks: Impaired cognitive control accounts.**

Contemporary frameworks of information processing in depression have focused on the cognitive mechanisms underlying the predicted negative attention and memory biases. Many propose the idea of deficiencies in executive control structures, namely, inhibitory process of working memory (e.g., Hartle, 1997; Joormann et al., 2007).

Working memory is involved in all of our conscious manipulations. The original model of working memory proposed by Baddeley and Hitch (1974) outlined three main components, (1) the *central executive*, which acts as a supervisory system and controls the flow of information to and from its *slave systems* the (2) *phonological loop* and (3) *visuospatial sketchpad*. These slave systems are said to be short-term storage systems dedicated verbal and visuospatial domains, respectively. The central executive is similar to the construct of a supervisory attentional system regulating thought and goal setting (Norman & Shallice, 1986) and to the construct of attentional control (Engle & Kane, 2004). In 2000, Baddeley added a third slave system to his model, the *episodic buffer*, which is said to hold representations that integrate phonological, visual, and spatial information (see Figure 1). An important characteristic of working memory, and one that differentiates it from long-term memory, is its capacity-limited focus. Successful task performance thus requires the contents of working memory be effectively monitored and updated to reflect task needs. That is, it is reliant on effective inhibitory control over the access and removal of irrelevant stimuli to the task (Hasher & Zacks, 1988; Hasher, Zacks, & May, 1999).
In their meta-analysis of studies of inhibitory processing, Friedman and Miyake (2004) found evidence for three latent inhibition factors. The first, *prepotent response inhibition* refers to the non-controversial form of motor inhibition that requires the suppression of unwanted reflexive behavioural actions. The second, *resistance to distractor interference*, denotes a form of cognitive inhibitory control that resists or resolves interference from information in the external environment that is irrelevant to the current task. The third, *resistance to proactive interference*, is also a form of cognitive inhibition, which involved the ability to resist or remove working memory intrusions that were once, but are no longer, relevant. See Figure 1 for a visual depiction of Baddeley’s (2000) working memory model that has been adapted to include Friedman and Miyake’s (2004) resistance to distractor interference and resistance to proactive interference cognitive inhibitory factors.
Figure 1. A modification of Baddeley’s working memory model adapted to include Friedman and Miyake’s (2004) resistance to distractor interference and resistance to proactive interference cognitive inhibitory factors. Image adapted from Dati, Cutmore, and Shum (2012).
Joormann’s impaired cognitive control model. Joormann et al. (2007) outlined a model of depression that argued that impaired ability to exert top-down inhibitory control over the contents of working memory results in the negative cognitive biases and ruminative response styles related to the disorder. They implicate both deficits in resistance to distractor interference and resistance to proactive interference inhibitory control. Joormann et al. suggests that inhibitory control in these domains results in sustained activation of irrelevant negative stimuli in consciousness, leading to rehearsal of this material and its enhanced long-term memory. Thus, the depressed individual’s cognitive resources are thereby depleted, limiting their ability to reappraise or recall positive information to regulate their affect. Together this serves to sustain and exacerbate depressed mood.

Beck’s neurobiological model of depression. Beck (2008) recently updated his 1967 cognitive model of depression to implicate diminished top-down inhibitory control over negative schemas at the core of depressive cognition and maintenance. As can be seen in Figure 2, Beck (2008) proposes that Amygdala hyperactivity in individuals with genetic predisposition to depression (i.e., polymorphisms of the short allele serotonin transporter gene 5-HTTLPR) is associated with heightened reactivity to negative stimuli (Munafo, Brown, & Hariri, 2008) and elevated Hypothalamic-Pituitary-Adrenal (HPA) activation (Gotlib, Joormann, Minor, & Hallmayer, 2008). This hypersensitivity is postulated to mediate the bottom-up negative information processing bias of mood-congruent information (Dannlowski et al., 2007; Monk et al., 2008). Empirical evidence examining the efficacy of cognitive therapy indicates depressive symptom reduction appears to operate via reduced activation of the Amygdala-Hippocampal subcortical regions and increased activation of higher-order prefrontal cortex (PFC) regions implicated in control of negative emotion (see Clark &
Beck, 2010). In support of these predicted associations, pharmacological interventions for depression appear to influence the initial bottom-up deployment of attention via reduction of the Amygdala-based stimulus appraisal system, whereas psychological interventions influence top-down processing by altering activity in the lateral PFC (Browning, Holmes, & Harmer, 2010).

*Figure 2.* Neurophysiological aspects of Beck's (2008) cognitive model of depression. Image reprinted from Clark & Beck (2010). All rights reserved.
Summary. As illustrated in Figure 3, the cognitive models of depression have demonstrated a progression of philosophy towards defective top-down inhibitory processes as the mediating catalyst (model c) for the traditionally emphasized, bottom-up negative attention and memory biases (models a,b,c). Contemporary cognitive frameworks of depression have focused on the mechanisms underlying the predicted negative attention and memory biases. Many propose the idea of deficiencies in executive control structures. Namely, that inhibitory process of working memory underlie (in)effective emotional regulation in depression (e.g., Beck, 2008; Hartle, 1997; Joormann et al., 2007).

Figure 3. Illustration of interrelations among cognitive biases implicated in the major cognitive models of depression. Image adapted and reprinted from Everaert, Koster, and Derakshan (2012).

The aim of this dissertation was to investigate the association between negative attention and memory biases with cognitive inhibitory control deficits in participants with current and remitted depression. Study 1 and 2 of this dissertation
aim to examine the predictions of mood congruent cognitive biases based on the traditional models of depression. Whereas, Studies 3 and 4 aim to investigate the validity of the contemporary depression model’s claims that working memory inhibitory functioning mediates these bottom-up negative attention and memory biases. Joormann et al.’s impaired cognitive control model was selected to guide the dissertation hypotheses—opposed to other contemporary theories which also implicate working memory deficits (e.g., Beck, 2008)—as its predictions that can be readily measured with behavioural, ERP, and self-report data. This is compared to the genetic and fMRI data that would be required to test the predictions of Beck’s (2008) updated neuro-cognitive model of depression (which is beyond time and financial means for the current work).

Cognition intimately depends on the functioning of the cerebral cortex. Understanding the neural basis of cognition, therefore, offers the potential to increase understanding of cognitive processes, particularly in relation to subtle cognitive activities. The high temporal resolution of ERPs allows the elucidation of the steps of information processing potentially impaired in depression. For this reason the ERP was employed as a key dependent variable in the dissertation studies. To help inform the predictions of these ERP studies, a literature review of the ERP data on emotion information processing in depression is provided. To aid interpretation and critical analysis of these electrophysiological findings, the following section of the dissertation will first offer a summary of the relevant ERP technique and key information processing components.

2.2. Electrophysiological Methodology

Scalp EEG measures reflect summed activity of millions of pyramidal neurons in the neocortex or sub-cortical structures such as the Thalamus (Yamashita, Galka,
Ozaki, Discay, & Valdes-Sosa, 2003). Placement of electrodes of the EEG generally utilise the International 10-20 placement system (Jasper, 1958). This ensures standardisation across studies and laboratories. In the 10-20 placement system, distances between neighbouring electrodes are positioned either 10% or 20% of the total distance between the nasion and inion, as outlined in Figure 4. For identification purposes, each electrode site has a letter to specify its location over the brain lobes: O for occipital lobe, P for Parietal, T for temporal, F for frontal, and C for midline position. Electrode sites are also identified by their number, with right hemisphere electrodes labelled with even numbers, and left hemisphere electrodes labelled with odd numbers.

![Figure 4. 10-20 International placement system. Image adapted and reprinted from Sharbrough et al. (1991).](image)

2.2.1. Utility of the ERP in Cognitive Research

*The event related potential (ERP).* An ERP is a reliable electrical response to a repeatedly presented event, such as a stimulus word. It includes positive or negative
voltage fluctuations in the EEG that result from sensory, motor, or cognitive events (Luck, 2005). However, ERPs are relatively small (1-30 µV) in respect to the background EEG and therefore require the use of signal-averaging techniques for their elucidation (Luck, 2005). In this process, a stimulus cue is repeatedly presented while participants’ EEG data is recorded continuously. The EEG data is aligned at the time point of stimulus onset and averaged together to resolve the ERP from the flux of background EEG. Due to this background EEG being uncorrelated with the stimulus based event, it is cancelled in proportion to the square-root of the number of trials. This approach also extracts the resulting ERP from the non-event-related random noise (e.g., random movement artifacts; Knight, Woods, Neville, 1981). Averages with good signal-to-noise ratios can be achieved with approximately 30 trials if the stimulus delivery rate is relatively slow (Knight et al., 1981; Luck, 2005). This ERP extraction technique is illustrated in Figure 5.

Figure 5. The ERP extraction method. Image from Knight et al. (1981).
As shown in Figure 6, a 2000 ms signal-averaged trace of EEG may contain several ‘components’, each relating to individual perceptual and cognitive processes. Components are typically named by their voltage—P for positive and N for negative—and by their order of appearance (e.g., P1, N1, P2, N2, P3) or time of appearance (e.g., P100, N100, P200, N200, P300).

Figure 6. ERP components from an averaged waveform. The dotted line represents stimulus onset. LPP refers to late positive potential.

There are three significant aspects of the ERP waveform: amplitude, latency, and scalp topography. Amplitude provides an indication of the magnitude of neural activation, possibly reflecting the raw number of co-activated neurons (Luck, 2005). Latency reveals the temporal processing of stimuli, with early components (< 100 ms) being sensitive to exogenous properties of the stimulus and later components (> 100 ms) being sensitive to various endogenous cognitive factors such as attention, recognition, and evaluation (Luck, 2005). Scalp distribution, or topography, provides information on potential cortical neural network activity required for the cognitive
process (Friedman, Cycowicz, & Gaeta, 2001). When elicited by different stimuli, tasks, or in different samples, the comparisons of these three properties of the ERP allows researchers to conclude whether they generate different patterns of neural activity and, hence, indicate different cognitive processes (Luck, 2005). The main ERPs employed in studies of information processing are summarised in Table 1.

Table 1

Key ERP Components used in Information Processing Research

<table>
<thead>
<tr>
<th>ERP Component</th>
<th>Theorised Cognitive Process</th>
<th>Latency and Topography</th>
<th>Suggested Neural Generators</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Error Related Negativity (ERN) or Ne</td>
<td>Conflict detection</td>
<td>50-150 ms Frontal and central sites</td>
<td>Anterior cingulate cortex (ACC)</td>
<td>(Alexopoulos et al., 2007; Chiu &amp; Deldin, 2007; Ruchsow et al., 2008)</td>
</tr>
<tr>
<td>N1</td>
<td>Pre-attentive perceptual discrimination, especially for auditory stimuli</td>
<td>80-120 ms Frontal and central sites</td>
<td>Primary and association auditory cortices; superior temporal gyrus in Herschel’s gyrus; Planum Temporale</td>
<td>(Godey, Schwartz, de Graff, Chauvel, &amp; Liégeois-Chauvel, 2001; Spreng, 1980; Zouridakis, Simos, &amp; Papanicolaou, 1998)</td>
</tr>
<tr>
<td>P1</td>
<td>Visual processing, especially for facial perception</td>
<td>70-130 ms Occipital-temporal cortex</td>
<td></td>
<td>(Herrmann, Ehlis, Ellgring, &amp; Flaggter, 2005)</td>
</tr>
<tr>
<td>P2</td>
<td>Hypervigilance</td>
<td>150-270 ms Frontal and central sites</td>
<td>Medial frontal cortex</td>
<td>(Rossignol, Philippot, Crommelinck, &amp; Campanella, 2008; Shestyuk &amp; Deldin, 2010)</td>
</tr>
<tr>
<td>N2a</td>
<td>Automatic detection of deviant stimuli</td>
<td>150-250 ms Frontal sites</td>
<td>Supra-temporal auditory cortex and the right frontal lobe.</td>
<td>(Folstein &amp; van Petten, 2008; Lim et al., 1999)</td>
</tr>
<tr>
<td>N2b</td>
<td>Orienting response</td>
<td>150-350 ms Central maximum</td>
<td>Auditory and visual cortex.</td>
<td>(Folstein &amp; van Petten, 2008; Lim et al., 1999)</td>
</tr>
<tr>
<td>N400</td>
<td>Semantic incongruence</td>
<td>300-500 ms Left frontal and temporal Maximum</td>
<td>Left posterior temporal cortex, left superior temporal gyrus, and left inferior frontal cortex</td>
<td>(Khateb, Pegna, Landis, Mouthon, &amp; Ammoni, 2010; Kutas &amp; Hillyard, 1980, 1982)</td>
</tr>
<tr>
<td>ERP Component</td>
<td>Theorised Cognitive Process</td>
<td>Latency and Topography</td>
<td>Suggested Neural Generators</td>
<td>Reference</td>
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<tr>
<td>N450/Medial Frontal Negativity (MFN)</td>
<td>Cognitive inhibition as measured by the Stoop Task</td>
<td>450 – 600 ms Frontal-central maximum</td>
<td>Inferior frontal cortex and ACC</td>
<td>(McNeely, Christensen, West, &amp; Alain, 2003; McNeely, Lau, Christensen, &amp; Alain, 2008; Tays, Dywan, Mathewson, &amp; Segalowitz, 2008; Vanderhasselt &amp; De Raedt, 2009)</td>
</tr>
<tr>
<td>Novelty or P3a</td>
<td>Orientating attention towards deviant stimuli</td>
<td>220–350 ms Frontal-central maximum</td>
<td>Relatively unknown; suspected ACC and Hippocampal regions</td>
<td>(Friedman et al., 2001; Polich, 2003, 2007)</td>
</tr>
<tr>
<td>Classical or P3b</td>
<td>Context updating</td>
<td>300–700 ms Parietal maximum</td>
<td>Parietal-temporal junction, Hippocampal sources, and the Cingulate cortex.</td>
<td>(Friedman et al., 2001; Polich, 2003, 2007)</td>
</tr>
<tr>
<td>Late Positive Potential (LPP)</td>
<td>Emotional regulation</td>
<td>300 – 4000 ms Parietal and occipital regions</td>
<td>Visual cortical structures such as lateral occipital, temporal, and parietal visual areas</td>
<td>(Hajcak, MacNamara, &amp; Olvet, 2010; Sabatinelli, Lang, Keli, &amp; Bradley, 2007)</td>
</tr>
<tr>
<td>Slow Wave (SW)</td>
<td>Sustained processing</td>
<td>600-5000 ms Prefrontal and parietal sites</td>
<td>Left and parietal regions</td>
<td>(Deldin, Deveney, Kim, Casas, &amp; Best, 2001; Deveney &amp; Deldin, 2004; Hansell et al., 2001; Shestuyk, Deldin, Brand, &amp; Deveney, 2005)</td>
</tr>
<tr>
<td>Mid-frontal Old/New effect (FN400)</td>
<td>Explicit episodic memory (most likely reflecting familiarity)</td>
<td>200–400 ms Left frontal and central sites</td>
<td>Left posterior Cingulate, medial temporal cortex.</td>
<td>(Curran &amp; Friedman, 2004; Dietrich et al., 2000; Kayser et al., 2010; Rugg &amp; Curran, 2007)</td>
</tr>
<tr>
<td>Late Positive Complex (LPC) Old/New effect</td>
<td>Explicit episodic memory (most likely reflecting recollection)</td>
<td>400–800 ms Left parietal and central sites</td>
<td>Left posterior Cingulate, medial temporal cortex.</td>
<td>(Curran &amp; Friedman, 2004; Dietrich et al., 2000; Kayser et al., 2010; Rugg &amp; Curran, 2007)</td>
</tr>
<tr>
<td>ERP Component</td>
<td>Theorised Cognitive Process</td>
<td>Latency and Topography</td>
<td>Suggested Neural Generators</td>
<td>Reference</td>
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</tr>
<tr>
<td>Nogo-N2</td>
<td>Inhibitory control (most likely reflecting conflict monitoring)</td>
<td>220-380 ms Frontal sites</td>
<td>Rostral ACC and the Nigrostriatal dopamine-system</td>
<td>(Beste, Willemssen, Saft, &amp; Falkenstein, 2010; Chiu et al., 2008; Smith, Smith, Provost, &amp; Heathcote, 2010)</td>
</tr>
<tr>
<td>Nogo-P3</td>
<td>Inhibitory Control (most likely reflecting conflict regulation)</td>
<td>310-410 ms Frontal –central maximum</td>
<td>Rostral ACC, Obito-Fronto-Cortex, and Mesocortico-limbic dopamine system</td>
<td>(Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010)</td>
</tr>
<tr>
<td>Go-P3</td>
<td>Attention</td>
<td>300-700 ms Parietal maximum</td>
<td>Parietal-temporal junction, Hippocampal sources, and the Cingulate cortex.</td>
<td>(Chiu et al., 2008)</td>
</tr>
</tbody>
</table>

Relevant ERP methodologies and their evoked components. This section will provide a brief review of the most commonly employed methodologies and their evoked components implicated in the emotional information processing literature. It will then discuss the implications that stimuli characteristics, participant characteristics, and ERP measurement and extraction procedures have on these ERPs. Stimuli characteristics include things such as valence and use of verbal versus pictorial or facial stimuli. Sample characteristics include things such as gender, depression and/or medication status. The ERP measurement and extraction procedures include electrode reference placement, and use of principal component analysis (PCA) versus epoched ERP point extraction processes. This material will assist in critical analysis of ERP literature on information processing biases and deficits in depression. It also forms the arguments for the dissertation selected ERP methodologies.

The oddball paradigm. The oddball paradigm is a frequently employed ERP measure of attention processes in the depression mood-congruency literature (e.g., Illardi, Atchley, Enloe, Kwasny, & Garratt, 2007; Yang, Zhu, Wang, Wu, & Yao,
As such, it is important to have an understanding of this task and its evoked components. The traditional oddball paradigm typically involves a Bernoulli (randomised) presentation of two classes of stimuli. One is a frequently occurring standard stimulus, and the other is an infrequently occurring novel or oddball stimulus (Fabiani, Gratton, Karis, & Donchin, 1987). Within this paradigm, the early succession of ERP components elicited by all stimuli is the N1, P2, and N2, as shown in Figure 7. As summarised in Table 1, these initial components are argued to represent the pre-attentive activation of the sensory input to subcortical relay nuclei and cortical sensory areas (Friedman et al., 2001). For the oddball stimulus, however, Figure 7 shows that these early processing waves are followed by the P300 component (Fabiani et al., 1987; Friedman et al., 2001; Polich, 2007).

**Figure 7.** Schematic representation of context updating hypothesis of P3 generation. Image from Polich (2003). All rights reserved.

The P300. The P300 is an endogenous ERP, said to appear most typically around 300 to 900 ms following the presentation of a meaningful stimulus (Polich 2003, 2007). Its peak amplitude usually ranges from 5 µV to a usual limit of 20µV for auditory and visual evoked potentials (Polich, 2003, 2007). Within the oddball paradigm, the amplitude of the P300 has shown to be inversely proportional to the
frequency of the occurrence of the oddball stimulus. It is largest when this event is the rarest (Donchin & Coles, 1988; Linden, 2005).

Some studies have identified neural generators of the P300 in the temporal-parietal junction (Knight, Scabini, Woods, & Clayworth, 1989; Verleger, Heide, Butt, & Kömpf, 1994; Yamaguchi & Knight, 1991). Others have identified neural generators in frontal cortical regions, the Hippocampus and the Amygdala (Ardekani et al., 2002; Kiehl & Liddle, 2001; Menon, Ford, Lim, Glover, & Pfefferbaum, 1997). Test–retest correlation coefficients for oddball task P300 range from .50 to .80 for peak amplitude and from .40 to .70 for peak latency (Fabiani et al., 1987; Segalowitz & Barnes, 1993; Walhovd & Fjell, 2002). Two subcomponents of the P300 have been identified: the P3a and the P3b (see Figure 8; Polich & Craido, 2006; Polich, 2007).

The three-stimulus oddball paradigm, as its name implies, presents three classes of stimuli. These include: frequent, attended standards, less frequent, attended oddball/targets, and perceptually novel distracters (Polich, 2004). For these novel distractor stimuli a frontal-central P300 is elicited, called the P3a or the novelty P300 (Polich, 2004). This potential has relatively short peak latency and habituates quickly (Courchesne, Hilyard, & Galambos, 1975; Knight, 1984). It is said to reflect frontal lobe activity related to the Hippocampus (Knight, 1996). With repeated stimulus presentation, the P3a decreases in magnitude, suggesting it is related to early attention responses, especially orienting (Knight, 1984; Kok, 2001; Rushby, Barry, & Doherty, 2005).

The P3b is another subcomponent of the P300. The P3b is thought to reflect current memory mechanisms triggered when the mental model of the stimulus environment is refreshed and revised (Donchin and Coles, 1988). This is known as context updating (see Figure 8; Donchin & Coles, 1988). Larger P3b magnitude
corresponds to a mismatch between the received and the expected stimulus information based on current working memory storage. Underlying the context-updating theory is the belief that the P3b is comparable to the amount of attention devoted to a given task. That is, stimuli that involve greater attention yield larger P3b amplitude (Gordeev, 2007; Kok, 2001; Polich, 2007; Miyakoshi, Nomur, & Ohira, 2007; Wronka, Kuniecki, Kaiser, & Coenen, 2007). This assumption is consistent with the research showing that remembered stimuli elicit greater P3b amplitudes during encoding than subsequently forgotten stimuli (Fabiani, Karis, & Donchin, 1986; Karis, Fabiani, & Donchin, 1984; Johnson, 1995). Figure 8 shows these two main theories underlying the P3a and P3b.

Figure 8. Schematic representation of theory underlying P3a and P3b activation. Image reprinted from Polich (2003).

The Old/New paradigm. Study 2 (Chapter 4) examined explicit memory processes using the ERP Old/New paradigm. As such, to appraise the study's findings it is paramount to have an understanding of the task and its associated ERP components. The ERP Old/New paradigm measures brain activity during a recognition test in which participants have to verify stimuli as presented for the second
or first time. These represent the old and new stimulus respectively. There are two forms of the Old/New paradigm: the continuous recognition protocol or the study-test protocol. In the continuous recognition protocol, the first and second stimulus presentations occur intermixed in one testing session. In the study-test protocol first presentations of stimuli occur in a learning phase, while second presentations occur in a latter test, in which these stimuli have to be recognised from a list of old target and new distractor stimuli.

Proponents of the dual-process theory of memory argue that recognition can be fractionated into recollection and familiarity processes (Rugg & Curran, 2007; Yonelinas et al., 2002). Recollection is evidenced when explicit memory for contextual information can be reconstructed about the stimulus (Gardiner & Java, 1993; Jacoby, 1991). Conversely, familiarity involves a feeling or sensation that a stimulus has been previously observed, but this is not accompanied by any contextual information about that experience (Jacoby, 1991; Mandler, 1980; Yonelinas, 1999).

*The ERP Old/New effects.* The ERP Old/New effect concerns the difference between the ERPs elicited by old and new stimuli. Evidence has accumulated to suggest that the ERP Old/New recognition effect can be fractionated into two distinct components that constitute familiarity and recollection processes. These components include the mid-frontal Old/New effect (FN00) and the late positive complex Old/New effect (LPC), respectfully (Friedman, 2000; Friedman & Johnson, 2000; Rugg, 1995; Rugg & Allan, 2000; Rugg & Curran, 2007; Yonelinas, 2002). The parietal Old/New effect produces an ERP signature characterised by enhanced positive amplitudes for correctly identified stimuli (Friedman, 1990; Rugg, 1987; Rugg & Nagy, 1989). It has an onset of approximately 400-500 ms post-stimulus presentation and frequently exhibits a left-hemisphere maximum (Figure 9, b; Rugg & Curran, 2007).
Functionally, the parietal Old/New effect is thought to reflect retrieval processes in discrimination of old from new items in explicit memory (Harris, Cutmore, O’Gorman, Finnigan, & Shum, 2009; Paller & Kutas, 1992; Rugg & Nagy, 1989). The mid-frontal Old/New effect or the FN400 is often observed in the same time epoch as the parietal Old/New effect, but with more anterior topography. The amplitude of the FN400 tends to be more negative for new than old stimuli (Curran, 2000). Supporting the theory that FN400 is linked to familiarity-driven cognitive processes, new distractor words that share similar features with old words evoke analogous mid-frontal ERPs (see Figure 9a; Curran, 2000; Nessler, Mecklinger, & Penney, 2001). Figure 9 displays the FN00 and LPC Old/New ERPs.

![Figure 9](image)

**Figure 9.** (a) ERPs evoked at frontal cites with the mid-frontal Old/New effect indicated by the arrow. (b) ERPs evoked at left-parietal sites with the parietal Old/New effect indicated by the arrow. Data and image from Curran (2000).

Another ERP associated with memory processes is the late parietal negativity (LPN). The LPN is a functionally heterogeneous ERP, which appears to comprise subcomponents sensitive to retrieval ease (600 – 1200 ms) and post-retrieval
judgments (1200 – 1900 ms; Herron, 2007). It is thought to reflect top-down signals emanating in the PFC that select the task-relevant attributes to search and to bind with the recognized items in memory (Miller & Cohen, 2001; Shimamura, 2000). The LPN is enhanced when old stimuli are not readily recovered during episodic memory retrieval and is associated with longer reaction times (see Johansson & McKlinger, 2003 and Herron, 2007 for reviews). It appears independent of memory performance success (e.g., does not correlate with behavioral data). Given that the LPN magnitude is larger when search conflict is higher (Curran, DeBuse, & Leynes, 2007), it appears to reflect memory search attempt (Friedman et al., 2005).

The Go/Nogo paradigm. Study 3 (Chapter 5) examined working memory inhibitory control using a modified version of the Go/Nogo paradigm. The Go/Nogo paradigm is among the most well characterised measures of response inhibition and can be easily adapted to include measures of cognitive inhibition (Aron, 2007; Chiu et al., 2008; Dillon & Pizzagalli, 2007; Smith, Johnstone, & Barry, 2008). In the task, a series of Go cues are presented to which participants are required to make a response as quickly as possible. These stimuli evoke a parietal P3, which is said to reflect attentional processes, similar to that underlying the conventional P3b (Falkenstein, Hoormann, & Hohnsbein, 1999). Go cues are intermixed with Nogo cues, to which the participant is required to prevent any behavioural or cognitive responses (inhibitory processing). Electrophysiological Go/Nogo investigations have reliably linked two ERP components to response inhibition, the Nogo-N2 and the Nogo-P3. They are thought to reflect conflict monitoring and conflict management, respectively.

The Nogo-N2. The Nogo-N2 is a negative going shift over the frontal-central scalp locations, with a peak between 200 to 400 ms post-stimulus (Chiu et al., 2008; Falkenstein et al., 1999; Folstein & Van Petten, 2008; Kirmizi-Alsan et al., 2006;
Kopp, Rist, & Mattler, 1996; Nieuwenhuis, Yeung, Cohen, 2004). It is said to operate at a stage prior to motor performance (Falkenstein et al., 1999; Folstein & Van Petten, 2008; Kaiser et al., 2003; Kim, Kim, Yoo, & Kwon, 2007). Traditionally the Nogo-N2 is said to reflect inhibition of prepotent response tendencies. However, there is disagreement as to whether this ERP represents conflict monitoring (Nieuwenhuis et al., 2004). Supportive of the conflict monitoring interpretation, previous work shows larger Nogo-N2s than Go-N2s are observed in tasks that require an overt response such as a key press to be withheld compared to tasks that require a covert response such as silent counting to be withheld (Bruin & Wijers, 2002; Pfefferbaum, Ford, Weller, & Kopell, 1985). The Nogo-N2 correlates to the rarity of the stimulus event (Donkers & van Boxtel, 2004; Nieuwenhuis et al., 2003). It seems to be sensitive to manipulations of stimulus congruency, with larger Nogo-N2 evoked to Nogo stimuli with similar characteristics to go stimuli (presumably due to greater response conflict; Bartholow et al., 2005; Ruchsow et al., 2008). The Nogo-N2 is also enhanced by the requirement to respond quickly (Jodo & Kayama, 1992), and is observed to be larger in participants with low false alarms compared to those with high false alarms (Falkenstein et al., 1999). That is, it is larger in participants with good conflict monitoring compared to those with poor conflict monitoring.

Two areas of the prefrontal cortex are critical in cognitive control. This includes the anterior cingulate cortex (ACC: van Veen & Carter, 2002) and the dorsolateral prefrontal cortex (DLPFC; Mansouri, Buckley & Tanaka, 2007). The ACC appears specifically sensitive to experimental conditions that require cognitive control (Botvinick et al., 2001; 2007; van Veen & Carter, 2002, 2007; Yeung, Botvinick & Cohen, 2004). It shows increased activation in tasks where conflict is detected (e.g., Stroop procedures) or when inhibition of prepotent responses are
required (e.g., Go/Nogo tasks; Botvinick et al., 2001). The ACC activation is greater when high-conflict trials are infrequent as compared to when they are frequent (Braver, Barch, Gray, Molfese, & Snyder, 2001; Jones, Cho, Nystrom, Cohen, & Braver, 2002). This suggests that the ACC is no longer required once control processes have been put into place. Thus, it appears that the ACC’s role in cognitive control is largely evaluative, rather than regulative. It is likely to serve to identify conflict then issues a call to other brain regions to carry out its resolution, rather than carry out the conflict resolution itself (Botvinick et al., 2001, Kerns, Cohen, MacDonald, Cho, Stenger & Carter, 2004. van Veen & Carter, 2002). The ACC seems to have strong connections to the DLPFC, which is understood to function to regulate cognitive conflict (Botvinick et al., 2001; Mansouri, Buckley & Tanaka, 2007). Orientating of attention processes and regulating behavioural output (e.g., updating rule changes during a Wisconsin Card Sorting task) are associated with increased activation of the DLPFC (Botvinick et al., 2001; Mansouri et al., 2007). Thus, it is thought to support appropriate “response selection” processing, thereby regulating cognitive conflict (Banich et al., 2001; Bench et al., 1993; Carter et al., 1995; Taylor et al., 1997). Previous work in the normative literature shows that under conditions of equi-probable presentation of Go and Nogo stimuli, the DLPFC becomes activated as it is required for response inhibition to regulate the cognitive conflict (Lavric, Pizzagalli, & Forstmeier, 2004).

The Nogo-P3. Pfefferbaum et al. (1985) found that following Nogo trials, the Nogo-N2 component is usually followed by a positive deflection around 300 to 700 ms post-stimulus also at frontal sites. He termed this component the Nogo-P3. The Nogo-P3 is proposed to reflect conflict regulation processes of inhibitory control (Herrmann, Jacob, Unterecker, & Fallgatter, 2003; Schmajuk, Liotti, Busse, & Woldorff, 2006), as well as processes related to motor inhibition (Smith, Johnson, & Barry, 2006; Smith et al., 2008; Zordan, Sarlo, & Stablum, 2008). The topography of
the component differs from the Go-P3 in that the Go-P3 is observed to be comparatively larger and has a more parietal distribution. The topography and morphology of the Nogo-P3 appears somewhat similar to the novelty P3a, which has likewise been linked to inhibitory processes as it is elicited by deviant stimuli that can be conceptualised as task-irrelevant distracters (Simons, Graham, Miles, & Chen, 2001). The Nogo-P3 versus Go-P3 topographic difference has been shown to be stable at differing stimulus presentation rates (one second: Simson, Vaughn, & Ritter, 1977; two seconds: Roberts, Rau, Lutzenberger, & Birbaumer, 1994), varied probability rates (Pfefferbaum & Ford, 1988), and different modalities of presentation (Tekok-Killic, Shucard, & Shucard, 2001). Thus, the consistency of the differences between these components support the hypothesis that the Nogo-P3 and the Go-P3 corresponds to distinct neuronal processes produced by distinct brain systems reflecting different cognitive processes (Pfefferbaum & Ford, 1988). Figure 10 presents the Go and Nogo ERPs evoked in a sample of young, healthy adults.
Figure 10. The Nogo-N2 and Nogo-P3 stimulus-locked at FCz for healthy, young adults. Go trails indicated by green line and No-Go trials by the red line. Combined scalp topography of these potentials, and the Go-P3, is provided to the left of the figure. Data and image from Beste et al. (2010).

Neural generators and circuits of the Nogo-N2 and Nogo-P3. Cortical generators of the Nogo-N2 have been modelled in the ventral PFC (vPFC; Bokura et al., 2001; Plizska, Liotti, & Woldorff, 2000), the orbitofrontal cortex (OFC; Bokura, Yamaguchi, Kabayashi, 2001; Kamarajan et al., 2006) and the ACC (van Veen & Carter, 2002), regions all associated with conflict monitoring processes (Kringelbach & Rolls, 2004; Ochsner et al., 2005; Paus, 2001). These two sub-mechanisms of response inhibition also appear modulated by distinct Basal Ganglia pathways (Beste et al., 2010). The pre-motor inhibitory mechanism indexed by the Nogo-N2 seems to be modulated by the Nigrostriatal circuit. The inhibitory management system indexed by the Nogo-P3 on the other hand appears reliant on the Mesocortico-Limibic Dopaminergic system (Beste et al., 2010). A visual depiction of this data is presented in Figure 11.
Figure 11. Illustration of the neural generator and dopaminergic circuits associated to the sub-processes of response inhibition as measured by the Nogo-N2 and Nogo-P3. The nigrostriatal dopaminergic-system is highlighted grey, and appears linked to inhibitory monitoring processes (Nogo-N2). The mesocortico-limbic dopaminergic circuits is highlighted black, and appears linked to inhibitory management processes (Nogo-P3). Image reprinted from Beste et al. (2010).

Controversies over the interpretations of the Nogo-N2 and Nogo-P3. There exists some controversy over the functional significance of the Nogo-N2 and Nogo-P3. In two recent studies (Smith et al., 2006; Smith, Johnstone, & Barry, 2007) it was proposed that the N2 reflects neither response inhibition nor conflict monitoring. In a cued Go/Nogo task, the N2 was found to be largest when response preparation was at a minimum, that is, following Nogo cues than Go cues. This was at odds with a response inhibition hypothesis which predicts that Nogo-N2 amplitudes should increase following Go rather than Nogo cues. In addition, larger Nogo-N2 amplitudes were associated with validly but not invalidly cued trials, which also contradicts the response conflict view of the N2 (Smith et al., 2007). Hence, the functional
significance of the Nogo-N2 is still not entirely clear and needs to be considered in the following literature and Study 3 interpretations.

In a later investigation, Smith et al. (2008) examined the contributions of movement-related potentials to the Nogo-N2 and Nogo-P3 by directly comparing count and press responses. Participants \( N = 20 \) were required to either mentally count or respond by pressing a button following presentation of frequent (60%) or rare (20%) Go stimuli and withhold all mental and physical responses to the equally rare Nogo stimuli (20%). In comparison to the press responses, count responses did not include motor preparation and implementation mechanisms. The researchers found reliable Nogo-N2 and Nogo-P3 effects for both the count and press conditions. When comparing these ERPs the researchers further found that the Nogo-P3 was amplified during the press task compared to the count task, whereas the Nogo-N2 did not differ. In light of these results, the authors argued that the Nogo-N2 may indicate a non-motoric level of inhibition. That is, being the manifestation of the recognition of the need for inhibition, or the conflict arising from that recognition. Given that the Nogo-P3 was evident for both covert and overt response inhibition, the authors argued that it reflects inhibition of both cognitive processes and movement-related potentials.

Specifically, they isolated a positive potential maximum over central regions at 220-260ms (the known time taken to prevent a response) contralateral to the responding hand. This positive activation was present on press but not count Nogo trials. Therefore, Smith et al. (2008) claimed that this positivity may reflect the motoric inhibition process embedded within the Nogo-P3. This research strongly suggests the importance of counterbalancing the response hand to reduce motor related laterality effects on this component. The impact of movement related artifacts also need to be taken into consideration during interpretation of this ERP.
2.2.2. Effects of Participant and Methodological Characterises on the ERP

Incorporating affectively salient stimuli into ERP investigations may provide insights into emotional information processing biases and deficits in depression (e.g., Deldin, Shestyk, & Chiu, 2003; Williams et al., 1997). However, prior to appraisal of this research it is first paramount to understand the effect that depression status is observed to have on ERPs in non-emotional paradigms. Further, it is necessary to avoid confusing ERP differences due to the hypothesised psychological dynamics with subtle differences due to gender effects, stimulus dynamics, or ERP protocols (Luck, 2005). Thus, the next section will review the impact that depression, gender differences, anxiety comorbidity, task design, and electrophysiological recording and extraction procedures have on evoked ERPs.

Sample characteristics.

Depression status. Electrophysiological studies of information processing typically show reduced component amplitudes in depressed samples (see Bruder, Kayser, & Tanke, 2013 for a review). This reduced ERP amplitude is observed in both early (P1, N1, N2a, N2b, P2, P3a: Bruder et al., 1998; Burkhart & Thomas, 1993; el Massioui & Leservre, 1988; Knott & Lapiette, 1987; Ogura, Nageishi, Omura, & Fukao, 1993; Pause et al., 2003; Pierson et al., 1996; Sandman, Gerner, O'Halloran, & Isenhart, 1987) and late latency components (P3b, mean Cohen's $d = 0.85$: Anderer, Saletu, Smlitsch, & Pascual-Marqui, 2002; Kawasaki, Tanaka, Wang, Hokama, & Hiramatsu, 2004; Roschke & Wanger, 2003; Urretavizcaja et al., 2003). Given the ERP amplitude is regarded as a marker of neural activation of mental effort, these findings suggest that depression is marked by deficits in cognitive exertion for task performance. This might explain the attention, concentration, and memory impairments associated with the disorder. However, results are not always consistent
with this simple view, with some studies finding enhanced ERP amplitudes in depressed samples (e.g., Blackwood et al., 1987; Bruder et al., 1995; Ogura et al., 1993; Pierson et al., 1996; Sara et al., 1994). Bruder et al. (2013) argues that differences in the characteristics of patient samples, such as clinical subtype (e.g., bipolar I or II; melancholic, psychotic, atypical depression), participant age, medication status, or the presence of unaccounted comorbid diagnoses can explain a considerable amount of error variance between ERP investigations with depressed individuals. It should also be noted that gender differences have been found in depression and in emotional information processing.

**Gender differences.** Behavioural data suggest that females show enhanced language-based abilities, especially during episodic memory tasks of word stimuli (Herlitz, Airaksien, & Nordstrom, 1999; Herlitz, Nilsson, & Backman, 1997; Maitland, Herlitz, Nyberg, Backman, & Nilsson, 2004). The neurobiological source for this gender difference remains vague. Some have argued that men and women differ in the neural lateralisation of language functions, with women showing reduced asymmetry in semantic decision making (Shaywitz, Shaywitz, Pugh, & Constable, 1995; Pugh et al., 1996; Baxter et al., 2003; but see Frost et al., 1999 and Sommer, Aleman, Bouma, & Khan, 2004). Gender-specific lateralisation differences in ERP tests of verbal (Walla, Hufnagl, Lindinger, Deecke, & Lang, 2001; Hill, Ott, & Weisbrod, 2005; Ortigue, Thut, Landis, & Michel, 2005), pictorial (Lithari et al., 2010), and facial (Gullem & Mograss, 2005) stimuli have been reported. Gender differences in cognitive processing appear most pronounced in judgments concerning emotional stimuli (Schirmet & Kotz, 2003; Schneider, Habel, Kessler, Salloum, & Posse, 2000; Killgore & Yurglun-Todd, 2001; Lee et al., 2002). Relative to males, females evoke greater ERP amplitudes to unpleasant and high arousing stimuli (Lithari
et al., 2010). Using an oddball task with valanced facial stimuli, Campanella et al. (2004) suggested that gender differences in emotional appraisal and elaboration appears to originate at the early stage of information processing where attention gates filter sensory inputs. That is, females attend and respond to emotional stimuli earlier in the processing stream than men (Lithari et al., 2010). Gender differences in ERP morphology have been linked to hormonal effects (Nolen-Hoeksema & Hilt, 2009), stronger right Amygdala activation to emotional events in females (Canli et al., 2002; Hofer et al., 2006; Wrase et al., 2003), emotional appraisal differences between genders (Garcia-Garcia et al., 2008; LeDoux, 2000; Li et al., 2008); and to increased ruminative responses subsequent to negative life events in women compared to men (Nolen-Hoeksema & Jackson, 2001; Nolen-Hoeksema, Larsen, & Grayson, 1999). This enhanced reactivity to negative events may represent a cognitive risk factor in depression specific to women. Another risk factor implicated in the literature is comorbid mental health condition, with the most prevalent being presence of anxiety disorders.

**Depression comorbidity with anxiety disorders.** Depression and anxiety are highly comorbid disorders (Kessler et al., 2007). Thus, in order to maintain ecological validity it is argued that researchers should not examine depression or anxiety in isolation (Beuke, Fischer, & McDowall, 2003; Ingram, 1989; Ingram & Hamilton, 1999). Although the two disorders look closely related, they are theorised to be associated with different effects on information processing (Williams et al., 1997). Without careful methodological considerations it is difficult to disentangle the effects of depression from anxiety. Thus, it is paramount that adequate psychometrics are utilised that are particularly suitable in discriminating anxiety from the depression. The Depression Anxiety Stress Scales (DASS-42 and DASS-21:}
Lovibond & Lovibond, 1995) and structured clinical diagnosis have shown marked benefit here. Data from EEG studies have shown differences in hemispheric asymmetries in individuals’ with MDD as compared to those with comorbid MDD and anxiety disorders (Bruder et al., 1997; Bruder, Wexler, Stewart, Prince, & Quitkin, 1999). Specifically, MDD appeared characterised by greater left temporal-parietal activation (greater alpha EEG), which is less pronounced in individuals with anxiety issues or depression and anxiety comorbidity. The extent of the effect that anxiety comorbidity has on ERP data in the depressed client is limited. One ERP study in this area was by Bruder and colleagues (2002). Using an auditory oddball task, the research observed greater P3 in patients with anxiety disorders as compared to control, depressed patients, or patients with comorbid depression and anxiety. Sympathetic autonomic nervous system hyper-arousal in anxiety disorders appears associated with increased $\alpha_2$-noradrenergic antagonistic yohimbine (Turetsky & Fein, 2002). Thus, Bruder et al. argued that this may result in enhanced orientating and hence increased P3a amplitudes in their anxiety sample. Individuals with comorbid depression and anxiety were found to exhibit enhanced P3b amplitudes compared to control participants, or to those having either disorder alone. This P3b modulation may reflect increased mental effort required to overcompensate for the comorbid patient’s earlier processing deficit (Bruder et al., 2002). These findings require replication, but nonetheless draw attention to the importance of examining depression-anxiety comorbidity in ERP information processing investigations.

**Stimulus characteristics.**

**Valence and arousal characteristics.** Neuroimaging studies have reported that divergent neural networks are implicated in the processing of valence and arousal characteristics of emotional information (Canli, Desmond, Zhao, Glover, & Gabrieli,
1998; Dolcos & Cabeza, 2002; O’Doberty, Kringelbach, Rolls, Hornak & Andrews, 2001; O’Doberty, Critchley, Deichmann, & Dolan, 2003). Specifically, the left and ventromedial prefrontal regions appears activated during processing of positive cues; while the right and lateral Orbital prefrontal regions appears activated during processing of negative cues (Canli et al., 1998; Dolcos & Cabeza, 2002; O’Doberty et al., 2001; O’Doberty et al., 2003).

Valence and arousal effects are observed in the ERP. The ERP valence effect describes the observation that emotional stimuli reliably modulate ERP components, appearing as early as 100 ms post stimulus presentation (Esslen, Pascual-Marqui, Hell, Kochi, & Lehmann, 2004; Ortigue et al., 2004; Pizzagalli et al., 2002; Pourtois, Grandjean, Sander, & Vuilleumier, 2004). The ERP arousal effect describes the enhanced positivity occurring around 300-400 ms following presentation of high-arousing compared to low-arousing stimuli (Chiu et al., 2008; Codispoti, Ferrari, & Bradley, 2006; Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Delplanque, Silver, Hot, Rigoulot, & Sequeira, 2006; Dolcos & Cabeza, 2002). In their emotional Go/Nogo study, Chiu et al. (2008) observed both positive and negative Go words to elicit enhanced P3 responses compared to neutral words, possibly reflecting greater attentional engagement by motivationally salient stimuli. Delplanque et al. (2006) observed the valence effect in both the P3a (333-384 ms) and P3b (439-630 ms) components in their emotional oddball paradigm, with arousal effects only observed in the P3b. However, both of these studies utilised neutral stimuli as the comparison condition to examine the arousal effect. This introduces an experimental confound as the two arousal conditions, high versus low, were not matched on the valence dimension. Whenever possible, ERP researchers should apply the same physical stimuli conditions to avoid confounds (The Hillyard Principle; Luck, 2005). Thus, for
the current collection of studies the positive and negative stimuli were matched on arousal ratings.

**Stimulus type.** Facial, pictorial, and word stimuli can also stimulate differing processing networks (Eimer, 2000; Halgren, Raij, Marinkovic, Jousaki, & Hari, 2000; Liu, Higuchi, Marantz, & Kanwisher, 2000; Rossion, Joyce, Cottrell, & Tarr, 2003). This effect is associated with divergences in ERP modulation, as early as 130-170 ms post stimulus (Rossion et al., 2003). These ERP differences are complemented by observed hemisphere differences in object processing. Faces appear primarily processed in the right hemisphere, words in the left hemisphere, and pictorial stimuli seem to implicate bilateral activation (Rossion et al., 2003). However, the right hemisphere is also preferentially activated in response to emotional word stimuli (Atchley, Illardi, & Enloe, 2003). Data from behavioural, fMRI, and lesion studies also support a distinction between faces, objects, and word processing at both the functional and neural level (Farah, 1994). For instance, some patients with brain damage have been observed to show an inability to read printed words in the absence of facial or object recognition deficits or other form of language processing problems (e.g., intact communication skills; Farah, 1994). Impaired ability to interpret facial information is observed in the absence of other pictorial or verbal processing deficits. Similarly, object agnosia without prosopagnosia is observed wherein the individual cannot correctly identify objects by visual inspection, but show no problems with verbal or facial recognition (e.g., Moscovitch, Winocur, & Behramm, 1997).

The above research recommends that caution be placed on collating ERP data from research utilising different stimulus categories. Although some have argued that pictorial or facial stimuli may have more ecological validity than word stimuli, they are also more complex. This means that arousal and valence characteristics are more
difficult to control in pictorial and facial stimuli compared with verbal stimuli.

Further, both theory and research evidence indicate that idiosyncratic data consistent
with one’s internal schematic set is required to induce mood-congruent information
processing biases (Beck, 1967; Matthews & MacLeod, 2005). The self-referential
attention task (SRET; Rogers, Kuiper, & Kirker, 1977) with subsequent recall memory
analysis has proved to be useful here. Verbal stimuli can be easily incorporated into
this task. For these reasons the dissertation studies included verbal stimuli, which
were encoded using self-referential judgments.

2.2.3. Task Considerations and ERP Procedures

Given the complexities of the human brain neuro-electrical output measured by
the EEG, there are some fundamental guidelines for the design and analysis of ERP
experiments (Luck, 2005). First, studies should focus on specific, large, and easily
isolated ERPs. They should utilise established ERP paradigms (e.g., Old/New
paradigms for memory processing, or the Go/Nogo task for inhibitory processing).
Studies should also try to avoid behavioural confounds, such as the need to provide
motor responses to one type of stimuli but not to another (Luck, 2005). This is an
inherent limitation of the Go/Nogo task, which as described in Section 2.2.1. requires
a key press response following Go stimulus presentations and inhibition of such
responses following Nogo stimulus presentations. In such a case, an effort should be
made to counterbalance response hand within condition trials. In these cases, the
researcher should not assume that the motor planning and performance differences
cannot explain divergent ERP effects (Luck, 2005), especially in regards to the Nogo-
P3 potential (Smith et al., 2008). ERP amplitudes tend to be larger when signal-to-
noise ratios are lower (Luck, 2005). Thus, researchers need to be careful when
comparing averaged ERPs based on a different number of evoking trials. Equal-probable trials in each experimental condition may reduce the risk of Type II errors.

Event related potential analysis is known to be significantly influenced by the location of the reference electrode (see Michel et al., 2004). Changing the location of the reference electrode can change statistical outcomes and the magnitude of the extracted component’s peak amplitude. At times, this can cross the zero-baseline, thereby effectively converting the ERP polarity (Luck, 2005). Extraction procedures, such Principal component analysis (PCA) are reference independent procedures (Spencer, Dien, & Donchin, 2001). The PCA method identifies covariance across sets of time points over all waveforms and subjected to a rotation procedure. It is argued to result in better phrasing of orthogonal factor structures (Kayser, Bruder, Tenke, Stewart, & Quitkin, 2000; Pourtois et al., 2008). Thus this can be particularly beneficial in the extraction of multiple overlapping ERPs accompanying information processing, such as the P3a and P3b (see Figure 12; Kayser et al., 2000; Vuilleumier & Pourtois, 2007). However, correlated peaks are not enough to justify the existence of ERP components (Spencer et al., 2001). Rather than researchers a priori exploring all PCA extracted peaks, component identification should be combined with known or hypothesised cognitive or psychological ERP appearance at specified epochs given the experimental methodology (Spencer et al., 2001, Spencer, Dien, & Donchin, 1999).

The PCA is also thought to be sensitive to latency jitters and misallocation of variance (Beauducel & Debener, 2003; Chapman & McCrary, 1995; van Boxtel, 1998,). However, this is to a lesser degree than traditional baseline-to-peak ERP extraction procedures. The drawbacks of the PCA seem insignificant in contrast to the protocols benefits (Pourtois, Delplanque, Michel, & Vuilleumier, 2008). For these reasons researchers suggest that PCA extraction methods should be utilised in studies of
information processing in depression (see Bruder, Kayser, & Tenke, 2012). Thus, this protocol was employed in the current collection of ERP studies. Figure 12 presents an illustration of the PCA technique for ERP extraction.

Figure 12. Illustration of PCA in an emotional oddball task. (A). Grand averaged ERPs. A 300-600 ms analysis window is analysed based on planned comparisons. Corresponding topography of this late apex is shown on the horizontal scalp map. (B) Extracted components of the PCA analysis, showing two separate but overlapping factors most likely reflective of the P3a and P3b waveforms. Topographical distributions of these factors are provided. Image reprinted from Pourtosis et al. (2008).

Summary of the ERP technique. In summary, the ERP technique is a non-invasive method of measuring brain activity during cognitive processing. These transient electric potential shifts are time-locked to the stimulus onset and are believed
to represent neural activation associated with mental operations. The ERP provides extremely high time resolution as opposed to typical neuroimaging or behavioural procedures. This effectively increases the capacity of the ERP to elucidate subtle cognitive activity. Whenever possible, ERP investigations should apply the same physical stimuli and ERP recording and extraction methods across different psychological conditions to avoid confounding the data (Luck, 2005). For example, previous studies have found neutral and emotional stimuli appear to be processed differently due to the ERP arousal and valence effects. Thus, neutral stimuli should not be an experimental control or baseline criteria to which emotional processing be compared. For this reason, neutral stimuli were not employed in the current dissertation studies. Rather, the results of healthy control participants were employed as the comparative/baseline condition. Further, peak-to-ratio ERP amplitudes tend to be larger when signal-to-noise ratios are lower. Researchers need to be careful when comparing averaged ERPs based on a different number of evoking trials. Use of equal-probable trials in each experimental condition may reduce this risk (Luck, 2005), and hence were applied in the current dissertation ERP methodologies. Researchers also need to ensure that in tasks where the need for motor responses differs between conditions, response hands be counterbalanced and motor potentials be considered in component interpretations (Luck, 2005; Smith et al., 2008) and ideally be extracted with PCA protocols (Bruder et al., 2012). The dissertation studies also employed these two latter methodologies. The dissertation ERP studies also ensured to make use of similar stimuli, stimuli presentation rates, and ERP recording protocols in the same sample of female participants. This further allowed for greater generalisation of results across the dissertation studies. This improved the amalgamation of their findings in an attempt to validate the relationships outlined in
Joormann et al.’s (2007) cognitive model of depression. The research for the attention and memory mood-congruent biases argued in Beck (1967) and Bower’s (1981) models, and working memory inhibitory deficits implicated in contemporary depression models are outlined next.

2.3. Evidence for Cognitive Biases and Inhibitory Deficits in Depression

This dissertation evaluated the behavioural and ERP correlates of emotional information processing in individuals diagnosed with current and remitted MDD. Thus far, this overview chapter has outlined the definitions of MDD, its global impact, the key cognitive models of the disorder and has summarized the relevant ERP methodology for this research. The last section of the chapter aims to review the current evidence supportive of abnormalities in affective attention, memory, and inhibitory control in depression. From this, the aims and hypotheses of the dissertation’s four studies are constructed.

2.3.1. Mood-congruent attention and encoding in depression. It is important to understand the methodological selections of the first dissertation study in this section. Specifically, study 1 investigates encoding processes during self-descriptive judgments of positive and negative stimuli in a sample of MDD, remitted depressed, and healthy control participants.

The empirical evidence regarding attentional biases in depression is relatively rare and diverse, making it difficult to draw firm conclusions. Contrary to the predictions based on Beck (1967) and Bower’s (1981) cognitive models, early behavioural research into negative attention biases in depression typically produced null results (Bradley et al., 1997; Hill & Dutton, 1989; MacLeod, Mathews, & Tata, 1986; Mogg et al., 2000; Mogg, Bradley, Williams, & Mathews, 1993; Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 2000). This failure to observe attention biases in
depression led to Williams et al.’s (1997) argument that depression is not characterised by biased attention processes (see Section 2.1.). More recent behavioural studies have suggested that negative attention biases emerge only under conditions of long stimulus exposure (e.g., >1000ms) in both depressed (Gotlib, Krasnoperova, Yue, & Joormann, 2004; Joormann & Gotlib, 2007; Joormann, Talbot, & Gotlib, 2007; Karparova, Kersting, & Suslow, 2007) and dysphoric samples (Caseras, Garner, Bradley, & Mogg, 2007; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005; Koster, Leyman, De Raedt, & Crombez, 2006; Shane & Peterson, 2007).

Negative attention biases in depression and dysphoria have been more reliably observed during attentional processing, indexed by longer latency ERPs (e.g., P3, Slow Wave: Blackburn, Roxbourought, Muir, Glabus, & Blackwood, 1990; Deldin et al., 2001; Deldin, Naidu, Shestyuk, & Casas, 2009; Deveney & Deldin, 2004; Nandrino, Dodin, Martin, & Henniaux, 2004; Shestyuk & Deldin, 2010; Shestyuk et al., 2005), compared to early processing stages (e.g., P1, N1, P2: Deldin et al., 2001; Deldin, Keller, Gergen, & Miller, 2000; Dietrich et al., 2000; Serfaty et al., 2002; Shimizu, Saito, & Hoshiyama, 2006). These observations have led researchers to argue that depression is associated with biases in elaborative attention rather than orientating attention (Williams et al. 1988, 1997; Wisco, 2009). However, a meta-analysis of this literature found no moderating effect for stimulus duration on negative attention biases in depression (Phillips, Hine, & Thorsteinsson, 2010). Specifically, associations between depression and negative attention biases were observed in both early (≤ 400ms, n = 11, r = .19, p < .001) and late (≥ 500ms, n = 11, r = .19, p < .001) stage attention processing. It might be that self-referential encoding tasks are necessary to observe depressive attention biases.
In their meta-analysis (using pooled sample of 7032), Phillips et al. further found that studies employing self-descriptive encoding procedures produced the largest effect sizes for negative attention biases in depressed samples. This was observed at both early and late stages of attention processing. Previous literature reviews also suggest that self-referential encoding enhances power for studies of attention biases in depression (Matthews & MacLeod, 2004; Wisco et al., 2009). Specifically, these reviews report that depressed individuals show better memory for negative than positive words encoded self-referentially, or any words encoded in reference to others (e.g., Wisco, 2009). Non-depressed individuals show better memory for positive than negative words encoded self-referentially. Shestyuk and Deldin (2010) found ERP early attentional processing biases—as evidenced by greater left frontal-central P2 amplitude in their depressed participants. They used an equi-probable self-referential attention task of positive and negative personality adjectives. Correlational analysis found that greater positive free recall memory in the self-referential condition was associated with this P2 effect ($r = 0.50$). They found an enhanced P2 for their remitted depressed participant’s in response to negative compared to positive stimuli. However, the remitted participants exhibited no corresponding correlations with memory performance, perhaps reflecting active suppression of further processing of this information in this sample. For either group, no ERP effects were observed in the other-referential condition, despite the same stimuli words being employed. There are two explanations for the self-referential effect in this literature. First, some researchers argue that negative thoughts in depression are specifically self-focused, and thus, such cognitive negativity does not extend to their thinking about others (Derry & Kuiper, 1981; Kuiper & Derry, 1982). This is supported by previous work which show people to be motivated to recall...
memory relevant to their self, and to forget memory that are discrepant with or threatening to their sense of self (Conway, 2005). Alternatively, according to the level of processing model, self-descriptive encoding serves to increase the depth of processing which strengthens memory (Cermack & Craik, 1978; Craik & Lockhart, 1972; Craik & Tulving, 1975). Specifically, self-referential encoding promotes greater elaboration and organisation of material in memory (Klein & Loftus, 1988; Symons & Johnson, 1997). Greater elaboration promotes memory by allowing multiple routes for later recall (Craik, 1979; Klein & Loftus, 1988). Thus, the lack of observation of negative attention biases in the early depression literature may be due to use of experimental stimuli not personally salient to the research participants. The method employed in Study 1 of this dissertation ensures to avoid this limitation by using a self-referential attention task (see section 3.2).

Other inconsistencies exist in the ERP attention bias studies for depression. For instance, there are differences in interpretation of a well-studied ERP correlate of attention, the P3b. Ilardi et al. (2007) employed a classic oddball paradigm, in which frequently occurring (80%) neutral words were interposed with rare (20%) negative target words. The researchers found no P3b oddball effects for negative words in their never-dysphoric or previously dysphoric (remitted) control samples. However, their dysphoric sample showed larger P3b magnitude to negative oddball stimuli compared to neutral stimuli, and compared to the two control samples. Ilardi et al. argued that the P3b effect was indicative of a depressive negative attention bias, which appears to be a state-like, rather than trait-like, experience of dysphoria. This interpretation is consistent with the assumption that the P3 amplitude is a measure of attentional allocation (Kok, 2001), but inconsistent with context updating hypotheses (Donchin & Coles, 1988).
Kayser et al. (2000) measured ERPs during passive viewing of a hemifield display of pictures of diseased (negative) and healed (neutral) dermatological facial stimuli in their sample of un-medicated depressed and control participants. The researchers found control participants showed no early P3 (peak latency 330ms) differences to negative or neutral faces. Unlike, Ilardi et al. who employed a peak detection procedure, the researchers used PCA methodologies to obtain the P3a and P3b subcomponents. They found their clinically depressed participants did demonstrate enhanced early P3 to negative faces compared to the neutral faces, and compared to non-depressed participants. Similar to Ilardi et al., Kayser et al. also argued that the results suggest depressed individuals show biased attention (discriminative) to negative stimuli. However, in contrast to Ilardi et al., the depressed participants in Kayser et al.’s investigation did not reveal the expected later P3 enhancement for negative compared to neutral stimuli, but instead exhibited overall smaller size during this later epoch (460 ms) as compared to control participants. Control participants showed larger positive activation in the right parietal hemisphere during processing of negative stimuli. This replicated previous research demonstrating enhanced ERP positivity for affective stimuli in the normative population (Kayser et al., 1997). The observation that this P3b was largest over the right parietal hemisphere is consistent with the hypothesis that this area controls recognition and evaluation of affective information (Heller, 1990). The lack of observance of this effect in the depressed participants suggests impaired activation of this region during emotional processing in the disorder (Heller, 1990, 1993).

Blackburn et al. (1990) employed an equi-probable presentation of positive, negative, and neutral word stimuli. Participants were required to read each word passively. Statistical analyses were conducted on ERP difference-waves, with neutral
stimuli acting as the comparative baseline (negative minus neutral and positive minus neutral). Blackburn et al. found their dysphoric participants exhibited smaller P3 in response to negative than positive word stimuli compared to both the remitted-dysphoric and control participants. Yang et al. (2011) employed an oddball task with positive and negative stimuli acting as emotional targets, and frequent neutral stimuli acting as the standards. Participants had to identify the target cue and quickly respond by pressing a designated key. The MDD exhibited significantly reduced parietal P3s to negative as compared to positive target stimuli. This ERP effect was not found for the control participants. The context-updating view argues that P300 amplitude corresponds to a discrepancy between received and expected data (Donchin & Coles, 1988). Depressed patients talk about and ruminate on negative events and therefore anticipate negative information in their environment. In line with context-updating hypotheses of the P3b, both Blackburn et al. and Yang et al. argued reduced P3s for negative information are anticipated in depressed samples. Specifically, depressed individual’s increased expectation of negative materials given their negative cognitive schemata would not result in a context mismatch, and thus would not increase P3b amplitudes (Beck, 1967; Beck et al., 1979). However, positive stimuli would result in a context mismatch, resulting in larger P3b in response to presentation of this information.

Nandrino et al. (2004) investigated the role of depression history on emotional attention biases, comparing ERP results of first-episode and recurrent depressed patients to a non-depressed control group. All participants completed an emotional oddball task twice: Once before psychotherapy (therapeutic approach was unspecified) and again after clinical improvement (28 days). The researchers found recurrent depressed participants exhibited larger P3s at frontal and parietal sites to negative
stimuli compared to both the first-episode depressed and non-depressed patients. In line with Ilardi et al. (2007), but in contrast to Blackburn et al. and Yang et al., the researchers’ argued that the observed increased P3 to the negative stimuli represented a negative attention bias in the recurrent depressed group. They reported that these results are consistent with the hypothesis that repeated depressives experience strengthen depressive thinking, enhancing these individuals’ reactivity to negative stimuli (Beck, 1967; Teasdale, 1988). First-episode depressed participants showed significantly smaller P3s to positive words compared to both non-depressed and recurrent depressed participants. In a similar line of reasoning, they argued this to represent attentional avoidance to positive material in first-episode depression. This is consistent with Clark and Watson’s (1991) tripartite model which argues that depression is characterised by symptoms of anhedonia and diminished positive affect. After clinical improvement, previously observed negative attention bias in the recurrent depressed group disappeared. This observation is consistent with predictions of the cognitive models of depression—in which depressive schemas and associated negative biases become dormant (cf. Beck, 1967; 2008; Joormann et al., 2007). However, despite similar clinical improvement, first-episode depressed patients continued to demonstrate attentional avoidance—as evidenced by decreased P3 amplitude—to positive stimuli. Post-treatment, first-episode depressed patients also showed attentional avoidance of negative stimuli—also evidenced by decreased P3 amplitude—that was not evident prior to treatment. This decreased neural reactivity to negative stimuli in both the first-episode and the recurrent patient groups appears to reflect the beneficial effect of psychotherapy in decreasing negative cognitive biases in depression. The researchers argued that the absence of improved attention to positive stimuli in these first-episode patients most likely reflected treatment effects.
Specifically, they argued that most traditional cognitive interventions focus on decreasing activation of negative cognitions rather than fostering connection and processing of positive material. Overall, this ERP study provides valuable insights into the impact of depression disorder history on emotional information processing in depression.

**Summary and relevance to Study 1’s aims and methodology.** The majority of ERP evidence supports the existence of negative attention bias in depression. However, there is controversy over the ERP morphology of this attentional bias, especially for context updating as indexed by the P3 component. Conflicting P3 results may represent differences in sample characteristics, ERP data-analysis techniques, experimental stimulus (words versus faces), and methodology conditions (oddball tasks, passive viewing tasks, use of neutral stimuli as the experimental baseline). Further, these mixed results may be in part a result of use of mixed gender samples. As outlined in Section 2.2.2., males and females are known to process emotional information differently (see Nolen-Hoksema & Hilt, 2009 for a review). Females displayed greater ERP amplitudes to unpleasant and arousing stimuli (Lithari et al., 2010). Thus, the use of mixed gender samples without statistical distinction of their specific effects may result in inaccurate and misleading ERP conclusions regarding emotional information processing. Further critical analysis of these divergent P3b results is provided in the background to Study 1 (see Section 3.1.). The current dissertation studies employed a heterogeneous sample of female participants. A sample of matched control participants were employed as the baseline condition to examine hypothesised negative attention biases rather than using misleading internally invalid neutral as the stimuli baseline. The above analysis also suggested that negative attentional biases in depression are more reliably observed under conditions of self-
referential processing (Shestyuk & Deldin, 2010). Specifically, it might be that depressed individuals experience an inability to disengage from personally salient negative information (Gotlib & Joormann, 2008; Joormann et al., 2007; Wisco, 2009). Thus, attention biases in depression were examined in Study 1 using a self-referential encoding protocol. It is hypothesised this facilitated elaborative processing of negative information in depression may result in deeper encoding of this information into long-term memory (Williams et al., 1997). Explicit memory processes are examined in Study 2. The current empirical evidence of episodic memory biases in depression is outlined next.

2.3.2. Episodic mood-congruent memory biases in depression. It is important to understand the methodological selections of the second dissertation study, in which episodic memory processes for emotional stimuli are examined this section. Behavioural investigations consistently show that non-depressed individuals recall significantly more positive than negative stimuli. Depressed samples recall more negative than positive stimuli (on average 10% more; see Matt, Vazquez, & Campbell, 1992 for a review). Memory biases appear dependent on the particular subcomponent of memory examined. They are more reliably found in explicit free-recall or working memory tasks (Deldin et al., 2001; Shestyuk et al., 2005), compared to implicit (Ellwart, Rinck, & Becker, 2003; Watkins, Martin, & Stern, 2000) or recognition memory tasks (Deldin et al., 2000; Deldin et al., 2001; Dietrich et al., 2000). In addition, emotional memory shows a linear relationship with depressive severity. Memory biases for positive materials are more typically observed in non-depressed samples (Deldin et al., 2001; Deldin et al., 2009), while dysphoric samples show no memory biases (Jermann, van der Linden, & D'Argembeau, 2008; Ridout, Noreen, & Johal, 2009), and clinically depressed samples typically show memory
biases for negative material (Gilboa-Schnechtman, Erhard-Weiss, & Jeczemien, 2002; Ridout, Astell, Reid, Glen, & O’Carroll, 2003). Negative memory biases in depression have been associated with impaired problem solving, increased negative expectation, and delayed recovery from depressive episodes (Peeters, Wessel, Merckelbach, & Boon-Vermeeren, 2002; Raes et al., 2005). Unlike attention (cf. Nandrino et al., 2004, Section 2.3.1.), memory biases appear independent of depressive disorder history. That is, similar negative memory effects have been observed in first-episode and recurrent depressed samples (Nandrino, Pezard, Poste, Revillere, & Beaune, 2002; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001). Remitted depressed patients exhibit similar emotional memory functioning as never-depressed samples (Nandrino et al., 2002; Shestyuk & Deldin, 2010). Although there is strong support for negative memory biases in depression in the behavioural literature, the ERP evidence for these effects is more ambiguous.

Electrophysiological investigations of explicit memory performance typically use the Old/New paradigm (see Section 2.2.1.). Dietrich et al. (2000) used the Old/New paradigm to test recognition memory for positive, negative, and neutral words in depressed and non-depressed participants. Behavioural results found that both non-depressed and depressed participants exhibited enhanced memory for negative words and reduced memory for neutral words. However, non-depressed participants showed larger LPC old/new effects compared to depressed participants. Control participants also showed larger LPC old/new effects for emotional (both positive and negative) stimuli compared to neutral stimuli. The authors argued that this reflected “intact recognition processes” in their non-depressed sample (Dietrich et al., 2000, p.26). The LPC old/new effect is hypothesised to reflect neural integration of memory processing of subsystems of the medial temporal lobe structure,
particularly in the Hippocampus and Amygdala projections to the prefrontal and parietal cortex (Gordeev, 2007; Paller & Kutas, 1992; Rugg, Fletcher, Frith, Frackowiak, & Dolan, 1996; Rugg, Roberts, Potter, Pickels, & Nagy, 1991). This integration processes is an essential task of the central executive system of working memory (see Section 2.1.; Baddeley, 2000; Baddeley & Hitch, 1974). Thus, the overall reduction of the LPC old/new effect in the depressed participants might be the result of global deficits in working memory functioning in the disorder.

In another study, Deldin et al. (2002, as cited in Deldin et al., 2003) presented participants with lists of self-referential stimuli that they later recalled. Non-depressed participants exhibited greater recall and increased slow wave (SW) amplitude for positive relative to negative stimuli. They also showed greater SWs for positive stimuli relative to the depressed participants. This is consistent with other ERP studies that have found non-depressed participants to show greater SW amplitudes in response to positive relative to negative stimuli (Deldin et al., 2001, 2009; Deveney & Deldin, 2004) and demonstrate greater sustained brain activation in response to positive stimuli than depressed participants (Deldin et al., 2001). In contrast, Deldin et al.’s (2002) depressed participants exhibited increased SW amplitude for processing of negative relative to positive stimuli during self-referential encoding. Therefore, depression may be associated with not only preferential processing biases for negative stimuli, but also with the absence of processing biases toward positive stimuli. This latter positive bias may serve a protective function in non-depressed samples (Alloy & Abramson, 1988). This study also demonstrated that, similar to the attention literature, memory biases in depression also seem to be dependent on stimulus salience. That is, experiments that employ personally salient stimuli (Deldin et al., 2009; Deldin et al., 2003; Moritz, Voigt, Arzola, & Otte, 2008) or self-referential encoding procedures
(Shestyuk & Deldin, 2010) appear more successful in eliciting negative memory biases.

*Summary and relevance to Study 2’s aims and methodology.* Behavioural investigations consistently show that non-depressed individuals recall significantly more positive than negative stimuli while depressed samples recall more negative than positive stimuli (Matt et al., 1992). However, it is difficult to separate in the literature whether this effect is the result of enhanced memory operations for negative stimuli or due to preferential encoding of negative information. Electrophysiological investigations have the benefit of locating which stage(s) of information processing result in this effect. This can specifically be achieved using the Old/New paradigm, which is known to evoke the FN400 and LPC old/new effects. Depressed individuals’ seem to show reduced old/new effects, which have been interpreted as indicative of working memory deficits in the disorder (Dietrich et al., 2000). This premise underpins the contemporary cognitive models of depression as discussed earlier in this chapter (cf. Beck, 2008; Joormann et al., 2007; see Section 2.1.). For this reason, Study 2 measured ERPs whilst participants completed the Old/New paradigm to investigate episodic memory biases. The research support for the predictions and methodological considerations for the working memory inhibitory control problems in depression—as analysed in Study 3—is discussed next.

2.3.3. **Deficit working memory inhibitory control in depression.** This section reviews the literature on deficits in cognitive inhibitory control in depression. Cognitive control refers to the processes by which individual cognitive functions are coordinated in the service of higher-level goals. For example, this might invoke the ability to inhibit the processing of irrelevant information in working memory. As outlined in section 2.1., cognitive inhibitory control involves two distinct processes.
(1) **Resistance to distractor interference:** the ability to keep irrelevant negative material from entering working memory.  (2) **Resistance to proactive interference:** the ability to remove negative material from working memory. These two inhibitory processes are investigated in Study 3 of this dissertation. The majority of the research in this area is guided by the theoretical predictions outlined in the contemporary models of depression (see Section 2.1.), specifically those of Joormann et al.’s (2007) cognitive model.

Recall from Section 2.1., Joormann et al. (2007) outlined a model of depression that argued that impaired working memory inhibitory processing results in lack of control over bottom-up negative memory and attention biases and ruminative response styles (Hertel, 1997; Joormann, 2004; Joormann, 2005; Joormann et al., 2008; Linville, 1996; Nolen-Hoeksema et al., 2008). They implicate deficits in both resistance to distractor interference and resistance to proactive interference in their model (see Figure 13). Joormann et al.’s model does not specify that current depressed mood state is required to observe attention, memory, and emotional regulation biases. Rather, it assumes that these biases should be pronounced as long as the working memory inhibitory control deficiency persists. The following sections will examine the behavioural, ERP, and neuroimaging results for resistance to distractor interference and resistance to proactive interference inhibitory control in depression, respectively. This will lead into the rationale for the aims and methodological sections of Study 3 of this dissertation.
Figure 13. Joormann et al.’s (2007) integrated cognitive model of depression. Image adapted from Joormann (2010).

Researchers have developed a number of alternative experimental tasks to measure cognitive inhibition. Details of the Go/Nogo task and the Nogo-N2 and Nogo-P3 are outlined in Section 2.2.2. Other key working memory tasks and their related ERP components are outlined in Table 2.
Table 2

*Tasks Used to Assess Inhibitory Processing & Associated ERP Components*

<table>
<thead>
<tr>
<th>Task</th>
<th>ERPs</th>
<th>Key References</th>
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<tr>
<td>Stroop task</td>
<td>ERN</td>
<td>(Holmes &amp; Pizzagalli, 2008; McNeely et al., 2008; Vanderhasselt &amp; DeTeadt, 2009; West, Choi, &amp; Travers, 2010)</td>
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<td></td>
<td>N450/MFN</td>
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<td></td>
<td>SW</td>
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<tr>
<td>n-back/ SI-S2 tasks</td>
<td>SW</td>
<td>(Deldin et al., 2001; Deveney &amp; Deldin, 2004; Shestyuk et al., 2005)</td>
</tr>
<tr>
<td>Negative Affective Priming</td>
<td>N200</td>
<td>(Daurignac, Houdé, &amp; Jouvent, 2006; Gibbons, 2006; Yao et al., 2010)</td>
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<tr>
<td>(NAP) task</td>
<td>P200</td>
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<td></td>
<td>LPC</td>
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<tr>
<td>Directed Forgetting/Think-No-Think task</td>
<td>LPP</td>
<td>(Hauswald, Schulz, Irodanov, &amp; Kissler, 2011; Paz-Caballero, 2004; Menor, &amp; Jiménez 2004)</td>
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**2.3.3.1. Resistance to distractor interference.** Resistance to distractor interference is referred to as just “distractor interference” throughout the remainder of this thesis. A number of behavioural studies have demonstrated deficits in inhibiting
distractor interference stimuli in depressed individuals (Benoit et al., 1992; Harvey, Watkins, Mansell, & Shafran, 2004; Kaiser et al., 2003; Lemelin et al., 1996; Linville, 1996; Ruchsow et al., 2008; Zhang, Zhao, & Xu, 2007). This deficit appears to be linked with a specific inability to inhibit irrelevant negative material from working memory (Joormann, 2004; Goeleven et al., 2006). Prospective experiments have found these distractor interference deficits for negative information to be predictive of future depressive symptoms and rumination (Zetche & Joormann, 2011). There is some evidence of distractor interference inhibitory impairment in remitted depressed samples, with this relationship fully mediated by rumination (Demeyer, De Lissnyder, Koster, & De Readt, 2012).

The ERP evidence for distractor interference inhibition deficits in current and remitted depression shows mixed results. Using a Go/Nogo paradigm of neutral auditory tones, Kaiser et al. (2003) found depressed patients to make more errors of commission for Nogo stimuli in comparison to the non-depressed participants. They also exhibited an early frontal-temporal negatively in the N2 time window, whereas the control participants exhibited a polarity-inverted N2. The researchers suggested that this ERP profile was indicative of deficits in distractor interference inhibitory processing in the depressed participants. The polarity-inverted N2 is not compatible with previous reported morphology of the Nogo-N2, which tends to show negative amplitude (Falkenstein et al., 1999). It is possible that the differing results are due to EEG referencing protocols. Previous investigations finding the typical negative Nogo-N2 have used either linked mastoid or earlobe reference points (e.g., Zhang et al., 2007). However, Kaister et al. (2003) used an average reference procedure in their study, which might have resulted in their polarity inverted Nogo-N2. No group differences were found for the P3 time-window or for the Go stimuli. Kaiser et al.’s
Go/Nogo ERP results and spherical spline map at 255ms (mean of N2 time window) is shown in Figure 14.

![Go/Nogo ERP results and spherical spline map at 255ms (mean of N2 time window) from Kaiser et al. (2003).](image)

**Figure 14.** Go/Nogo ERP results and spherical spline map at 255ms (mean of N2 time window) from Kaiser et al. (2003).

In their Go/Nogo task, Zhang et al. (2007) also found ERP evidence of working memory inhibitory control deficits in their sample of older depressed individuals (age >60 years). This was indexed by reduced Nogo-N2s in the depressed participants, which correlated with depressive symptoms ($r = 0.53$; Zhang et al., 2007). In a sample of partially remitted depressed patients, Ruchsow et al. (2008) found no group profile differences in the Nogo-N2 component. However, the researchers found remitted depressed patients to exhibit reductions in the frontal Nogo-P3. It is important to note that the participants in Rushcow et al.’s study were only in partial remission, thus, it is difficult to generalise their data to either current or remitted depressed samples.

Research suggests that depressive subtype may result in different cognitive inhibitory control deficits (Quin, Harris, & Kemp, 2012). Specifically, patients with melancholic depression have been observed to exhibit greater number of commission...
errors during the Go/Nogo task in comparison to non-melancholic or control participants (Quin et al., 2012). However, depressive subtype does not appear to result in different ERP data when correct responses are examined (Quin et al., 2012).

Vanderhasselt and De Readt (2009) provided ERP evidence that distractor interference inhibitory control deficits are moderated by depressive history. The researchers used a word-colour Stroop task. As outlined in Table 2, the Stroop task involves the presentation of a series of colour names in different coloured ink. Participants are required to report ink colour of each word. The Stroop effect is that word reading is an automatic ability that interferes with colour naming when a colour word is printed in a different colour (i.e., word red printed in green). Vanderhasselt and De Readt (2009) observed larger N450d ERPs were observed for incongruent compared to congruent trials in the sample of never depressed participants. The authors argued that this represented normal working memory inhibitory functioning. However, this N450 effect was not observed in their remitted depressed participants. This appeared reflective of remitted depressed participant’s cognitive deficits inhibiting negative information from entering working memory. This ERP finding correlated with number of participant’s previous depressive episodes ($r = .51$), but not with age of depressive onset or duration of remission. These results suggest that deficits in the ability to keep irrelevant material from entering working memory are enhanced with each successive depressive episode. Interestingly, the researchers found no behavioural evidence for this ERP effect. This might reflect the unique advantage of ERP to detect rapid cognitive control processes that are not always evident in overt behaviour. Overall, these investigations provide support for the hypothesis that depressed individuals show distractor interference deficits for non-emotional stimuli.
In the context of inhibition of emotional information—the interest of the current collection of dissertation studies—Goeleven et al. (2006) used a NAP task to investigate resistance to distractor interference inhibitory control in a sample of non-depressed, depressed, and remitted-depressed participants. As outlined in Table 2, in the NAP task participants are required to respond to a target cue while simultaneously inhibiting responding to a task-irrelevant distracter. The negative priming effect corresponds to the delayed response/brain activation when the distracter becomes the target cue on the successive trial. The magnitude of this effect indexes strength of distractor interference performance. The study found that non-depressed and remitted depressed participants were slower to recognize the valence of positive and negative facial expressions following similarly-valanced distracters, indicating efficient inhibitory processes. Whereas, their depressed participants demonstrated significantly faster responses to negative faces presented after negative distracters, indicating impaired inhibition functioning. As presented in Figure 15, this effect was not found for positive stimuli. This suggests a specific impairment for negative information in depression. Joormann (2004) found a similar result in their dysphoric participants and remitted dysphoric participants using a NAP task with emotional word stimuli.
Figure 15. Mean negative affective priming for negative and positive trails in depressed, formerly depressed and never-depressed participants. Positive values indicate effective distractor interference inhibitory functioning. Image adapted from Goeleven et al. (2006). All rights reserved.

The cross-sectional nature of the above investigations makes it difficult to conclude that the reported inhibition deficits are a symptom of experiencing depression or if they actively contribute to the perpetuation of the disorder. Addressing this, Zetsche and Joormann (2011) used a prospective design to assess the association between inhibitory functioning, depressive symptoms, and rumination over a six month interval (time 1: \( n =111 \); time 2: \( n =40 \ [36\%] \)). They found that depressive symptoms at time 1 were associated with distractor interference inhibitory deficits for negative information in a NAP task. Individual differences in this inhibitory performance predicted depressive symptoms and rumination at time 2, providing supportive evidence to the theory that depression is preceded and possibly maintained by poor inhibitory control over negative material. A few limitations of this study need to be outlined. First, Zetsche and Joormann’s results are based on a non-clinical sample of university students, with low overall BDI-II scores. This limits the
applicability of these results to severely depressed populations. Further, given the high attrition rate of the sample (64%), the time 2 results are to be interpreted with caution. It may be argued that participants who completed the follow-up analysis were qualitatively dissimilar to those who did not. This suggests poor internal validity of these results. Arguably, time 1 BDI-II and RRS scores and performance on the inhibition experimental tasks did not differ between participants who took part in the time 2 follow-up data with those who did not. Although it may be argued that participants did not differ on the measured variables, they could have differed on unmeasured variables, such as motivation. This unmeasured attribute may interact with depression expression and/or individual differences in inhibitory control. Future research should aim to replicate Zetche and Joormann’s (2011) findings in a large clinical sample.

As implicated in Beck’s (2008) neurobiological cognitive model of depression, neuroimaging studies have found a link between working memory inhibitory dysfunction and depression. Specifically, previous fMRI and positron emission tomography (PET) work has found that non-depressed people engage greater rostral ACC activation to inhibit irrelevant positive information. It further finds depressed individuals to engage greater rostral ACC activation to inhibit irrelevant negative information (Elliott, Rubinsztein, Shakian, & Dolan, 2000; Eugene, Joormann, Cooney, Atlas, & Gotlib, 2010; Mitterschiffthaler et al., 2008). These neuroimaging results support the hypothesis that depression is associated with not only deficits of distractor interference inhibitory control over negative information. It also is associated with an apparent lack of preferential processing of positive stimuli, which appears characteristic of healthy individuals (protective bias).
In their fMRI investigation of remitted depression, Kerestes et al. (2012) found remitted depression to be related to greater neural activity in the DLPFC during completion of an n-back task with negative emotional distracters. In the n-back task (as outlined in Table 2) participants are to monitor the identity or location of series of a presented stimuli (S1). After a brief interval they are presented with a probe stimulus (S2) and asked to indicate if it was associated with the S1 presented n trials previously. The DLPFC appears implicated in directing attention away from task-irrelevant emotional distracters (Dolcos & McCarthy, 2006; Kerestes et al., 2012), a key task of emotional regulation (Phillips, Ladouceur, & Drevets, 2008). These abnormalities were found predominately in the left hemisphere and occurred in the absence of any distinct group differences in performance. Thus, it is possible that remitted depressed participants need to call on greater neuro-inhibitory resources, specifically those over the left hemisphere, for the purposes of stopping a prepotent response to negative stimuli with the same level of skill as the control participants. These results are consistent with previous studies that have shown deficits in emotional processing and executive control persist into depression remission (Bhagwager & Cowen, 2008; Clark, Sarna, & Goodwin, 2005; Neumeister et al., 2006; Joormann & Gotlib, 2007; Kerestes et al., 2012; Preiss et al., 2009).

Behavioural research has identified the presence of emotional information processing differences between non-depressed and depressed samples. Neuroimaging methods have allowed the identification of the precise locations of neural mechanisms that underlie these processes. Nevertheless, these two techniques are relatively insensitive to temporal fluctuations associated with automatic cognitions involved in information processing. As argued in Section 2.2., due to its high temporal resolution, ERPs can provide key indicators for the specific stage of processing that may lead to
this disturbance of emotional information processing in depression. The ERP directly reflects cognitive processes, with neural activity recorded almost simultaneously. The detailed temporal information of this ERP can be particularly advantageous in helping to determine whether working memory inhibitory control deficits for negative information in depression is the result of early (Nogo-N2) or later (Nogo-P3) cognitive inhibition abnormalities, or both.

Electrophysiological studies suggest that the negative priming effect the behavioural and neuroimaging literature is the result of abnormal early frontal cognitive inhibition processes. This is indexed by deviant early frontal-central ERPs, such as reduced frontal amplitude of P2 (Gibbons, 2006; Yao et al., 2010), or increased frontal negativity of N2 (Daurignac et al., 2006). For instance, Yao et al. found non-depressed participants exhibited larger P2 to negative distractor stimuli using the NAP task. Given the relationship between attention allocation and P2 amplitude (see Table 1, Carretié, Mercado, Tapia, & Hinojosa, 2001a; Carretié, Martin-Loeches, Hinojosa, & Mercado, 2001b), the enhanced P2 in their results may reflect increased cognitive effort to overcome interference from the distractor stimuli. Depressed participants showed abnormally reduced P2 amplitude at central-parietal electrodes for negative but not for positive distractor stimuli. Therefore, the researchers argued that this reflected diminished cognitive control over negative distractor interference stimuli for the depressed individuals.

It is important to note however, that inhibition effects of NAP and n-back tasks cannot be readily equated to inhibition effects observed in the Go/Nogo task. It is likely that the tasks assess different cognitive inhibition processes. For instance, the NAP task might allow for greater measurement of information interference/conflict processes. The n-back task might allow for measurement of working memory
capacity and manipulation. The Go/Nogo task, on the other hand, might allow for
greater measurement of inhibition of prepotent response tendencies. Despite this, it is
apparent that distractor interference inhibitory control deficits are associated with
depressive symptoms and rumination.

Summary for distractor interference inhibitory control deficits. Overall, the
above findings suggest a specific deficit in depressed individuals in inhibiting
irrelevant negative items from entering working memory. Given the hypothesised link
between rumination and negative memory intrusions, Joormann et al. (2007) also
argue that depression is also associated with deficits in removing irrelevant negative
material from working memory. That is, resistance to proactive interference control.
Supporting evidence for these predictions is outlined next.

2.3.3.2. Resistance to proactive interference. Resistance to proactive
interference is referred to as just “proactive interference” throughout the remainder of
this thesis. Experimental studies on proactive interference inhibitory control
typically use paradigms that require participants to encode stimuli that they are then
required to remove from working memory. Two promising tasks are the directed-
forgetting task (Hauswald et al., 2011; Paz-Caballero et al., 2004) and the Sternberg
working memory task (Oberauer, 2001, 2005a, 2005b). In the directed-forgetting task,
participants are first required to learn a list of words. They are then instructed to
remember (think) of some of these words, and to forget others (no-think).
Unexpected to participants, memory is assessed for all items, regardless of the
previous memory instructions. Efficient proactive inhibitory control is indexed by
significantly more ‘to-be-remembered’ items recalled than ‘to-be-forgotten’ material.
A proactive interference deficit would thus correspond to more ‘to-be-forgotten’
material recalled than would be anticipated (e.g., more than the ‘to-be-remembered’
items, or more than shown in the control sample). Using a directed-forgetting task, Power, Dalgleish, Claudio, Tata, and Kentish (2000) found that non-depressed students (Experiment 2) recalled more positive to-be-forgotten items compared to the dysphoric students, and compared to negative to-be remembered-items items. Dysphoric students (Experiment 2) recalled an approximately equal number of to-be-forgotten positive and negative items. Clinically depressed patients (Experiment 3), on the other hand, exhibited enhanced memory for negative to-be-forgotten stimuli relative to positive stimuli and to non-depressed participants. These results suggest that, similar to memory biases (see Section 2.3.2.), proactive interference deficits for emotional material appears to show a linear relationship with depressive symptoms. Proactive inhibition deficits for positive relative to negative stimuli are found in non-depressed samples. No proactive inhibitory deficits for either positive or negative stimuli are observed in dysphoric samples. While, greater proactive inhibition deficits for negative stimuli are found in clinically depressed samples. Consistent with this hypothesis, Hertel and Gerstle (2003) found that impaired ability to inhibit to-be-forgotten negative words during the directed-forgetting task was significantly correlated with depressive symptoms ($r = .36, d = .77$; Hertel & Gerstle, 2003).

Joormann and Gotlib (2008) utilised a modified Sternberg working memory task (Oberauer, 2001, 2005a, 2005b) to assess resistance to proactive interference for emotional information in depression. Participants learnt two lists of words. They were then instructed to remember only one of the word lists, which required participants to remove the irrelevant words from working memory. A single word probe was subsequently presented to participants, who were required to indicate if the word was presented in the previous relevant memory set by pressing ‘yes’ or ‘no’ on the computer keyboard. Probes from the irrelevant list must be rejected, as must new
It was suggested that differences in reaction times to an intrusion probe (word from irrelevant list) compared to a new probe (an entirely new word) indexes the strength of proactive interference inhibitory control processes (Oberauer, 2005a, 2005b; Ochsner & Gross, 2005). Joormann and Gotlib (2008) found that control participants—in both natural and induced sad mood conditions—had faster responses to negative proactive interference stimuli compared to the depressed group. This effect was not found for the positive stimuli. Figure 16 presents the mean intrusion effects observed in Joormann and Gotlib (2008) for their three participant groups.

![Figure 16. Mean intrusion effects (response time to intrusion probes minus new probes) for MDD, control (CTL), and sad mood-induced (CTL-SAD) participants for positive and negative stimuli (Joormann & Gotlib, 2008).](image)

The control or mood-induced groups did not differ from each other. These findings suggest that clinical depression—not just transient sad mood—is associated with specific difficulties in removing irrelevant negative information from working memory. Hierarchical regression analysis found that depressive symptoms (measured by the BDI-II; $\beta = .71$) and intrusion effects for negative material (step 2; $\beta = .34$)
together predicted 61% of the variance in individual rumination score (measured by the RRS). These associations are consistent with Joormann et al.’s (2007) impaired cognitive control model of depression.

Demeyer et al. (2012) found that deficits in proactive interference inhibitory control for negative information were predictive of one-year follow-up depression severity (BDI-II scores). Mediation analysis found that this relationship was fully mediated by rumination (RRS scores). Berman et al. (2011)’s fMRI study suggested metabolic evidence of weak frontal activity during working memory updating of negative information in depression. Specifically, using a directed-forgetting procedure, Berman et al. found their depressed participants exhibited greater errors in expelling negative, but not positive words from their working memory. This effect was correlated with greater spatial variability of activation on the left inferior frontal gyrus in the depressed sample: a region critical for inhibiting irrelevant information (Swick, Ashley, & Turken, 2008). Berman et al. also found this effect to be associated with greater rumination scores. No performance deficits were observed for the healthy control participants. The controls showed less activation of the left inferior frontal gyrus during successful proactive inhibitory control processing. The results suggest that individuals with depression are associated with poor recruitment of the prefrontal regions to inhibit negative proactive interference stimuli. This cognitive control deficit is associated with greater rumination. This work supports Joormann et al.’s (2007) theory that impaired inhibitory control for negative information is the catalyst underlying depressive rumination, which serves to perpetuate depressive experience.

Joormann et al.’s (2007) impaired inhibitory control model of depression does not specify that current depressed mood state is required to observe attention, memory, and emotional regulation biases. Rather, it assumes that these biases were pronounced
as long as the cognitive control deficiency persists. There is some evidence that reduced ability to ignore or inhibit goal-irrelevant negative information persists in remitted depression (Bhagwagar & Cowen, 2008; Joormann & Gotlib, 2007; Vanderhasselt et al., 2012). For instance, Vanderhasselt et al. (2012) used a cued emotional conflict task. Each trial first presented one of three cue words: (1) “Actual”, which instructed participants to press a key that corresponded to the emotional expression of the upcoming target face (e.g., press the happy key when a happy face is presented). (2) “Opposite”, which indicated for participants to press a key that corresponded the opposite emotional expression of the upcoming target face (e.g., press the happy key when a sad face is presented). Or (3) “Press”, which promoted participants to press a separate key when a face appeared (control condition). The researchers found their remitted depressed participants exhibited slower reaction times to cognitive inhibition (during the opposite condition) for sad facial expression compared to positive facial expressions using a cued emotional conflict task. This effect was complemented by contrasting ERP data with remitted participants exhibiting reduced N450 difference scores when asked to disengage from sad facial stimuli (opposite-sad condition). This effect was not evident for the happy stimuli (opposite-happy condition). See Figure 17 for a graph of these ERPs for the control and remitted depressed participants.
Figure 17. Grand average waveforms at electrode Cz for (A) control and (B) Remitted MDD (rMDD) participants during completion of an emotional conflict task (Vanderhasselt et al., 2012).

Enhanced N450 amplitudes in the context of the cued emotional conflict task has been interpreted to reflect the appropriate recruitment of cognitive control processing to overcome interference from conflicting mental representations (Vanderhasselt et al., 2013; West & Alain, 2000). Source localisation analyses have identified regions within the ACC as the potential neurogenerators of this N450 effect (Miniussi, Ruzzoli, & Walsh, 2010). These cognitive control deficits emerged in the absence of a mood priming manipulation. This supports the notion that individuals in complete remission from depression have relatively poor cognitive control in response to negative information. This cognitive deficit likely increases their risk for future depressive episodes. The finding that this effect was not observed during the N2
window suggests that remitted depression is not characterized by cognitive deficits during early stages of cognitive inhibition. To date, only one experiment has examined both resistance to distractor interference and resistance to proactive interference inhibitory processes together in the same task (Joormann, Nee, Berman, Jonides, & Gotlib, 2010). Hence, it requires further investigation (as achieved in Study 3 of this dissertation). In Joormann et al.’s (2010) study, participants were required to memorize a set of stimuli while ignoring concurrently presented distractor stimuli. They were then told to remember a subset of the previously memorized materials (probe stimuli), and forget the others, which were the suppress stimuli.

Distractor interference inhibition was indexed by longer response latencies on a subsequent recognition memory task of the distractor stimuli (never encoded into working memory/ participants were told to not attend to this stimuli) compared to new items. Note. New items were not presented in the previous task; they acted as the control condition. Proactive interference inhibition was indexed as longer response latencies on a subsequent recognition memory task of the suppress stimuli (stimuli that participants were first required to encode into working memory then later told to suppress or inhibit from working memory) compared to new items.

Non-depressed and depressed participants showed no deficits in the inhibition of negative distractor interference material. However, compared to non-depressed participants, depressed participants exhibited difficulties keeping proactive interference negative information from intruding on, or remaining active in, working memory (effect size: \( d = 1.17 \)). This is suggestive of a specific deficit in proactive interference inhibitory control in the depressed group. No group differences were observed for positive distractor interference or proactive interference information. This proactive interference deficit for negative material significantly correlated with the
level of depressive symptoms (measured by BDI-II; $r = .50$) and with recurrent negative cognitions (measured by RRS; $r = .41$).

**Associations of inhibitory control deficits with emotional regulation deficits.**

Previous work finds that deficits in disengaging from proactive interference negative material might explain impaired emotional regulation in depression. Consistent with Joormann et al.'s model (2007), cognitive inhibitory deficits in depression also appear to reduce the depressed individual’s ability to evoke mood-incongruent (positive) memories as an emotional regulation technique (Rusting & DeHart, 2000). Rather, depressed individuals are more likely to respond with maladaptive coping strategies, such as rumination, suppression, or avoidance to improve their negative mood (see Joormann & Gotlib, 2010 for a review). For instance, Joormann & Gotlib (2010) found that reduced inhibition of negative material was associated with greater self-reported habitual rumination in a group of depressed participants. In addition, reduced inhibition of negative information was related to less frequent use of reappraisal and more frequent use of expressive suppression across the entire sample of depressed, formerly depressed, and never-depressed participants.

**Summary for proactive interference inhibitory control deficits.** In summary, depressed individuals have difficulties removing irrelevant negative information in working memory. This proactive interference cognitive control deficit is associated with poor emotional regulation, resulting in sustained negative mood (Joormann et al., 2007; Gotlib & Joormann, 2010). Joormann et al.’s (2007) model suggests that amending working memory inhibitory control deficits in depression would disengage the engine that drives the maladaptive recycling of negative thinking/rumination. This would allow the individual greater cognitive capacity to engage emotion regulation
strategies—such as problem solving or evoking pleasant memories—to achieve mood repair (see Koster, De Lissnyder, Derakshan, & De Raedt, 2011).

**Implications to Study 3’s aims and methodology.** To the author’s knowledge, no ERP or neuroimaging studies of resistance to proactive interference inhibitory control for emotional stimuli in depression have been published. This is a limitation of the current literature as cognitive inhibitory control is a process that consists of various sub processes, including initial detection and subsequent regulation. Given the extremely fast temporal sequencing of these processes—within hundredths of milliseconds between them—revealing them with behavioural or neuroimaging procedures is extremely difficult. The high temporal resolution of ERP allows this technique to clarify the level of processing that might lead to these disruptions of cognitive control in depression. The Go/Nogo paradigm, as outlined in Section 2.2., is among the most well characterised measures of response inhibition in the electrophysiological literature (Aron, 2007; Chiu et al., 2008; Dillon & Pizzagalli, 2007). Electrophysiological Go/Nogo investigations have reliably linked two ERP components to inhibition, the Nogo-N2 and the Nogo-P3. These two ERPs are thought to reflect initial detection and subsequent regulation of interference material, respectively (Krompinger & Simons, 2011). Thus, Study 3 employed an emotional Go/Nogo protocol in an attempt to gauge further data on the hypothesised maladaptive inhibitory control processes identified in Joormann et al.’s (2007) model. Further, only a few previous investigations have examined resistance to distractor interference inhibitory control and resistance to proactive interference inhibitory control in the same task (Joormann et al., 2010). No previous investigations have examined both of these processes in remitted depressed samples. In an attempt to measure distractor interference and proactive interference inhibitory control within the one task, Study 3
modified the Go/Nogo task to include an initial Sternberg working memory task protocol, similar to that employed in Joormann and Gotlib (2008). This protocol is outlined in detail in Section 5.2. As neural stimuli are found to be processed differently to emotional stimuli in the context of the Go/Nogo task (Chiu et al., 2008), control participants were used as the baseline condition.

2.4. Overall Summary of Information Processing in Depression

The high recurrence rate in depression suggests that there are specific factors that increase an individual’s risk for developing repeated episodes of the disorder. One factor implicated in the literature is biases in the cognitive processing of negative information. This includes the tendency for sustained attention and elaboration of negative information versus positive information and biased autobiographical memory for negative versus positive events. Empirical evidence and contemporary models suggest that these negative cognitive biases are mediated by the impaired ability to utilize inhibitory control over the entry and removal of extraneous negative information in working memory. This deficit leaves the individual susceptible to emotional regulation deficits, such as rumination or impaired ability for positive memory recall (Demeyer et al., 2012; Joormann et al., 2007). Thus, the ability to effectively manage the contents of working memory can help discriminate individuals who are likely to cycle down into depression to those who do not. Currently there is moderate support (medium effect sizes) for each of these hypothesised cognitive processes as summarized in Table 3.
Table 3

Effect sizes for Emotional Effects for Attention, Memory, and Executive Control in Major Depressive Disorder

<table>
<thead>
<tr>
<th>Cognitive Domain</th>
<th>Task</th>
<th>Level of Evidence</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention</td>
<td>Dot-probe task</td>
<td>Meta-analysis</td>
<td>.52</td>
</tr>
<tr>
<td></td>
<td>Affective dichotic listening</td>
<td>1 to 5 studies</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>Oddball task</td>
<td>1 to 5 studies</td>
<td>n/a</td>
</tr>
<tr>
<td>Memory</td>
<td>Autobiographical Memory</td>
<td>Meta-analysis</td>
<td>.53 to .58</td>
</tr>
<tr>
<td></td>
<td>Old/New task</td>
<td>Meta-analysis</td>
<td>.19</td>
</tr>
<tr>
<td>Working Memory</td>
<td>Emotional Stroop task</td>
<td>Meta-analysis</td>
<td>.17</td>
</tr>
<tr>
<td>Memory</td>
<td>Affective Go/Nogo task</td>
<td>6 to 10 studies</td>
<td>.42</td>
</tr>
<tr>
<td></td>
<td>Affective n-back task</td>
<td>1 to 5 studies</td>
<td>.31</td>
</tr>
</tbody>
</table>

*Note.* Data is based on comparisons between individuals diagnosed with Major Depressive Disorder and healthy controls. n/a refers to not applicable. Data adapted from Christensen and King (2013).

2.5. Gaps in the Current Literature

The empirical evidence for these above inferences is still far from conclusive. Current research inconsistencies appear to be the result of different methodologies and to differences in stimuli and sample characteristics between studies. For instance, despite known gender differences in emotional information processing, many researchers analyse averaged data from samples that included both males and females. Others seem to use neutral stimuli as a baseline to compare participant’s reactions to valence stimuli regardless of known internal validity limitations with this approach (see section 2.2.3). A further limitation of previous research has been the narrow
focus on examining the relationship between depression and these different cognitive processes in different studies. Although the results from these independent experiments are connected by theory and review papers, to the researcher’s knowledge, no previous study has attempted to examine and statistically integrate them in one sample. This is a significant weakness. That is, the current literature effectively informs us about the possible noxious prognosis that each of these maladaptive cognitive patterns may have on depressed individuals. However, it does not explain how these processes may be related, or combine to predict depression status. This formed the main objective of the following four dissertation studies.

2.6. Aims, overview and significance for the Dissertation Studies

This dissertation aimed to assess the validity of the impaired cognitive control model proposed by Joormann et al.’s (2007). Specifically, it sought to examine the behavioural and ERP correlates of the implicated emotional information processing biases and deficits in female undergraduate individuals with MDD, remitted depression, and never-depressed healthy controls. To accomplish this, participants completed three ERP tasks aimed to assess behavioural and ERP markers of attention and encoding (Study 1), recent episodic memory (Study 2), and working memory inhibitory control (Study 3). It employed equal-probable presentation of positive and negative word stimuli. The data from these three ERP cognitive tasks were collated so as to test the theorised associative inferences and predictive ability of Joormann et al.’s (2007) model. This was verified through correlation (Study 1, 2, 3) and discriminant analysis (Study 4). Pilot investigations were first conducted for the three ERP tasks. An overview of the pilots and the four main studies is provided below.

Pilot studies. A pilot study was completed for each of the three experimental studies using a sample of dysphoric and control participants. Details are provided in
Appendix B, C, and D for the pilot of Study 1, 2, and 3, respectively. The pilot investigations were conducted to determine the required intensity of stimulus valence, arousal characteristics, and repetition rate. It verified the modified Sternberg Go/Nogo task designed for Study 3. Specifically that it elicited the expected Nogo-N2 and Nogo-P3 ERP components. It was also important to measure the time required to complete the three cognitive tasks (of Study 1, 2, and 3) in the single testing occasion, whilst being mindful not to induce fatigue effects on the data. This data is outlined in Appendix E. Last, the pilot studies were conducted to provide an indication of experimental power for the research questions. This information was used to help determine sample size requirements for the main experimental studies (Chapters 3-5). A brief outline of the key observations of the pilot studies and their implications for the design of the main dissertation studies is provided below.

The pilot studies identified a number of key design considerations. The first concerned stimulus repetition rates. In the pilot studies each word stimulus was presented five times during self-descriptive encoding (pilot for study 1), another five times during memory testing (pilot for study 2), and a further five times during the inhibition task (pilot for study 3). The high stimulus repetition rate was selected to ensure that participants encoded all items into memory. It was thought that this greater stimulus exposure would allow for subtle differences in proactive interference inhibitory control to be examined in study 3. However, the stimulus familiarity appeared to increase participant’s expectation for this information. In result this reduced the need for context updating during encoding, which served to flatten anticipated P3b effects (cf. the diminished P3b results in the pilot for Study 1). This is consistent with previous work that shows stimulus repetition results in habituated P300s (e.g., Polich & Kock, 1995; Ranganatha & Rainer, 2003). Increased stimulus
presentation also seemed to result in enhanced recognition memory accuracy in the pilot study’s dysphoric sample. This likely had the impact of diminishing any observable Old/New ERP or recognition memory negative biases for the dysphoric group (cf. pilot for Study 2). It also appeared that the small number of stimuli in the pilot studies may have rendered the cognitive tasks too simplistic, thereby not putting enough demand on the central executive. The small pool of negative and positive word stimuli used in the pilot studies could have also resulted in reduced chances for participant self-identification with the stimuli. This is a key limitation, as attention and memory biases in depression tend to be more reliably observed under conditions of self-descriptive encoding (see section 2.3). For these reasons a larger pool of stimuli were selected for the main dissertation studies. Each stimulus was selected to only be presented once during each of the three experimental tasks. This stimulus presentation rate replicates that employed in previous Old/New ERP work (e.g., Deldin et al., 2001; Dietrich et al., 2000; Langeslag & van Strien, 2008), and inhibitory control investigations (Chiu et al., 2008; Joormann & Gotlib, 2008).

The second design consideration concerned the arousal ratings of stimuli. In the collection of pilot studies neutral stimuli were included as a ‘baseline condition’ (as in previous work: e.g., Chiu et al., 2008; Ilardi et al., 2007). However, dysphoric participants were observed to exhibit significantly larger Nogo-N2 and diminished Nogo-P3s to successful inhibition of neutral proactive interference stimuli in the pilot for study 3 (see Appendix C). This effect might reflect less attentional engagement and thus, less access into working memory by these less arousing words. If this interpretation was correct it posed a significant problem to the study’s internal validity, as effective encoding of information into working memory was a prerequisite for proactive interference stimuli. The impacts of stimulus arousal was also evident
in Chiu et al.’s (2008) Go/Nogo study, which observed both positive and negative Go words to elicit enhanced P3 responses compared to neutral words. The researchers also agreed that this valence effect likely represented less engagement by the less arousing neutral stimuli. It was thus determined that use of neutral stimuli would confound the experimental results. That is, it would be difficult to draw conclusions regarding cognitive inhibition processes if the ERP effects could similarly be interpreted as an index of arousal differences’ between stimuli. For these reasons, in the dissertation studies all stimuli were specifically matched on arousal dimensions so to reduce any confusion in interpretation of ERP data (especially for Study 3). Accordingly only positive and negative stimuli were included.

The PCA of each of the pilot studies (see Appendix B-D) identified components that appeared reflective of the ERPs known to be evoked by the employed experimental tasks. This supports the validity of the cognitive tasks (especially for the modified Sternberg Gog/Nogo task of Study 3), and the selected epochs used for ERP extraction in the main studies. Appendix E suggested that participants’ fatigued over the course of ERP testing. Thus, the main studies need to keep testing to a minimum whilst continuing to collected adequate ERP samples to achieved good signal-to-noise ratings.

**Study 1.** Theory and experimental data suggest the existence of a negative attention bias in depressed, positive encoding bias in healthy controls, and similar processing/no bias in remitted depressed. Study 1 measured ERPs continuously during participants’ performance on a self-referential encoding task of equi-probable presentations of positive and negative personality adjectives. The aim of the study was to assess for predictions concerning attention orienting (enhanced P2 or P3a), context updating and schema expectation (diminished P3b and N400s, respectively, to
mood-congruent stimuli) and sustained elaboration (enhanced LPPs) in current and remitted depression. Their results were compared and contrasted to that of never-depressed healthy controls.

**Study 2.** There is robust support for biases in explicit memory for negative information in depression. Contrary to the ironic process theory (Wenzlaff et al., 2001), these biases have not been found to normalise after clinical remission (Dietrich et al., 2000). Previous ERP work has found overall reductions of the FN400 and LPC old/new effects in depressed samples (Dietrich et al., 2000). It is possible that this reflects deficits in working memory functioning in the disorder. However, to date limited research has been conducted here. Thus, the second study measured the FN400, LPC, and LPN ERPs in response to correctly recognised items and old items (those presented in Study 1) intermixed with new items. The study aimed to further investigate familiarity and recollection processes underlying memory for emotional information in current and remitted depression. It also sought to investigate the electrophysiological evidence of anomalous working memory functioning in these samples, and how this correlates to depression symptoms and emotional regulation.

**Study 3.** Previous findings have suggested that impaired inhibitory control of working memory may explain biased attention and memory for negative information in depression (Joormann et al., 2007; Joormann & Gotlib, 2008, Wisco, 2009; Zetsche & Joormann, 2011). Study 3 thus focused on investigating cognitive inhibitory processes. There were three main aims of the study. The first was to explore current and depressed participant’s ERP profiles during early (N2) and late (P3, LPP) cognitive inhibition of emotional material during a modified Sternberg working memory Go/Nogo task. The second sought to test if participant profiles differed in the context of the inhibitory control process examined: resistance to distractor
interference versus resistance to proactive interference. The third was to test the correlated predictions of Joormann et al.’s (2007) impaired cognitive control model of depression, such as the associations between participants’ inhibitory control deficits with their self-reported depression severity, rumination, and emotional regulation. The findings of this study may better help inform how working memory control and emotional regulation deficits interact in depression disorders, and how they are/or if they are amended with remission. This may help inform treatment interventions and future research avenues.

**Relevant statistical analysis procedures for Studies 1-3.** The dissertation hypotheses for Studies 1-3 pertained to main effects or interactions with stimulus valence and diagnostic classification. Thus, mixed ANOVA is the dominant statistical approach, and that suggested by Luck (2005) for analysis of ERPs. The benefit (and thus selection) of mixed ANOVA for the dissertation ERP analysis is that each subject is treated as a block; that is, they act as their own control condition. This reduces individual variability in ERP inflating results, which is a paramount consideration given the typically smaller samples analysed in ERP investigations as compared with behavioural analyses (Luck, 2005; Polich, 2007). It is argued that when ERP effects are large relative to the noise level, you can have some faith in the details of the results that are being analysed statistically (Polich, 2007). Thus, in an attempt to reduce the possibility of error in the current sample only large, well-established ERP components were analysed (e.g., P3, N400, Nogo-N2). Unless otherwise stated, an alpha level set at .05 was used for all statistical tests. Post hoc comparisons were Bonferroni corrected to ensure that Type I error was not inflated, with the exception of planned comparisons (Mitchell & Joley, 2004).
Study 4. The equivocal findings in depression emotional information processing have led researchers to call for the inclusion of tasks and measures that tap the different cognitive processes in a single study (Joormann et al., 2007). Thus, the aim of Study 4 was to integrate the associations of the cognitive processes measured in the three previous ERP studies. Discriminant function analysis (DA) was performed to determine whether the three groups could be distinguished on the linear combination of the cognitive processes implicated in Joormann et al.’s model. It sought to determine which variables contribute to the separation (Norusis, 2008).

Relevant statistical analysis procedures for Study 4. Discriminant analysis (DA) is a multivariate procedure, originally proposed by Fisher (Fisher, 1936), for predicting group membership and/or describing group separation on multiple variables. Predictive discriminant analysis is part of the general linear model and combines some of the features similar to multiple regression analysis. However, unlike regression, DA is used when the criterion variable is categorical or nominally scaled rather than interval or ratio scaled. In predictive DA, a set of rules is formulated that consists of as many linear combinations of predictors as there are groups. It is usually followed by classification procedures to categorize new cases based on the obtained discriminant function/s. Thus, the common objectives of DA are (1) to investigate differences between groups; (2) to discriminate groups effectively; (3) to identify key discriminating variables; (4) to perform hypothesis testing on the differences between the expected groupings; and (5) to classify new observations into pre-existing groups (Tabachnick & Fidell, 2007).

Significance of Dissertation Studies

The benefit of this dissertation is that it is the first time attention, memory, and working memory inhibitory processing have been examined in the same sample of
MDD, remitted depression, and never-depressed control healthy individuals. Further, it is the first ERP study of proactive interference inhibitory control in MDD, and the first time that resistance to distractor interference and resistance to proactive interference inhibitory control were investigated in the same sample of remitted depressed participants. Further, the study utilises a young, medication-free remitted sample. This is a particular strength of the dissertation, since older age (~ medium 40 years), medication use (typically SSRI), and recurrent depression (~ medium past major depressive episodes = 4) have already been shown to affect cognitive performances in remitted-depression (see Synder, 2012 for a review; Table A1; Appendix E). This new data will contribute to the MDD literature. Each study is built upon the last in a logical manner and culminated in the presentation of an integrated model, which has significant research utility. The main strength of the work resides in its examination of multiple facets of attention and memory within the same MDD and remitted depressed sample. It also includes longitudinal measurement of depression and anxiety levels using validated psychometrics. At the clinical level, it is expected that this research will inform how cognitive control predicts depression prognosis and risk for depressive relapse. This knowledge may provide a promising avenue for improvement of clinical interventions for female clients with depression.
Chapter 3

Study 1 – Emotional Attention Biases in Depression

Definition of Terms

*Cognitive Schema*Schemata*: An organised mental structure of an individual’s idiosyncratic knowledge and assumptions of self, other, and the world which is used for interpreting and processing information.

*ERP Arousal Effect*: The enhanced ERP positivity occurring around 300-400 ms following presentation of high-arousing compared to low-arousing stimuli.

*ERP Valence Effect*: The observation that emotional stimuli reliably modulate ERP components, appearing as early as 100 ms post stimulus presentation.

*Context Updating*: Updating one’s mental representations of the current environment.

*Mood-Congruent Processing*: The tendency to favour processing of information in the environment that is consistent with a person’s current mood or mental status.

*Orienting Attention*: Automatic attention directed towards a novel or potentially threatening stimulus.

*Semantic Incongruence*: When the content of presented semantic information is unexpected. For instance, “I take coffee with milk and dog” (Kutas & Hillyard, 1980). It evokes the N400 ERP.

*Suppression*: A response-focused strategy that consists of attempts to prevent or reduce ongoing emotion-expressive behaviour or thoughts (Gross & John, 2003).

3.1. Background and Hypotheses

The aim of the study was to assess for predictions concerning attention orienting (enhanced P2 or P3a), context updating and schema expectation (diminished P3b and N400s, respectively, to mood-congruent stimuli) and sustained elaboration (enhanced LPPs) in current and remitted depression. In an attempt to test for these biases, ERPs known to represent different components of schema activation and attention processing were recorded under conditions of self-referential encoding in a sample of MDD, remitted depressed, and healthy control participants.
3.1.1. Study background. As discussed in section 2.1., the traditional cognitive models of depression suggest that negative core schemas contribute to the development, maintenance, and recurrence of MDD by skewing information processing towards negative information (Beck, 1967; Teasdale & Barnard, 1993). However, failure to observe such global biases in depression has led to the development of alternative theories. The most popular of these was that proposed by Williams et al. (1988), later revised by the authors in 1997. As outlined in Section 2.3.1., the majority of ERP evidence supports the existence of a negative attention bias in depression for self-referential stimuli, whereas a positive attention bias is found for healthy controls (Deldin et al., 2001; Shestyuk & Deldin, 2010; Wisco, 2009). Both neuroimaging and ERP studies suggest that these attention biases should be pronounced over the right hemisphere given Heller’s (1990, 1993) theory of hypoactive right parietal -temporal cortex in depression.

Depressive schemata are presumed to become inactive upon symptomatic recovery from depression. However, they remain available, thereby constituting a vulnerability to future episodes of depression (Beck, 1967; Beck et al., 1979; Segal & Ingram, 1994). Based on this premise, the detection of depressive cognitions in remitted depression is dependent on these latent depressive schemas becoming primed, such as by stressful events or sad mood induction (see Ingram, Miranda, & Segal, 1998; Miranda & Persons, 1988; Segal & Ingram, 1994). Several studies have detected depressive thoughts in individuals recovered from depression when participants were primed in a sad, but not a euthymic mood (Ingram, Bernet, & McLaughlin, 1994; Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990). In times of euthymic mood it is theorised that remitted depressed individual will engage in suppression over negative thoughts that are consistent with their latent negative schemata (Wegner,
This serves to maintain their positive state. This theory is supported by previous studies that have failed to find evidence of a negative self-referent encoding bias in remitted depressed subjects under conditions of eurythmic mood (e.g., Blackburn & Symth, 1985; Dobson & Shaw, 1987; Moretti et al., 1996). In fact, some studies report remitted depressed participants do endorse more positive than negative adjectives as self-descriptive, and less negative words as self-descriptive compared to clinically depressed samples (Moretti et al., 1996). However, some previous investigations have observed remitted depressed samples to exhibit depression-specific self-endorsement biases without mood-induction protocols (e.g., Fritzche et al., 2010). It might be speculated that such biased cognitive processing in these studies are only present in sub-groups of remitted-depressed patients, for example, those continuing to use antidepressant medications (e.g., 30% of the remitted depressed participants in Fritzche et al.).

Depression does not appear to be related to enhanced orientating attention to negative stimuli (see Section 2.3.1.; Bradley et al., 1997; Hill & Dutton, 1989; MacLeod et al., 1986; Mogg et al., 2000; Mogg et al., 1993; Neshat-Doost et al., 2000; Williams et al., 1997). This is contrary to the predictions based on Beck and Bower’s cognitive models. This failure to observe attention biases in depression led to Williams et al.’s (1997) argument that depression is not characterised by biased early attention processes. More recent studies have suggested that negative attention biases emerge only under conditions of long stimulus exposure (i.e., >1000 ms) in both depressed (Joormann & Gotlib, 2007; Joormann et al., 2007; Karparova et al., 2007) and dysphoric samples (Caseras et al., 2007; Koster et al., 2005; Koster et al., 2006; Shane & Peterson, 2007). A similar pattern of results has emerged in the ERP literature. Negative attention biases in depression have been more reliably observed at
longer latency ERPs (e.g., P3, SW: Blackburn et al., 1990; Deldin et al., 2001; Deldin et al., 2009; Deveney & Deldin, 2004; Nandrino et al., 2004; Shestyuk & Deldin, 2010; Shestyuk et al., 2005), compared to early processing stages (e.g., P1, N1, P2: Deldin et al., 2000; Dietrich et al., 2000; Serfaty et al., 2002; Shimizu et al., 2006). This suggests that depressed individuals may experience an inability to disengage from salient negative information once it reaches awareness (Gotlib & Joormann, 2008; Wisco, 2009). However, as outlined in detail in Section 2.3.1., there are divergences in the interpretation of ERP components employed to measure these encoding and attention processes in the depression literature. This is particularly the case for the P3b.

Recall from Section 2.2.1., the P3b is thought to reflect current memory mechanisms triggered when the mental model of the stimulus environment is refreshed and revised (Donchin and Coles, 1988). This is known as context updating (Donchin & Coles, 1988). Larger P3b magnitude corresponds to a mismatch between the received and the expected stimulus information based on current working memory storage. It is typically evoked in the oddball task (see Section 2.2.1.).

Ilardi et al. (2007) employed an oddball task. Recall, from Section 2.2.1., within the oddball paradigm, the amplitude of the P3b has shown to be inversely proportional to the frequency of the occurrence of the oddball stimulus. It is largest when the stimulus is rarest (Donchin & Coles, 1988; Linden, 2005). Using negative stimuli acting as the rare/oddball stimulus, Ilardi found enhanced P3bs to negative stimuli in their dysphoric participants. They argued that this effect was indicative of a depressive negative attention bias. This interpretation is consistent with the assumption that the P3 amplitude is a measure of attentional allocation (Kok, 2001). However, it is inconsistent with the findings of Blackburn et al. (1990) and Yang et al.
(2011), who found smaller P3bs to negative stimuli in their dysphoric and depressed participants, respectively. In line with context-updating hypotheses of the P3b, both Blackburn et al. and Yang et al. argued that given the P3 is considered to be associated with a person’s evaluation of their hypotheses about future events or stimuli (Donchin & Coles, 1988; Fabiani et al., 1986; Karis et al., 1983), reduced P3s are therefore anticipated in this sample given their negative cognitive schemata (Beck, 1967; Beck et al., 1979). That is, they expect negative information to be presented.

It is possible that the divergent findings and interpretations of the P3b in this literature are due to methodological and sample differences between studies. For instance, Ilardi et al.’s MDD participants presented with only mild severity of depressive symptoms at ERP testing (average BDI-II score = 18.3). Further, these depression scores were not stable, as indicated by marked decreases in participants’ BDI-II scores at follow-up assessment. Further, the patient sample in the Ilardi et al. study only used the Mood Disorders module of the SCID-I/NP in participant recruitment. Thus, it is unknown if MDD is the primary or only diagnosis in this sample. It is possible that unaccounted comorbid anxiety, substance misuse, eating, or psychotic disorders account for their divergent P3b findings to that observed in previous work. This raises concerns over the external validity of their depression classifications. These results do not readily generalise to the severely depressed patients investigated in Blackburn et al. (1990) and Yang et al. (2007).

Nandrino et al.’s (2004) also identified larger P3b in their sample of inpatient, medicated MDD patients. This sample contrasts to the unmedicated, outpatient sample analysed in Yang et al. (2007). It is possible that medication status in Nandrino et al. introduced an experimental confound, as antidepressants are known to influence emotional information processing, particularly towards the positive...
Antidepressants have also been observed to increase the magnitude of the P3b (see Bruder et al., 2012 for a review). Last, both Ilardi et al. and Nandrino et al. utilised an emotional oddball paradigm, with the negative words acting as the rare/target stimulus that required a specific response (20% occurrence in both studies). Thus, the P3b is known to be larger for rare stimuli that require a specific response. This is in contrast to the interpretation of the P3b evoked under conditions of passive viewing and equiprobable presentation of stimuli, as used in Blackburn et al. In the framework of context updating (Donchin & Coles, 1988), larger P3b magnitude is expected here to correspond to a mismatch between the received and the expected information. For instance, due to their greater negative schemata, depressed individuals should naturally expect negative stimuli in their environment. This means that the positive stimuli would act as the unexpected material, and thus will evoke larger P3bs compared to the negative stimuli (Blackburn et al., 1990; Yang et al., 2011). Using Heller’s (1990) theory of aberrant right posterior activity in depression, this effect was anticipated to be particularly noticeable in, or specific to the right parietal sites (Deldin et al., 2001).

Another key ERP known to be evoked in response to contextual anomaly is the N400. The N400 is held to mark elaborative stimulus processing that is modulated by semantic content (Kutas & Federmeier, 2000). Larger N400 amplitudes are associated with words judged to be semantically unlikely, whereas smaller N400 amplitudes are associated with expected semantic stimuli (Kutas & Hillyard, 1980; van Petten & Kutas, 1990). Similar to the reasoning provided above, negative items are expected to be less distinctive and more frequent with regard to depressive cognitive schemata. Previous work supports this prediction, reporting reduced frontal N400 for the
negative items in depressed samples (Dietrich et al., 2000). The frontal distribution of this effect may represent poor executive control activation to inhibit depressive rumination here (e.g., Berman et al., 2011).

The P2 component is a positive-going deflection maximally occurring at anterior and central sites around 200 ms after stimulus presentation. Studies have shown that it is sensitive to affective evaluation (Begleiter, Porjesz, & Garozzo, 1979) and has been linked to greater orientating attention towards threat, such as negative information (Bartholow et al., 2003). Enhanced P2s towards negative information have been observed in the normative literature. This effect has been interpreted as automatic attentional orientating to threatening information, typically subserving defensive coping or reparative coping. Null ERP evidence has been found for early components thought to reflect orientating attentional processes (i.e., $P_{100}$, $P_{200}$, $N_{200}$: Deldin et al., 2000; Dietrich et al., 2000; Shimizu et al., 2006). These null findings in attention orientating contradict Beck’s (1967) cognitive theory of depression, which emphasises the function of automatic biases towards schema-congruent negative information. However, see Shestyuk and Deldin (2010).

The ability to quickly detach attention from further processing of unhelpful negative stimuli (when it is deemed to be not relevant) represents a fundamental aspect of emotional regulation (Derryberry & Rothbart, 1997; Joormann et al., 2007; Nolen-Hoeksema, 1991; Posner & Rothbart, 2000). For instance, Ellenbogen, Schwartzman, Stewart, and Walker (2002) found that an aversive stressor (continually losing at a computer game) was associated with rapid disengagement of attention from negatively valanced words in their healthy control sample. They interpreted this attentional avoidance behaviour as an adaptive means to regulate emotional arousal. This effect was not found for positive or neutral word stimuli. These results are consistent with
the social psychology literature, which has shown healthy control individuals to actively inhibit the processing of negative information related to the self (Taylor & Brown, 1988). Theoretical accounts of self-regulation highlight the importance of attention in modulating affect (Derryberry & Rothbart, 1997; Joormann et al., 2007; Nolen-Hoeksema, 1991; Posner & Rothbart, 2000). The LPP is thought to be specifically sensitive to deliberate emotional regulation strategies such as reappraisal (DeCiccoa et al., 2011; Foti and Hajcak, 2008; Hajcak and Nieuwenhuis, 2006; MacNamara et al., 2011), directed attention (Ferrari et al., 2008; Hajcak et al., 2009), and directions to increase and decrease subjective emotional responses (Moser et al., 2006, 2009; Krompinger et al., 2008). Its amplitude is known to decrease when participants follow reappraisal instructions to decrease distress elicited by unpleasant stimuli (Hajcak & Nieuwenhuis, 2006; Krompinger et al., 2008; Moser et al., 2006; Moser, Most, & Simons, 2012; Moser et al., 2009) and increases when they follow reappraisal instructions to increase distress elicited by unpleasant stimuli (Moser et al., 2012; Moser et al., 2009).

Never-depressed individuals show a greater LPP and superior recall for pleasant personality adjectives (Herbet, Junghofer, & Kissler, 2007). This suggests that positive material attracts sustained attention in non-depressed samples, which results in its deeper encoding. The lateral PFC—a region implicated in the generation of the LPP (Nieuwenhuis, Aston-Jones, & Cohen, 2005)—is also known to be associated with sustained attention and approach behaviours (Gray, 2001; Gray, Braver, & Raichle, 2002). Thus, the LPP enhancement for positive stimuli would be

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1 It should be noted that attentional avoidance of negative stimuli in the normative population is inconsistent with previous reports that suggested that all humans are pre-wired to automatically orient toward potential threat in the environment for survival (e.g., LeDoux, 1995). However, it could be argued that the bias toward threat stimuli in the general population may be specific to naturalistic or biologically valid stimuli (e.g., pictures of blood or sounds of screams) and not hold for negatively valanced word stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007).
expected over anterior electrode positions. In the depression literature, Shestyuk et al.,
(2005) found their MDD exhibited reduced late ERPs to positive relative to negative
self-descriptive word stimuli compared with control individuals. These researchers
suggested that cognitive deficits in MDD may thus originate from inadequate
sustained processing of positive information, which serves to repair mood.

Most of the previous work outlined above has focused on older depression
samples (~ medium 40 years). Given the prevalence rates of pre-adult onset
depression is reported to be approximately 22-40% (Alpert et al., 1999; van Noorden
et al., 2011; Zisook et al., 2007) it is possible that the majority of previous work
comprise adult-onset depression. Researchers have questioned whether pre-adult and
adult-onset depression represents different subtypes of the disorder (Kaufman, Martin,
King, & Charney, 2001). Specifically, prior investigation has found that pre-adult
onset of depression predicts a more severe course of the disorder, with it associated
with more suicidal behaviour and comorbid DSM-IV-TR diagnoses (Noorden et al.,
2011), greater social and occupational impairments, female prevalence, and depressive
relapse and chronicity (Benazzi, 2000; Croyell et al., 2009; Zisook et al., 2007). Pre-
adult onset MDD appears more strongly associated with genetic predispositions and
prejudicial early childhood risk factors, such as attachment instability, abuse, and/or
psychopathology in the family (Jaffee et al., 2002; Klein, Lewinsohn, Seeley, &
Rohde, 2001). Cognitive and emotional processing undergoes significant development
during adolescence. Onset of depression earlier in life, especially during crucial
developmental periods such as adolescence may have implications for later
compromised emotional regulation as a result of disrupted maturity in these neural
networks (Forbes & Dahl, 2005). Few studies have investigated the cognitive profile
3.1.2. Study hypotheses. Based on the above theory and research, the following experimental hypotheses were made:

**Hypothesis 1** predicted control and remitted-depressed participants to endorse significantly more positive words more self-descriptive than the MDD participants. Alternatively, MDD participants would endorse more negative words as self-descriptive compared to both the control and remitted groups. This hypothesis was tested using 3 (group) × 2 (valence) factorial ANOVA, with the simple main effects of the predicted interaction analysed using planned comparisons ($\alpha = .05$).

**Hypothesis 2(a)** predicted MDD to exhibit significantly larger P3bs for positive than negative stimuli over the right hemisphere. Whereas, control and remitted participants were expected to show larger P3b for negative than positive word stimuli across all lateral positions. To analyse this hypothesis peak P3b amplitudes were subjected to a 3 (group) × 3 (laterality) × 2 (valence) factorial ANOVA. Predicted simple main effects examined positive and negative stimuli P3bs at left, midline, and right lateral positions for each group separately. Given the inconsistency in P3b effects in the depression literature, the alpha level was Bonferroni corrected (.017: = .05/3 tests) in an attempt to reduce inflation of Type I error.

**Hypothesis 2(b)** predicted that across all participants greater right hemisphere P3bs for positive relative to negative stimuli (P3b bias scores) would positively correlate with self-reported depressed mood, rumination and suppression scores, and to negatively correlate with self-reported emotional regulation scores.

**Hypothesis 2(c)** predicted that MDD participants would also show reduced/diminished N400 (schema incongruence) for negative relative to positive
stimuli. This should be pronounced at frontal electrode sites (Dietrich et al., 2000). This hypothesis was analysed using a one-way ANOVA.

Hypothesis 3 predicted control participants to exhibit significantly larger P2 for negative than positive stimuli, suggesting orientating attention to this threatening information. To evaluate this hypothesis peak average P2 amplitudes were subjected to a 3 (group) × 3 (laterality) × 3 (caudality) × 2 (valence) factorial ANOVA. The predicted simple main effect at right frontal sites for the controls were analysed using planned comparisons (\( \alpha = .05 \)).

Hypothesis 4 predicted a laterality-by-caudality-by-valance-by-group interaction for the LPP data. Simple main effect analysis should show both control participants to exhibit significantly smaller LPP for negative than positive stimuli over right frontal sites (Herbet et al., 2007). Suggestive of poor emotional regulation and deficient executive control, this effect was not expected in the MDD or remitted samples. The hypothesis was analysed using a 3 (laterality) × 3 (Caudality) × 2 (Valence) × 3 (Group) ANOVA. Predicted simple main effects examined positive and negative stimuli LPPs at frontal, central, and parietal caudal positions for each group separately. Given the larger variability of the LPP (large time epoch) and the exploratory nature of the hypothesis, the alpha level was corrected to .017: (.05/3 tests) in an attempt to reduce inflation of Type I error.

Replication predictions/null expectations. As outlined throughout the literature review (see section 2.3.1.), MDD does not appear to be associated with attention biases at the level of attentional orienting. Thus, similar frontal P3a and P2 amplitudes were expected for positive and negative stimuli during the self-descriptive attention task for participants.
3.2. Method

3.2.1. Participants

Participants were recruited from a pool of 234 female undergraduate students aged between 17 – 35 years. All these students completed an online screening questionnaire to measure depression, anxiety, and stress levels via the DASS-21 (Lovibond & Lovibond, 1995) and details of any personal history of psychopathology and mental health treatment, and family history of psychopathology. Potential participants who met preliminary eligibility criteria—for instance, reported no personal history of psychosis, bipolar disorder I and II, attention deficit hyperactivity disorder, obsessive compulsive disorder, eating disorder, or substance abuse disorder—were contacted via telephone by the PhD candidate. Here, additional exclusion criteria were assessed. Exclusion criteria here included history of neurological disorders or insults, history of electroconvulsive therapy, presence of visual or reading disabilities or issues such as reading fluency, current or past substance abuse, and presence of medical problems or current medications that might confuse the EEG recordings.

A sample of 102 qualifying participants was invited to the cognitive laboratory for diagnostic assessment. This was completed using Structured Clinical Interview for DSM-IV, Non-patient Edition (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 2002) and Module K (psychotic disorders and mood disorder with psychotic features) of the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). All clinical assessments were conducted face-to-face by a psychologist with Masters-level training in clinical psychology. Consistent with DSM-IV-TR diagnostic procedure (APA, 2000), the primary diagnosis of each participant was the disorder
that was reported to be most distressing at the time of assessment. Participants were interviewed a few days prior to ERP testing. The time interval between diagnostic assessment and the electrophysiological testing ranged between two to 15 days, with a mean time interval of 10.51 days ($SD = 5.75$). Inter-rater reliability analysis using Cohen’s kappa statistic was performed to determine reliability of clinical diagnosis of the participants. A random selection of 11 (approximately 10% of the assessed sample) audio-taped SCID-I/NP and MINI interviews was reassessed by an independent psychologist, with a similar degree of training. This psychologist was blind to the first rater’s scores and diagnosis. Items of the SCID-I/NP that were scored with a ‘?’ due to insufficient data were rescored as absent (score = ‘1’).

According to Fleiss (1981), kappa values lower than 0.40 can be interpreted as ‘poor agreement’, between 0.41 and 0.75 can be interpreted as ‘fair agreement’ and above 0.75 as ‘excellent agreement’. A kappa of 0.80 ($p < .001$) was found for the total inter-rater reliability for the SCID-I/NP interview, with a kappa of 0.79 ($p = .007$) observed for the consistency of MDD diagnoses.

A sample of 75 participants was selected to proceed to the ERP experiment phase. Two participants were excluded from data analysis, one due to a corrupted data file which could not be accessed, and the other due to low ERP signal-to-noise ratios (50% of their data’s baseline peak EEG amplitude exceeded 100µV or baseline-to-peak drift exceeded 100µV.). This resulted in a final sample of 73 participants. Participants were placed into one of three groups according to their SCID-I/NP diagnoses. These included:

1. **Current depressed** group (MDD; $n = 30$). This group included participants who currently met SCID-I/NP criteria for MDD. Participants with a comorbid current or past anxiety disorder (with the exclusion of obsessive compulsive
disorder) were eligible provided that MDD was their primary diagnosis. Details of participant co-morbid anxiety diagnoses are outlined in Appendix F.

2. **Remitted depressed** group (remitted; \( n = 13 \)). This group included participants who met past, but not current, SCID-I/NP criteria for MDD. Participants were excluded from this group if they were currently on or had recently stopped (< one month) antidepressant medications, were currently involved in psychotherapy, or met DSM-IV criteria for another Axis I disorder.

3. **Healthy control/never depressed** group (control; \( n = 30 \)). This group included participants who never met DSM-IV-TR criteria for a past or present mental disorder.

The age mean age of the selected participants was 20.21 years with a standard deviation of 3.99 years. All were right-handed, spoke and read English fluently and possessed normal or corrected-to-normal vision and hearing. All reported a medical history devoid of neurological disorders that might confuse EEG results. Participants reported no colour-blindness, reading disorders, or past or current substance abuse or addiction. Each reported a psychological history devoid of disorders that are said to be characterised by deficit inhibitory processes (e.g., psychosis, mania, attention deficit hyperactivity disorder, and bulimia nervosa). As can be seen in Table 4, participants in the three groups were similar in age and years of education.

Participants in the MDD group self-reported higher prevalence of emotional disorder in their immediate family. A quarter of the MDD group, and almost one seventh of the remitted group, had undergone previous mental health treatment (typically based on CBT interventions or prescribed antidepressant medication). All participants in the MDD and remitted group reported pre-adult onset depression.
Table 4

**Participant Demographics: Means and Standard Error of the Mean (in parenthesis)**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
<th>Test of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>19.45 (0.33)</td>
<td>20.64 (1.05)</td>
<td>21.03 (0.94)</td>
<td>(F(2, 72) = 2.35, \ p = .152)</td>
</tr>
<tr>
<td>Years of Education</td>
<td>13.02 (0.26)</td>
<td>13.75 (0.33)</td>
<td>13.54 (0.33)</td>
<td>(F(2, 72) = 1.58, \ p = .212)</td>
</tr>
<tr>
<td>Number of Past Major</td>
<td></td>
<td></td>
<td></td>
<td>(F(2, 70) = 92.93, \ p &lt; .001)</td>
</tr>
<tr>
<td>Depressive Episodes</td>
<td>0</td>
<td>1.69 (0.95)</td>
<td>2.30 (0.84)</td>
<td>- MDD &gt; control  (p &lt; .001)</td>
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<td></td>
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<td>- RMT &gt; control  (p &lt; .001)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD = remitted  (p = .023)</td>
</tr>
<tr>
<td>History of Mental Health Treatment</td>
<td>0%</td>
<td>14.3%</td>
<td>25%</td>
<td></td>
</tr>
<tr>
<td>First Episode Depression</td>
<td>0%</td>
<td>46.15%</td>
<td>16.67%</td>
<td></td>
</tr>
<tr>
<td>Recurrent Depression</td>
<td>0%</td>
<td>53.84%</td>
<td>83.34%</td>
<td></td>
</tr>
<tr>
<td>Family History of Depression</td>
<td>25.8%</td>
<td>50%</td>
<td>67.9%</td>
<td></td>
</tr>
<tr>
<td>Family History of Anxiety</td>
<td>12.9%</td>
<td>35.7%</td>
<td>35.7%</td>
<td></td>
</tr>
</tbody>
</table>

At the time of ERP testing, four participants in the MDD group self-reported that they were being treated with selective serotonin reuptake inhibitor (SSRI) medication for their depressive symptoms\(^2\). The impact of these medications on the experimental results is outlined in each of the studies (subsequent analyses were conducted on all results with these participants excluded from the dataset). No participants in the control or remitted group were using psychotropic medications.

All participants were informed of the experimental procedures and their rights and obligations (Appendix G). All had the capacity to, and provided written consent

\(^2\) Medications and dosages were as follows: *Participant a* - 75mg Effexor (venlafaxine hydrochloride)/daily; *participant b* - 40mg Celexa (citalopram hydrobromide)/daily; *participant c* - 150mg Zoloft (sertraline) /daily; *participant d* - 75mg Zoloft (sertraline) /daily.
to participate in the study (Appendix H). All were treated in strict accordance with the National Statement on Ethical Conduct in Research Involving Humans (Griffith University Ethics protocol number: PSY/66/10/HREC). Participants were compensated with one credit point towards their first-year psychology course for each hour of participation in the study and entry into a $200 prize draw.

3.2.2. Materials

**Self-report psychometric assessments.** A summary of the psychometric properties of the following self-report tests is provided in Appendix I.

**Subjective Ratings of Mood and Fatigue.** A nine-point Likert scale was used for participants to rate their mood and fatigue levels over the course of the ERP testing. The two scales were presented to participants via a computer monitor. They were asked to ‘rate your overall mood, from 1 (positive) to 9 (depressed)’. Responses were made using number keys on the computer keyboard. Participants were then asked to ‘rate your level of energy, from 1 (energetic) to 9 (exhausted)’. Responses again were made using number keys on the computer keyboard.

**The Depression Anxiety Stress Scale-21 item version** (DASS-21; Lovibond & Lovibond, 1995). The DASS-21 was used to provide measures of the severity of depressive symptomology, anxious agitation, and generalised stress for participant screening. Each item is rated on a 4-point scale from 0 (did not apply to me at all) to 3 (applied to me very much, or most of the time) to indicate the frequency that respondents have experienced each symptom over the past week. An example of a depression item is: “I couldn’t seem to experience any positive feeling at all”. The DASS-21 was selected over the DASS-42 as it is shorter and maintains similar psychometrics. The DASS-21 has omitted items identified as problematic in the DASS-42 and has a more specific latent structure (Antony et al., 1998; Clara, Co, &
The DASS-21 scales possess good convergent and discriminant validity and high internal consistency in clinical and nonclinical samples (Antony, Bieling, Cox, Enns, & Swinson, 1998; Brown, Chorpita, Korotitsch, & Barlow, 1997; Daza, Novy, Stanley & Averill, 2002; Lovibond, 1998; Lovibond & Lovibond, 1995; Norton, 2007).

**Beck Depression Inventory-Second Edition** (BDI-II: Beck et al., 1996). The BDI-II was used to determine the presence and severity of depression in individuals at ERP testing and one-month follow-up. Answers are recorded on a 4-point scale (0 to 3) designed to determine levels of severity. Each item represents a symptom characteristic of depression as prescribed by the DSM-IV, such as sadness, guilt, suicidal thoughts and loss of interest (good face validity). The summed score for the BDI-II (range 0 to 63) is compared with pre-established degrees of depression: minimal (1-13), mild (14-19), moderate (20-28) and severe (29-63; Beck et al., 1996). The BDI-II has demonstrated a test-retest reliability of .90 (Beck et al., 1996). It has an internal consistency of .92 to .93 in a psychiatric outpatient sample, and .93 in a sample of college students (Beck et al., 1996). It has demonstrated a high convergent validity with other depression measures, such as the Hospital Anxiety Depression Scale, but less impressive discriminative validity (Beck et al., 1996).

**Emotion Regulation Questionnaire** (ERQ; Gross & John, 2003). The ERQ was used to assess self-reported frequency of (1) emotional reappraisal or (2) emotional suppression in participants. Emotional reappraisal is an antecedent-focused strategy that works to alter the experience of emotion by changing the focus of a person’s thoughts (usually towards positive events or problem solving) during stress. Emotional suppression is a response-focused strategy that consists of attempts to prevent or reduce ongoing emotion-expressive behaviour or thoughts. The habitual
use of these different emotional regulation strategies has implications for well-being. People who use reappraisal experience less depression and greater life-satisfaction than those who habitually suppress emotions (Gross & John, 2003). The ERQ uses a 7-point-Likert scale from *strongly disagree* to *strongly agree*. The reappraisal subscale consists of six items: e.g., “I control my emotions by changing the way I think about the situation I’m in”. The suppression subscale consists of four items: e.g., “I keep my emotions to myself”. Higher scores indicate more frequent use of each strategy. Previous studies have shown adequate internal consistencies (.88) for both subscales, and a two-month test-retest reliability of .70 (Gross & John, 2003).

**The State Trait Anxiety Inventory – State Subscale** (STAI-S; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The state subscale of the STAI-S was used to quantify the intensity of current anxiety levels in the participants at ERP testing and one-month follow up. State anxiety is conceptualised as subjective feelings of tension, apprehension, nervous symptom hyper-arousal, and worry. Items use a 4-point scale of symptom intensity (ranging from *not at all*, *somewhat*, *moderately so*, and *very much so*). Scores range from 0-60, with higher scores corresponding to greater levels of state anxiety. According to studies by Spielberger (1970), test-retest correlations for the STAI-S is .54 and an internal consistency ranging from $\alpha = .88$ to $\alpha = .93$. It shows adequate convergent validity with the Manifest Anxiety Scale and the Anxiety Scale Questionnaire ($\alpha = .73$ to $\alpha = .75$) (Spielberger, Sydeman, Owen & Marsh, 1999).

**The 22-item Ruminative Responses Scale** (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The RRS was used to determine how participants tend to respond to symptoms of dysphoria and stress. The RRS assesses responses to dysphoric mood that are focused on the self (e.g., “I think about all your shortcomings, failings, faults, mistakes”), on symptoms (e.g., “I think about how hard it is to concentrate”), or on
potential consequences and causes of moods (e.g., “I analyse recent events to try to understand why you are depressed”). It uses a 4-point scale from *almost never* to *almost always*. The RRS has a reported alpha coefficient of .90 and test-retest correlation of .67 (Treynor et al., 2003). It significantly correlates with the BDI, suggesting convergent validity; here higher correlations are observed for the brooding subscale, $r = .44$, as compared with the reflective subscale, $r = .12$ (Treynor et al., 2003). The division of the RRS into brooding and reflective subscales was modelled after Treynor et al. (2003), who found both subscales to have acceptable internal consistencies and test-retest reliabilities. Showing good convergent and ecological validity, the RRS has shown to correlate strongly with ruminative entries in a 30-day diary study (Nolen-Hoeksema, Morrow, & Fredrickson, 1990).

**Structured clinical administered interviews.**

*The Structured Clinical Interview for DSM-IV, Non-patient Edition* (SCID-I/NP: First et al., 2002). The SCID-I/NP is a semi-structured clinical interview that assesses current or past DSM-IV-TR diagnosis for anxiety, mood, psychotic, alcohol and substance abuse, somatoform, and eating disorders. It consists of a number of modules that correspond with DSM-IV axes and classes of disorders. It can only be used by trained professionals who specialised in clinical psychology (Williams et al., 1992). Each module is constructed as a schematic algorithm that leads to a diagnostic conclusion, based upon the interviewee’s reporting and the assessor’s clinical judgement. The SCID-I has shown adequate inter-rater reliability for all disorders ($r$ range from .69 –1.0; Zanarini & Frankenburg, 2001) and adequate test–retest reliability over a one-to-three-week interval ($r$ range from .40 –1.0; Williams et al., 1992; Zanarini & Frankenburg, 2001). For the current study, the January 2010 SCID Screening Module, Mood Episodes (Module A), Mood Disorders (Module D),
Module K of the MINI was used to assess the potential presence of psychotic symptoms in the sample. It was selected over the Psychotic Symptoms (Module B) and Psychotic Differential (Module C) of the SCID-I/NP due to its brevity. Module K of the MINI maintains high reliability and validity. It is based on questions frequently asked by physicians when assessing psychotic symptoms, making it user friendly. It has found to exhibit acceptable reliability (kappa coefficient: 0.41 - 0.68), sensitivity (.41 - .86), and specificity statistics (0.84 - 0.97; Amorim, 2000; Sheehan et al., 1998).

Experimental stimuli. Experimental stimuli included trait words selected from standardized databases for valence, familiarity, and number of syllables (Anderson, 1968; Dumas, Johnson, & Lynch, 2002; Kirby & Gardener, 1972). These databases have been used in previous behavioural and neural studies of information processing (Beer & Hughes, 2010; Ochsner et al., 2005). The PhD candidate selected potential words from this database to be used as the experiment stimuli using several constraints. Words that were not familiar to the PhD candidate and variants of an already selected word (e.g., admirable, admired) were eliminated. This left a pool of 282 words, from which 135 positive and 135 negative words were randomly selected.

These 270 adjectives were then evenly distributed into three experimental lists: List A, B, and C (see Appendix J). Each list included 45 positive adjectives (e.g., considerate, friendly, nice), and 45 negative adjectives (e.g., selfish, vulgar, worthless). As shown in Table 5, the three lists were counterbalanced for use as the target list for the self-referential encoding task used in Study 1 (and subsequent old
list for the Old/New task for Study 2). The new list for the Old/New task, the third for the distractor list for the modified Sternberg working memory Go/Nogo task.

Table 5

**Counterbalance Order for Lists across Dissertation Studies**

<table>
<thead>
<tr>
<th>Counterbalance Condition</th>
<th>Number of Participants</th>
<th>Target list (Study 1) &amp; Old list (Study 2)</th>
<th>New list (Study 2)</th>
<th>Distractor list (Study 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13</td>
<td>List A</td>
<td>List B</td>
<td>List C</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>List B</td>
<td>List C</td>
<td>List A</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
<td>List C</td>
<td>List A</td>
<td>List B</td>
</tr>
<tr>
<td>4</td>
<td>12</td>
<td>List A</td>
<td>List C</td>
<td>List B</td>
</tr>
<tr>
<td>5</td>
<td>14</td>
<td>List C</td>
<td>List B</td>
<td>List A</td>
</tr>
<tr>
<td>6</td>
<td>11</td>
<td>List B</td>
<td>List A</td>
<td>List C</td>
</tr>
</tbody>
</table>

Within each list, positive stimuli had significantly greater likeableness, familiarity, and frequency of use (per 10 000 words) than negative words. These statistics are shown in Table 6. Between list differences did not exist for stimuli likeableness or familiarity ratings, word length, number of syllables, or frequency of use (all \( p > .100 \)). The number of syllables and word length did not differ between positive and negative stimuli within each list.
### Table 6

*Means and SEM (in parentheses) and Test of Difference for Word List Stimuli*

<table>
<thead>
<tr>
<th></th>
<th>Positive</th>
<th>Negative</th>
<th>Test of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>List A</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likeableness Rating***</td>
<td>5.04 (0.07)</td>
<td>1.69 (0.03)</td>
<td>$F(1, 89) = 1898.01, \ p &lt; .001$</td>
</tr>
<tr>
<td>Familiarity Rating*</td>
<td>5.76 (0.03)</td>
<td>5.64 (0.03)</td>
<td>$F(1, 89) = 6.76, \ p = .011$</td>
</tr>
<tr>
<td>Word Length</td>
<td>8.64 (3.90)</td>
<td>8.78 (0.41)</td>
<td>$F(1, 89) = 0.56, \ p = .814$</td>
</tr>
<tr>
<td>Number of Syllables</td>
<td>2.84 (1.65)</td>
<td>2.93 (0.18)</td>
<td>$F(1, 89) = 0.13, \ p = .717$</td>
</tr>
<tr>
<td>Word Frequency**</td>
<td>51.16 (13.96)</td>
<td>5.95 (1.31)</td>
<td>$F(1, 89) = 10.38, \ p = .002$</td>
</tr>
<tr>
<td><strong>List B</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likeableness Rating***</td>
<td>5.17 (0.03)</td>
<td>1.71 (0.04)</td>
<td>$F(1, 89) = 4968.45, \ p &lt; .001$</td>
</tr>
<tr>
<td>Familiarity Rating***</td>
<td>5.82 (0.01)</td>
<td>5.54 (0.05)</td>
<td>$F(1, 89) = 28.37, \ p &lt; .001$</td>
</tr>
<tr>
<td>Word Length</td>
<td>8.40 (0.35)</td>
<td>8.07 (0.42)</td>
<td>$F(1, 89) = 0.37, \ p = .543$</td>
</tr>
<tr>
<td>Number of Syllables</td>
<td>2.80 (0.18)</td>
<td>2.49 (0.15)</td>
<td>$F(1, 89) = 1.65, \ p = .203$</td>
</tr>
<tr>
<td>Word Frequency*</td>
<td>42.40 (9.60)</td>
<td>16.31 (6.05)</td>
<td>$F(1, 89) = 5.28, \ p = .024$</td>
</tr>
<tr>
<td>Likeableness Rating***</td>
<td>5.15 (0.03)</td>
<td>1.69 (0.03)</td>
<td>$F(1, 89) = 5292.89, \ p &lt; .001$</td>
</tr>
<tr>
<td>Familiarity Rating***</td>
<td>5.82 (0.01)</td>
<td>5.64 (0.03)</td>
<td>$F(1, 89) = 27.83, \ p &lt; .001$</td>
</tr>
<tr>
<td>Word Length</td>
<td>8.78 (0.33)</td>
<td>8.29 (0.39)</td>
<td>$F(1, 89) = 091, \ p = .344$</td>
</tr>
<tr>
<td>Number of Syllables</td>
<td>2.87 (0.13)</td>
<td>2.89 (0.18)</td>
<td>$F(1, 89) = 010, \ p = .921$</td>
</tr>
<tr>
<td>Word Frequency***</td>
<td>48.51 (15.04)</td>
<td>16.31 (6.05)</td>
<td>$F(1, 89) = 11.59, \ p = .001$</td>
</tr>
</tbody>
</table>

*Note.* * denotes $p < .01$; ** denotes $p < .001$, *** denotes $p < .001$. Frequency is per 10000 words. Data based on Dumas et al. (2002).

**EEG recording apparatus.** A 32-channel system using Ag/AgCl electrode Quick-Cap (Neurosoft., Sterling, Virgins) with an unlinked mastoid (M1) was used for recording the ERPs. The experiment was conducted in a Faraday cage (electrically shielded, sound attenuated chamber) located in Griffith University’s School of Applied Psychology cognitive psychology laboratory. The Faraday cage was fitted with an intercom and video monitoring system. A lounge chair was
positioned 100 cm from a 43.2 cm computer monitor. The computer ran Windows™ XP operating system and Presentation™ software (Neurobehavioural Systems, available at http://www.neurobs.com/). It included a response keypad with one key labelled ‘Yes’ and another key labelled ‘No’. Another computer running Windows™ XP and Scan 4.3.1. Acquire and Edit Neurocan™ was also used in the investigation.

3.2.3. Procedures

3.2.3.1. EEG recording and data reduction. Each participant was tested individually. Event-related brain potentials (ERPs) were acquired using a lycra stretchable Quick-Cap (Neurosoft., Sterling, Virgins) with 32 Ag/AgCl electrodes with an unlinked mastoid (M1). Electrode placement corresponded to the following 28 sites of the International 10-20 Electrode Positioning System: FP1, FP2, F3, F4, F7, F8, Fz, FT7, FT8, FC3, FC4, C3, C4, CZ, CP3, CP4, T7, T8, TP7, TP8, P3, P4, P7, P8, Pz, O1, O2, Oz. Electrooculogram (EOG) data were recorded using Ag/AgCl drop electrodes placed lateral to the outer canthi and at the left supraorbital and suborbital positions. All impedances were kept below 5kΩ, which was assessed before and following the EEG recording. Once the electrode cap was attached, participants were seated in a comfortable lounge chair in the air-conditioned, dimly lit, electrically shielded, and sound-attenuated faraday cage.

Participants’ EEG and EOG data were recorded continuously and sampled at 1000 Hz. High- and low-pass analogue filter settings were set at 0.01 Hz and 100 Hz, respectively. Data was referenced to the left mastoid (M1) and algebraically re-referenced to average mastoids off-line ([M1+M2]/2; Miller, Lutzenberger, & Elbert, 1991). In an attempt to reduce fatigue effects and circadian rhythm effects on the ERP data all ERP testing was conducted between 10.00 AM and 3.00 PM (Luck, 2005). To equalize cortical activation due to the motor activation required for task
responding, participants completed the first half of each of the three ERP tasks with one hand and the second with the other.

All EEGs were visually inspected for muscle artifact and submitted to a regression based blink correction method using the Neuroscan™ Edit 4.3.2 Software package (Semlitsch, Anderer, Schuster, & Presslich, 1986). To derive the ERPs, the continuous file was epoched from 200 ms pre-stimulus to 2000 ms post stimulus. Epochs were baseline corrected using the pre-stimulus interval. Individual trails exceeding +/- 80 µV were automatically rejected. Data were subsequently subjected to visual inspection to remove remaining trials with EOG and EMG artefact (which constituted less than 5% of the data).

3.2.3.2. Experimental task. To assess ERPs for encoding processes, participants viewed a Bernoulli presentation of 45 positive and 45 negative adjectives on the computer monitor. Presentation™ software (Neurobehavioural Systems Incorporated, http://www.neurobs.com) was used to deliver the experimental stimuli. Each word was presented separately and only once during the task. Prior to each word, a small white dot appeared on the computer screen for 1500 ms, it was used to cue participants that a new stimulus trial was about to begin. A word was then presented for 300 ms, after which the screen went black for 2200 ms. Thus, an ISI of 4000 ms was employed, of which 2500 ms was used to record the ERPs. During reading college students are known to fixate on words for about 200 to 250 ms before saccades of around 20 to 30 ms bring the next string to the fovea. The ERP is sensitive to ocular artifacts. Because the eyeball acts as a dipole, eye movements can create changes in scalp voltage that supersede activity related to word processing (Luck, 2005). Consequently, stimuli were selected to be presented in a way that renders eye movements unnecessary and likely improbable. That is, one word was presented at a
time, in the centre of the screen, for the shortest duration deemed necessary (300 ms). The 2200 ms inter-stimulus interval was selected to allow ample time for relevant long latency ERP components, such as the N400, P3b, and LPP, to peak and subside prior to the next stimulus presentation. The stimulus presentation rate replicates previous ERPs studies in the area. For instance, in their encoding task Kayser et al. (2003) used a 400 ms presentation rate for word stimuli. In their Old/new memory task Dietrich et al. (2000) used a 300 ms presentation for word stimuli. Chiu et al. (2008) used a 280 ms presentation rate for word stimuli during their emotional Go/Nogo task. The stimulus presentation rate also decreased testing time, which was important to reduce the impact of participant fatigue across the ERP testing.

As soon as the word was presented, participants were required to indicate, as fast as they could, whether or not they thought the adjective was self-descriptive by pressing the ‘Yes’ or ‘No’ key. The white cue screen then reappeared, beginning a new stimulus trial. This emphasis on fast behavioural responding was to ensure participant attention and activation of bottom-up schema processing in the MDD sample (Beck, 1967). Self-relevant stimuli offer the most sensitive test of cognitive biases in depression (see Wisco, 2009 and Section 2.3.1. for a review). Thus, self-descriptive encoding of stimuli were utilised to elicit deeper level of information processing. This ensured internal validity of the later assessed memory biases (Study 2) and inhibitory deficits (Study 3) in the dissertation studies. The instructions for the task were visually presented to participants via the computer screen and verbally explained by the PhD candidate to ensure understanding. They are shown in Appendix K. Figure 18 presents a visual illustration of the task. Dependent variables were frequencies of self-attributed positive or negative adjectives, reaction time (RT) data, and P2, P3a, P3b, and LPP ERPs.
Figure 18. Schematic depiction of the self-descriptive encoding task.

After completion of the self-descriptive encoding task, participants commenced with completing the behavioural (free recall) and ERP tasks for Study 2 (Chapter 4) and Study 3 (Chapter 5). The EEG cap was then removed. Participants were then seated in a private room and asked to complete the BDI-II, STAI-S, RRS, and the ERQ self-report questionnaires. The psychometric questionnaires were completed after the ERP testing to avoid any mood priming effects. The procedural steps and average timings for the electrophysiological testing are presented in Table 7.
Table 7

*Procedural Steps and Average Timings for the Cognitive and ERP Testing*

<table>
<thead>
<tr>
<th>Procedural Steps</th>
<th>Average Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG preparation (electrode placement, impedance reduction)</td>
<td>30 minutes</td>
</tr>
<tr>
<td>Study 1: Self-descriptive encoding task</td>
<td>10 minutes</td>
</tr>
<tr>
<td>Study 2: Behavioural free recall task</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Study 2: Old/New task</td>
<td>15 minutes</td>
</tr>
<tr>
<td>Study 3: Modified Sternberg working memory task</td>
<td>20 minutes</td>
</tr>
<tr>
<td>Removing EEG cap and completion of the self-report questionnaires</td>
<td>40 minutes</td>
</tr>
<tr>
<td></td>
<td><strong>Total:</strong> 120 minutes</td>
</tr>
</tbody>
</table>

3.2.3.3. Subjective ratings of mood and fatigue across the studies.

3.2.3.3.1. Mood ratings. A 3 (group) x 3 (study) factorial ANOVA was conducted to examine if participants’ subjective ratings of mood (9-point scale: *positive* to *depressed*) remained stable over the three ERP studies. No interaction was found between the variables. The main effect of study, $F(1.58, 110.23) = 12.65, p < .001, \eta^2 = .153$, and group, $F(2, 70) = 13.75, p < .001, \eta^2 = .282$, were significant. As shown in Figure 19, the MDD group showed overall greater levels of negative mood compared to the control and remitted groups. This slightly increased over the three ERP studies.
Figure 19. Participants’ subjective mood ratings across the three electrophysiological studies (1 = positive; 9 = depressed). Error bars refer to standard error of the mean.

For the main effect of study, Bonferroni corrected post hoc analysis ($\alpha/3$ tests = .017) found that participants reported a significant increase in depressed mood ratings from Study 1 to Study 3, $F(1, 70) = 19.99, p < .001, \eta^2 = .222$, and from Study 1 and 2, $F(2, 70) = 8.26, p = .005, \eta^2 = .106$. No differences were found between Study 2 and 3, $F(2, 70) = 5.81, p = .019, \eta^2 = .077$. For the main effect of group, Bonferroni post hoc analysis found that MDD participants reported significantly greater negative mood ratings than both the control (mean difference = 1.75, $p < .001$) and remitted participants (mean difference = 1.81, $p = .001$). No differences in mood ratings existed between control and remitted participants (mean difference = 0.7).

3.2.3.3.2. Fatigue ratings. A 3 (group) x 3 (study) factorial ANOVA was conducted to examine if participants’ subjective ratings of fatigue (9 -point scale: energetic to exhausted) remained stable over the three ERP studies. No interaction
was found between the variables. The main effect of study, $F(2, 140) = 25.49, p < .001, \eta^2 = .267$, and group, $F(2, 70) = 8.79, p < .001, \eta^2 = .201$ were significant. As shown in Figure 21 the MDD group reported greater levels of fatigue compared the control and remitted groups. There was a slight increase in fatigue levels between study 2 and 3 across all groups. Figure 20 presents these results.

Electrophysiological Study

*Figure 20.* Participants’ subjective fatigue ratings across the three electrophysiological studies (1 = energetic; 9 = exhausted). Error bars refer to standard error of the mean.

For the main effect of study, Bonferroni post hoc analysis ($\alpha/3$ tests = .017) found participants reported a significant increase in fatigue ratings from Study 1 to Study 3, $F(1, 70) = 32.78, p < .001, \eta^2 = .319$, and from Study 2 to Study 3, $F(1, 70) = 35.96, p < .001, \eta^2 = .339$. No differences existed between the Study 1 and Study 2. For the main effect of group, Bonferroni post hoc analysis found that MDD participants reported significantly higher fatigue ratings than control participants.
(mean difference = 1.23, \( p =.001 \)) and remitted participants (mean difference = 1.42, \( p =.004 \)). No differences in fatigue ratings existed between control and remitted participants (mean difference = 0.19, \( p > .999 \)).

3.2.3.4. Follow-up depression and anxiety assessment. Approximately 30 days following the completion of the electrophysiological testing (mean time lag = 31.64, \( SD = 3.55 \)), participants were re-contacted by the experimenter and re-administered the BDI-II and STAI-S questionnaires. Seventy-one of the original 73 participants completed the follow-up questionnaires (97.26% retention rate). Participants were thanked, credited, and debriefed on the study. The high month test-retest scores on the BDI-II, \( r (70) = .903, p <.001 \), and STAI-State, \( r (70) = .767, p <.001 \), suggested stability of depressive and state anxiety symptoms in the sample.

3.2.3.5. Statistical methodology. The first stage of analysis was to examine the collected data, checking for outliers and ascertaining whether assumptions were met for the conducted statistical tests. An alpha level set at .05 was used for all statistical tests (ANOVAs or Pearson’s correlation). In the interest of brevity, only ERP findings that included the research variable Group are presented. The exact significant \( p \) values provided by SPSS analyses will be reported, except in the case where SPSS printed .000 as the significance, in which case \( p <.001 \) is reported (APA, 2008). In line with Field (2005) guidelines, if Mauchly’s test of sphericity was found to be non-significant, the SPSS sphericity assumed ANOVA results are reported. If sphericity was significant however, the Greenhouse-Geisser epsilon value was assessed. When the epsilon value was smaller than 0.75, the Greenhouse-Geisser corrected ANOVA results were reported. In cases when the epsilon value was larger than 0.75, the Huynh-Feldt corrected ANOVA results were reported. In line with APA publication guidelines (2007), all measures are accompanied by the strength of
the relationships (partial eta squared: $\eta^2$). Given that Type I error is known to be inflated across multiple hypothesis testing, such as that used in factorial ANOVAs, it thus needs to be managed carefully (see Schochet, 2008 for a review). In an attempt to achieve this, all dissertation analyses were directed by their respective priori hypotheses. In line with recommended procedures (Keppel, 1991; Tabachnick & Fidell, 1996), the omnibus $F$ statistic was skipped, with a signal-$df$ planned comparison between group means conducted instead. This procedure allows more statistical power to be apportioned to the planned comparisons, and not ‘wasted’ on the omnibus $F$, which is unable to identify specific differences between means. Bonferroni correction was used to test the predictions of planned simple main effects. The Bonferroni method is recommended in a factor ANOVA design to control the Type I error rate, especially in three-way and four-way designs (e.g., Fletcher, Daw, & Young, 1989; Rosenthal & Rubin, 1984; Smith, Levine, Lachlan, & Fediuk, 2002; Stevens, 2002). Given the smaller size and heterogeneous nature of the remitted-depressed sample, assumptions of normality (skewness and kurtosis) are reported for their data (see Appendix L).

3.3. Results

3.3.1. Participant Characteristics

Table 8 displays participant group means and variability on the self-reported psychometrics. Univariate ANOVAs with subsequent planned comparisons found the three groups differed predictably on the self-report measures of depression, state anxiety, use of emotional reappraisal, and rumination. As can be seen the average BDI-II scores at testing and follow-up for the MDD group were in the severe range (scores > 29), while both the remitted and control groups scored in the minimal range (scores < 13). Of the MDD group, 3.3% (1 participant) scored in the minimum range,
13.3% scored in the mild range, 33.3% scored in the moderate range and 50% scored in the severe range on the BDI-II at testing. At follow-up, 3.3% (1 participant) scored in the minimum range, 23.3% scored in the mild range, 16.7% scored in the moderate range and 50% scored in the severe range on the BDI-II. Of the remitted depressed group, 69.2% scored in the minimum range, 23.1% scored in the mild range, 0% scored in the moderate range and 7.7% (1 participant) scored in the severe range on the BDI-II at testing. At follow-up, 84.6% scored in the minimum range, 7.7% scored in the mild range, 7.7% scored in the moderate range and 0% scored in the severe range on the BDI-II. Of the control group, 90% scored in the minimum range, 10% scored in the mild range, 0% scored in the moderate range and 0% scored in the severe range on the BDI-II at testing and follow-up. The control and remitted groups differed on use of suppression (ERQ subscale), with the remitted participants showing smaller scores.

Table 8

*Group Means (SD in parenthesis) for the Self-Report Psychometric Questionnaires*

<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
<th>Test of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression (BDI-II)</td>
<td>5.50 (5.08)</td>
<td>10.54 (8.05)</td>
<td>29.90 (9.86)</td>
<td>$F(2,70) = 76.34, p &lt; .001, \eta^2 = .686$</td>
</tr>
<tr>
<td>&quot;Minimal Depression&quot;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td>&quot;Minimal Depression&quot;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td>&quot;Severe Depression&quot;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>No difference existed between control and remitted ($p = .175$)</td>
</tr>
</tbody>
</table>

<p>| Depression Follow-up (BDI-II-FU) | 3.86 (4.87) | 8.54 (6.68) | 29.32 (11.19) | $F(2, 67) = 72.24, p &lt; .001, \eta^2 = .683$ |
| &quot;Minimal Depression&quot; | - | - | - | MDD &gt; control ($p &lt; .001$) |
| &quot;Minimal Depression&quot; | - | - | - | MDD &gt; remitted ($p &lt; .001$) |
| &quot;Severe Depression&quot; | - | - | - | No difference existed between control and remitted ($p = .285$) |</p>
<table>
<thead>
<tr>
<th>Questionnaire</th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
<th>Test of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rummation</td>
<td>35.40 (11.40)</td>
<td>42.54 (14.51)</td>
<td>61.637 (10.11)</td>
<td>$F(2, 70) = 39.62, p &lt; .001, \eta^2 = .531$</td>
</tr>
<tr>
<td>(RRS-Total)</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p = .016$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .197$)</td>
</tr>
<tr>
<td>Reflective</td>
<td>7.73 (3.37)</td>
<td>9.69 (4.46)</td>
<td>13.00 (2.95)</td>
<td>$F(2, 70) = 17.53, p &lt; .001, \eta^2 = .334$</td>
</tr>
<tr>
<td>Rummation</td>
<td>(RRS-R)</td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p = .016$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .279$)</td>
</tr>
<tr>
<td>Brooding</td>
<td>7.83 (2.90)</td>
<td>9.15 (3.13)</td>
<td>14.00 (3.54)</td>
<td>$F(2, 70) = 29.11, p &lt; .001, \eta^2 = .454$</td>
</tr>
<tr>
<td>Rummation</td>
<td>(RRS-B)</td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .663$)</td>
</tr>
<tr>
<td>Depressive</td>
<td>19.60 (6.21)</td>
<td>23.69 (8.69)</td>
<td>34.37 (6.97)</td>
<td>$F(2, 70) = 34.49, p &lt; .001, \eta^2 = .496$</td>
</tr>
<tr>
<td>Rummation</td>
<td>(RRS-D)</td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .249$)</td>
</tr>
<tr>
<td>Emotional</td>
<td>31.33 (5.17)</td>
<td>32.54 (6.39)</td>
<td>24.13 (8.16)</td>
<td>$F(2, 70) = 11.21, p &lt; .001, \eta^2 = .243$</td>
</tr>
<tr>
<td>Reappraisal</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &lt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td>(ERQ-R)</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &lt; remitted ($p = .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .999$)</td>
</tr>
<tr>
<td>Emotional</td>
<td>13.90 (4.28)</td>
<td>10.15 (5.10)</td>
<td>16.47 (3.51)</td>
<td>$F(2, 70) = 9.42, p &lt; .001, \eta^2 = .212$</td>
</tr>
<tr>
<td>Suppression</td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between MDD and control ($p = .094$)</td>
</tr>
<tr>
<td>(ERQ-S)</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- Control &gt; remitted ($p = .035$)</td>
</tr>
<tr>
<td>State Anxiety</td>
<td>29.00 (7.84)</td>
<td>32.92 (12.12)</td>
<td>50.97 (11.90)</td>
<td>$F(2, 70) = 35.53, p &lt; .001, \eta^2 = .504$</td>
</tr>
<tr>
<td>(STAI-S)</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .787$)</td>
</tr>
<tr>
<td>State Anxiety</td>
<td>31.24 (11.03)</td>
<td>38.46 (14.57)</td>
<td>57.39 (12.71)</td>
<td>$F(2, 67) = 32.73, p &lt; .001, \eta^2 = .494$</td>
</tr>
<tr>
<td>Follow Up</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; control ($p &lt; .001$)</td>
</tr>
<tr>
<td>(STAI-S-FU)</td>
<td></td>
<td></td>
<td></td>
<td>- MDD &gt; remitted ($p &lt; .001$)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No difference existed between control and remitted ($p = .258$)</td>
</tr>
</tbody>
</table>

**Note.** BDI-II = Beck Depression Inventory-Second Edition, STAI-S = State Trait Anxiety Inventory – State subscale. ERQ = Emotion Regulation Questionnaire for Reappraisal (ERQ-R) and Suppression (ERQ-S) subscales. RRS = Rumination Response Scale for Reflective (RRS-R), Brooding (RRS-B), Depressive (RRS-D) subscales, and Total (RRS-Total). Follow-up duration is one month. All group comparisons are based on planned analyses.
3.3.2. Behavioural Data

To reduce clutter and inflation of Type I errors, only results that pertain to the research hypotheses or to the key variable group are presented.

Self-descriptive word endorsement. Figure 21 displays the percentage of positive and negative word stimuli endorsed as self-descriptive during the self-referential encoding task for the three experimental groups. As can be seen, across all participants endorsed more positive stimuli as self-descriptive compared to the negative stimuli. To test the predictions of Hypothesis 1, a 3 (group) × 2 (valence) factorial ANOVA was conducted. It found a significant Group×Valence interaction $F(2, 70) = 19.14, p < .001, \eta^2 = .354$, a main effect for valence, $F(2, 70) = 418.26, p < .001, \eta^2 = .857$, and null result for group. To analyse the interaction, the impact of group was analysed using separate univariate ANOVAs for the positive and negative stimuli. Simple main effects were found for both the positive, $F(2, 70) = 9.98, p < .001, \eta^2 = .222$, and negative stimuli, $F(2, 70) = 16.96, p < .001, \eta^2 = .326$. For positive stimuli, planned comparisons ($\alpha = .05$) showed control participants endorsed significantly more words as self-descriptive compared to MDD ($mean$ difference = 18.22%, $p < .001$). Remitted participants also endorsed more positive words as self-descriptive compared to the MDD participants ($mean$ difference = 17.08%, $p = .010$). No differences existed between the control and remitted participants. For the negative stimuli, MDD participants endorsed significantly more words as self-descriptive compared to both the control ($mean$ difference = 17.48%, $p < .001$) and remitted participants ($mean$ difference = 12.29%, $p = .007$), who in turn did not differ from each other.
Figure 21. Mean percentage of positive and negative words endorsed as self-descriptive by the control, remitted, and MDD participants during the self-referential encoding task. *** $p < .001$. Error bars refer to SEM.

**Reaction time data.** Table 9 outlines participants’ average RTs for endorsed and rejected positive and negative stimuli during the self-referential encoding task. Where participants did not endorse any negative stimuli as self-descriptive (as occurred for five participants in the Control group and one participant in the Remitted group) their data was substituted with their respective group mean for this variable (Tabachnick & Fidell, 2007). As can be seen, all participants responded quite quickly to the self-descriptive judgments for the positive and negative stimuli (all within 1.5 seconds). A 3 (group) × 2 (endorsement) × 2 (valence) factorial ANOVA found no significant group differences or interactions.
Table 9

Means (SD in parentheses) for Control, Remitted, and MDD participants Reaction Times (ms) for Positive and Negative stimuli during Self-Referential Encoding

<table>
<thead>
<tr>
<th>Endorsement</th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>938.68 (208.25)</td>
<td>1029.33 (201.49)</td>
<td>1105.00 (235.36)</td>
</tr>
<tr>
<td>Negative</td>
<td>1150.19 (329.78)</td>
<td>1226.71 (301.96)</td>
<td>1235.60 (253.62)</td>
</tr>
<tr>
<td>Not Descriptive</td>
<td>Positive</td>
<td>1142.60 (377.62)</td>
<td>1250.79 (301.00)</td>
</tr>
<tr>
<td>Negative</td>
<td>965.02 (200.61)</td>
<td>1072.91 (223.64)</td>
<td>1131.94 (235.02)</td>
</tr>
</tbody>
</table>

3.3.3. Electrophysiological Data

3.3.3.1. Preliminary Analyses to Identify Relevant ERP Components

Of the original 32 electrode sites, nine were further analysed (F₃, F₇, F₄, C₃, Cz, C₄, P₃, Pz, P₄) to ascertain effects of caudality (frontal, central, and parietal) and laterality (left, central, right). A restricted number of electrodes were used in the current analyses to reduce the threat of family-wise error associated with multiple planned comparisons (Keppel, 1991; Tabachnick & Fidell, 2007).

To isolate the primarily ERP components, two analytic techniques were used. First, to examine the typically robust ERP components (e.g., P2, N2, P3; Luck, 2005), traditional windowed analysis were conducted on individual average files. Guided by theories and prior studies, these a priori time windows (outlined in Table 1 and then verified by the results of the pilot studies: Appendix B-D) were inspected for each hypothesised ERP component. If inspection of the waveforms indicated that these commonly identified temporal windows suitably captured the desired ERP component, grand average ERP waveforms were then submitted to a PCA to further confirm the validity of the epoch. The PCA selects activity with similar topography.
across time points (generally accepted as defining an ERP component). Covariance input matrix, followed by a promax rotation with Kaiser Normalization procedures were selected to retain the original individual component morphology. A criterion of eigen values > 1 was used to extract the higher-value orthogonal factors that explain most of the component and noise variance, as reported in Kayser et al. (1999). The PCA analysis was completed for the experimental groups separately given that ERP differences are expected between the groups.

**Control Group.** The PCA for the ERP data consisted of rows of participants \((n = 30)\), the two experimental conditions (positive and negative stimuli), and nine electrode sites \((F_3, F_Z, F_4, C_3, C_Z, C_4, P_3, P_Z, P_4)\). Data was originally recorded at 1000 points per second (Hz). To conform to PCA analysis assumptions, the original data was reduced to represent a sampling rate of 200 points per second, which reduced the number of variables (columns) from 2000 to 400 by averaging every five sequential data points (across 2000 ms). That is, the matrix included a total of \(30 \times 2 \times 9\) rows existed for the 400 columns of time samples of EEG. The PCA for the attention task data indicated that six components should be retained, and these accounted for 78.52% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated with these components are shown below in Appendix M. In temporal sequence, the approximate peak latency of loadings for

---

3 It is noted that to improve the stability and robustness of the ERP data it would have been optimal to conduct the PCA on better clusters of electrodes—e.g., across caudal positions: frontal \((F_3, F_Z, F_4)\), central \((C_3, C_Z, C_4)\), and parietal \((P_3, P_Z, P_4)\). Unfortunately, when the data file is split in this manner, PCA assumptions for greater rows than columns is no longer met when retaining an adequate number of cases \(>(300)\). Tabachnick and Fidell argue that “if different samples do produce the same factors, pooling them is desirable because of increase in sample size” (2005, pg. 612). The ERPs for the caudal (frontal, central, and parietal) and lateral (left, right, and midline) electrodes significantly correlate with each other for all Groups for each of the three studies (see Appendix Q: SPSS output files). Thus, it is argued to be appropriate to perform the PCA analysis of the electrodes sites together to improve the strength of the extracted results.
each component was: Component 6, 200 ms; Component 4, 370 ms; Component 5, 450 ms; Component 1, 600 ms; Component 3, 1000 ms; Component 2, 1655 ms.

An inspection of both the component loadings and the grand average ERP waveforms indicated that all components primarily represented positive peaks, with component 3 also appearing to show a negative peak at approximately 400 ms. The latency and morphology of Component 6, the combined window of Component 4 and 5, and the combined window of Components 1 and 3 appeared to be consistent with a priori epochs and grand average peaks for the P2, P3a, and P3b, respectively (Shestyuk & Deldin, 2010; Polich, 2003, 2007). Component 2 (20.37% explained variance) appeared to show a consistent late positive morphology. This sustained positivity appears similar to the LPP (Anokhin et al., 2006; Schupp, Junghofer, Weike, & Hamm, 2004). It was not evident in the grand average waveforms. This is likely due to the small magnitude of this waveform (approximately 2µV) compared to the other extracted components.

Thus, for the Control group, the positive peak within the 150-350 ms temporal window was deemed an appropriate epoch to measure the P2; 351-500 ms window to measure the P3a, the 501-800 ms window to measure the P3b, and the 801-2000 ms window to measure the LPP. The six extracted components and the selected epochs for each of the above ERPs are shown in Figure 22.
Component 6
Peak latency: 200 ms
3.60% explained variance

Component 4
Peak latency: 370 ms
7.20% explained variance

Component 5
Peak latency: 450 ms
4.19% explained variance

Component 1
Peak latency: 600 ms
33.23% explained variance

Component 3
Peak latency: 1000 ms
14.23% explained variance

Component 2
Peak latency: 1655 ms
16.05% explained variance

Figure 22. ERP epochs for the control group for study 1.
Remitted Group. The PCA for the ERP data consisted of rows of participants \( n = 13 \), the two experimental conditions (positive and negative stimuli), and nine electrode sites \( (F_3, F_Z, F_4, C_3, C_Z, C_4, P_3, P_Z, P_4) \). Data was originally recorded at 1000 points per second (Hz). To conform to PCA analysis assumptions the original data was reduced to represent a sampling rate of 400 points per second, reducing the number of variables (columns) from 2000 to 200 by averaging every 10 sequential data points (across 2000 ms). That is the matrix included a total of \( 13 \times 2 \times 9 \) rows for the 200 columns of time samples of EEG\(^4\). Given the smaller number of cases in this analysis, care is specifically taken to only analyse extracted components whose morphology is consistent with hypothesized epochs for robust ERPs (e.g., P2, P3, LPP); thus, maintaining the statistical integrity of the analysis (see Bentler, 2000; de Winter, Dodou, & Wieringa, 2009; Spanas & Zeller, 2002; Zeller, 2005).

The PCA for the attention task data indicated that five components should be retained, and these accounted for 77.07% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated with these components are shown in Appendix N. Components 1, 4, and 5 primarily represented positive peaks. Components 2 and 3 represented both positive and negative peaks. In temporal sequence, the approximate peak latency of loadings for each component was: Component 5, 200 ms; Component 4, 300 ms; Component 1, 600 ms; Component 3, 1000 ms, and Component 2, 1600 ms. The latency and morphology of Components 5, 4, and 1 appear consistent with a priori epochs and grand average peaks for the P2.

\(^4\) Given the smaller sample of remitted participants, for the PCA matrix to be valid, the original data for these the remitted sample was reduced to 200 EEG samples. This is smaller than the adequate number (300) recommended by Tabachnick and Fidell (2005). Thus, care is specifically taken to only analyse extracted components whose morphology is consistent with hypothesized epochs for robust ERPs (e.g., P2, P3, LPP.). When more stringent extraction protocols are followed like this previous work has shown that smaller PCA samples will continue to produce statically valid results (Bentler, 2000; de Winter, Dodou, & Wieringa, 2009; Spanas & Zeller, 2002; Zeller, 2005).
P3a, and P3b, respectively (Shestyuk & Deldin, 2010; Polich, 2003, 2007).

Component 2 and 3 appeared to show a consistently late positive morphology, likely representing the LPP (Anokhin et al., 2006; Schupp, Junghofer, Weike, & Hamm, 2004). The negative peaks in Components 2 seemed to represent inverted P3bs, which is not evident in the grand average waveforms likely due to their small magnitude.

Thus, for the Remitted group, the positive peak within the 150-350 ms temporal window was deemed an appropriate epoch to measure the P2; 351-500 ms window to measure the P3a, the 501-800 ms window to measure the P3b, and the 801-2000 ms window to measure the LPP. The five extracted components and the selected epochs for each of the above identified ERPs are shown in Figure 23. This conformed to the windows used to extract these ERPs for the Control participants.
Component 5  
**Peak latency:** 200 ms  
5.92% explained variance

Component 4  
**Peak latency:** 300 ms  
7.73% explained variance

Component 1  
**Peak latency:** 600 ms  
28.78% explained variance

Component 3  
**Positive peak latency:** 1000 ms  
**Negative peak latency:** 600 ms  
8.24% explained variance

Component 2  
**Positive peak latency:** 1600 ms  
**Negative peak latency:** 1000 ms  
15.67% explained variance

**Figure 23.** ERP epochs for the remitted group for study 1.
**Major Depression Disorder Group.** The PCA for the ERP data consisted of rows of participants \((n = 30)\), the two experimental conditions (positive and negative stimuli), and nine electrode sites \((F_3, F_Z, F_4, C_3, C_Z, C_4, P_3, P_Z, P_4)\). Thus, the matrix included a total of \(30 \times 2 \times 9\) rows existed for the 400 columns of time samples of EEG. The PCA for the attention task data indicated that six components should be retained, and these accounted for 74.15% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated these components are shown below in Appendix O. Component 1 represented both positive and negative peaks. In temporal sequence, the approximate peak latency of loadings for each component was: Component 6, 200 ms; Component 3, 370 ms; Component 5, 400 ms; Component 2, 600 ms; Component 4, 600 ms; Component 1, 900ms.

The latency and positive peak morphology of Components 6, 3, and 1 were consistent with a priori epochs and grand average peaks for the P2, P3a, and P3b, respectively. Components 1 and 5 showed a negative between 200-600 ms, which is consistent with the N400 (Kutas & Hilyard, 1980, 1982). This negative-going ERP is evident in the 200-500ms epoch of the MDD group’s grand average waveforms at frontal and central sites. Last, Component 1, 2 and 3 appeared to show a consistent late positive morphology, likely representing the LPP. Thus, based on the PCA results and previous research the following epochs were used: 150 -350 ms to measure the P2; 351-500 ms to measure the P3a, 501-800 ms to measure the P3b\(^5\), and 801-2000 ms to measure the LPP. The negative peak within the 200 – 600 ms temporal window was deemed appropriate to measure the N400. The six extracted components and the selected epochs for each of the above identified ERPs are shown in Figure 24.

\(^5\) Although a later latency epoch is suggested from above the PCA results to measure the P3b, it was determined more internally and externally valid to restrict the epoch to that utilized to measure the Control and Remitted participants’ P3b and that used in previous research (e.g., Ilardi et al., 2007).
Component 1
Negative peak latency: 500 ms
Positive peak latency: 900 ms
29.23% explained variance

Component 2
Peak latency: 600 ms
13.09% explained variance

Component 3
Peak latency: 370 ms
9.97% explained variance

Component 4
Peak latency: 600 ms
5.12% explained variance

Component 5
Peak latency: 450 ms
3.79% explained variance

Component 6
Peak latency: 200 ms
3.66% explained variance

Figure 24. ERP epochs for the MDD group for study 1.
3.3.3.2. ERP Results

Control, remitted, and MDD participants’ grand averaged ERPs to positive and negative stimuli evoked during the self-referential encoding tasks used in Study 1 are shown in Figure 25, 26, and 27 respectively. Visual inspection suggests that control participants’ positive stimuli appeared to show greater positivity than the negative stimuli during the P3b epoch, while greater negative magnitude is apparent for the negative stimuli compared to the positive stimuli during the N400 epoch. Both these effects appear maximum at central and parietal sites. The grand average waveforms were visibly noiser for the remitted-depressed sample as compared to the control and MDD groups. Across all caudal and lateral positions, the remitted-depressed participants appeared to show enhanced positive magnitude for negative than positive stimuli. MDD participants’ grand average data suggested a slight increased positivity for the positive relative to the negative stimuli during the P3b epoch across right lateral electrode sites. Frontal and central sites appear to show greater negative-going waves for negative than positive stimuli during the N400 epoch.
Figure 25. Grand average ERPs for positive (black line) and negative (blue line) stimuli, averaged over participant endorsement for control participants during the self-referential encoding task. Stimulus onset 200ms (dotted line).
**Figure 26.** Grand average ERPs for positive (black line) and negative (blue line) stimuli, averaged over participant endorsement for remitted participants during the self-referential encoding task. Stimulus onset 200ms (dotted line).
Figure 27. Grand average ERPs for positive (black line) and negative (blue line) stimuli, averaged over participant endorsement for MDD participants during the self-referential encoding task. Stimulus onset 200ms (dotted line).
**P2 (200 – 350 ms).** To evaluate Hypothesis 3, peak average P2 amplitudes were subjected to a 3 (group) × 3 (caudality) × 3 (laterality) × 2 (valence) factorial ANOVA. Results, as shown in Table 10, found the three-way interaction to be the only significant effect.

**Table 10**

**P2 Factorial ANOVA Results: Study 1**

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 0.03, p = .970, \eta^2 = .001$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Group</td>
<td>$F(2.46, 85.95) = 0.13, p = .915, \eta^2 = .004$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality×Group</td>
<td>$F(4, 140) = 1.36, p = .248, \eta^2 = .038$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 70) = 0.85, p = .431, \eta^2 = .024$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Laterality×Group</td>
<td>$F(5.79, 1.39) = 1.67, p = .476, \eta^2 = .013$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence×Group</td>
<td>$F(2.79, 97.91) = 2.45, p = .036, \eta^2 = .066$</td>
<td>*</td>
</tr>
<tr>
<td>Laterality×Valence×Group</td>
<td>$F(3.33, 116.36) = 1.07, p = .368, \eta^2 = .030$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Laterality×Valence×Group</td>
<td>$F(5.54, 194.05) = 1.27, p = .275, \eta^2 = .035$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05$, * $p < .05$.

The three-way interaction was the focus of further analysis. A caudality × valence ANOVAs was conducted for each group separately. The results of these sub-analyses are shown in Table 11. As can be seen, the MDD group showed a simple main effect
of caudality, with larger P2 evoked over parietal than central or frontal sites. The control group also showed a significant Caudality×Valence interaction.

Table 11

*Breakdown of P2 Caudality×Valence×Group Interaction: Study 1*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.27, 36.84) = 3.61, p = .056, \eta^2 = .111$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 0.96, p = .334, \eta^2 = .032$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence</td>
<td>$F(1.39, 40.52) = 6.12, p = .010, \eta^2 = .174$</td>
<td>**</td>
</tr>
<tr>
<td>Remitted Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.16, 13.93) = 2.21, p = .158, \eta^2 = .156$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 12) = 0.07, p = .802, \eta^2 = .005$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence</td>
<td>$F(1.34, 16.11) = 0.69, p = .461, \eta^2 = .054$</td>
<td>ns</td>
</tr>
<tr>
<td>MDD Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.93, 34.58) = 3.95, p = .048, \eta^2 = .120$</td>
<td>*</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 0.68, p = .416, \eta^2 = .023$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence</td>
<td>$F(1.41, 40.93) = 0.87, p = .392, \eta^2 = .029$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05$, *$p < .05$, **$p < .01$, ***$p < .001$.

Planned univariate ANOVAs for the impact of valence was conducted for each caudal position ($\alpha = 0.05$). Control participants were found to exhibit significantly larger P2s to negative compared to positive words over frontal sites, $F(1, 29) = 5.28, p = .029, \eta^2 = .154$ (mean difference = 0.79µV, SEM = 0.345). No differences were found over central or parietal sites. Control participants P2 results are presented in Figure 28.
Figure 28. Control participants’ grand average P2 ERPs over frontal, central, and parietal sites for positive and negative stimuli during the self-referential encoding task. * = p < .05, error bars refer to standard error of the mean.

**P3a (351-500ms).** Null results were expected for the P3a data. Thus, the results of the auxiliary ANOVA are outlined in Appendix P. As expected, no significant effects involving the key variable group were found.

**P3b (501-800ms).** To analyse the predictions of hypothesis 2(a), peak P3b amplitudes were subjected to a 3 (group) × 3 (laterality) × 2 (valence) factorial ANOVA. As can be seen in Table 12, the predicted three-way interaction was significant. No other effects were observed. To analyse the three-way interaction, 3 (laterality) × 2 (valence) ANOVAs were conducted on P3b data for each group separately. As shown in Table 13, the MDD group showed a simple main effect of valence, which was superseded by a significant Laterality×Valence interaction. No significant effects were found for the control or remitted groups.
Table 12

*P3b Factorial ANOVA Results: Study 1*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 1.69, p = .191, \eta^2 = .046$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Group</td>
<td>$F(3.83, 134.11) = 0.62, p = .642, \eta^2 = .017$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence × Group</td>
<td>$F(2, 70) = 0.196, p = .822, \eta^2 = .006$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Valence × Group</td>
<td>$F(4, 140) = 3.71, p &lt; .001, \eta^2 = .096$</td>
<td>***</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05$, **$p < .01$. 

Table 13

*Breakdown of P3b Laterality × Valence × Group Interaction: Study 1*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laterality</td>
<td>$F(1.62, 46.92) = 0.28, p = .757, \eta^2 = .010$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 3.12, p = .087, \eta^2 = .097$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Valence</td>
<td>$F(2, 58) = 1.80, p = .174, \eta^2 = .058$</td>
<td>ns</td>
</tr>
<tr>
<td>Remitted Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laterality</td>
<td>$F(2, 24) = 0.44, p = .651, \eta^2 = .035$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 12) = 2.75, p = .052, \eta^2 = .186$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Valence</td>
<td>$F(2, 24) = 1.95, p = .164, \eta^2 = .140$</td>
<td>ns</td>
</tr>
<tr>
<td>MDD Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laterality</td>
<td>$F(2, 58) = 2.65, p = .079, \eta^2 = .084$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 5.53, p = .026, \eta^2 = .160$</td>
<td>*</td>
</tr>
<tr>
<td>Laterality × Valence</td>
<td>$F(2, 58) = 4.90, p = .005, \eta^2 = .145$</td>
<td>**</td>
</tr>
</tbody>
</table>

(directional hypothesis)

*Note.* ns $p > .05$, *$p < .05$, **$p < .01$, ***$p < .001$. 

To analyse the significant interaction for the MDD group, univariate ANOVAs for the impact of valence was conducted for each lateral position. The alpha level was corrected to reduce inflation of Type 1 error with repeated tests to .017 (.05/3 tests). As presented in Figure 29, MDD participants were found to exhibit significantly larger P3b ERPs for positive compared to negative words over the right hemisphere, $F(1, 29) = 10.51, p = .003, \eta^2 = .266$ (mean difference $= 0.904\mu V$, $SEM = 0.28$). No valence differences were found for the left or midline electrode sites.

**Figure 29.** MDD participants’ grand average P3b ERPs over left, midline, and right hemispheres for positive and negative stimuli during the self-referential encoding task. ** p <.01, Error bars refer to standard error of the mean.

To evaluate Hypothesis 2(b), one-tailed Pearson correlations coefficients were calculated between participants’ right P3b scores for positive and negative stimuli and their difference, with self-report questionnaire results. As can be seen in Table 14, greater current and one-month self-reported depression symptoms, rumination, and one-month state anxiety levels were significantly associated with smaller right
hemisphere P3b amplitudes for negative than positive stimuli. No significant
correlations were found between the self-report measures and the right P3b amplitudes
for positive stimuli.

Table 14

*Correlations between Psychometric scores and Right Hemisphere P3b Amplitude*

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>n</th>
<th>Positive words</th>
<th>Negative words</th>
<th>Negative Bias Score (Negative - Positive)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BDI-II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td>73</td>
<td>-.152</td>
<td>-.234**</td>
<td>-.193, p = .052</td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>70</td>
<td>-.184</td>
<td>-.279**</td>
<td>-.218*</td>
</tr>
<tr>
<td><strong>RRS- Total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Reflective</td>
<td>73</td>
<td>-.134</td>
<td>-.218*</td>
<td>-.194*</td>
</tr>
<tr>
<td>- Brooding</td>
<td>73</td>
<td>-.158</td>
<td>-.240*</td>
<td>-.193</td>
</tr>
<tr>
<td>- Depressive</td>
<td>73</td>
<td>-.177</td>
<td>-.250**</td>
<td>-.179</td>
</tr>
<tr>
<td><strong>ERQ</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Reappraisal</td>
<td>73</td>
<td>.168</td>
<td>.138</td>
<td>.033</td>
</tr>
<tr>
<td>- Suppression</td>
<td>73</td>
<td>-.028</td>
<td>-.009</td>
<td>-.035</td>
</tr>
<tr>
<td><strong>STAI-State</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td>73</td>
<td>-.135</td>
<td>-.174</td>
<td>-.102</td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>70</td>
<td>-.171</td>
<td>-.282*</td>
<td>-.224*</td>
</tr>
</tbody>
</table>

* p >.05 ** p >.01

**N400 (200-600 ms) for MDD Participants.** To analyse the predictions of
hypothesis 2(c), MDD participants’ peak N400 amplitudes for positive and negative
stimuli at frontal electrode sites were subjected to a one-way ANOVA. The results of
this analysis were not significant, \(F(1, 29) = 0.89, p = .351, \text{Pr}^2 = .030\). SPSS
reported small power for this analysis (observed power = .150).
**LPP (801 – 2000 ms).** To analyse the predictions of hypothesis 4, peak average LPP amplitudes were subjected to a 3 (Laterality) × 3 (Caudality) × 2 (Valence) × 3 (Group) ANOVA. As can be seen in Table 15, a significant Caudality×Valence×Group interaction was observed.

Table 15

**LPP Factorial ANOVA Results: Study 1**

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 0.71, p = .495, \eta^2 = .020$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Group</td>
<td>$F(3.28, 114.79) = 2.50, p = .058, \eta^2 = .067$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality×Group</td>
<td>$F(3.27, 114.39) = 1.70, p = .166, \eta^2 = .046$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 70) = 0.26, p = .775, \eta^2 = .007$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Laterality×Group</td>
<td>$F(4.95, 173.12) = 0.36, p = .877, \eta^2 = .010$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence×Group</td>
<td>$F(2.69, 94.44) = 2.82, p = .048, \eta^2 = .075$</td>
<td>*</td>
</tr>
<tr>
<td>Laterality×Valence×Group</td>
<td>$F(3.49, 122.16) = 0.47, p = .736, \eta^2 = .013$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Laterality×Valence×Group</td>
<td>$F(6.54, 228.77) = 0.97, p = .451, \eta^2 = .027$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note.* ns $p>.05$, ** $p<.01$.

To analyse the significant three-way interaction, separate Caudality×Valence ANOVAs were conducted on LPP data for each group individually. The results of these sub-analyses are shown in Table 16. Significant interaction was found for the control group. A significant effect of caudality was found for the remitted group, with greater LPPs observed over central than parietal sites ($mean$ difference = 1.34µV, $SEM = .373, p = .011$). No effects were found for the MDD group.
Table 16

*Breakdown of LPP Caudality × Valence × Group Interaction: Study 1*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control Group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.59, 46.01) = 0.06, p = .902, \eta^2 = .002$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 4.09, p = .052, \eta^2 = .124$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Valence</td>
<td>$F(1.28, 37.16) = 4.32, p = .036, \eta^2 = .130$</td>
<td>*</td>
</tr>
<tr>
<td><strong>Remitted Group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.32, 15.85) = 4.88, p = .034, \eta^2 = .289$</td>
<td>*</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 12) = 2.45, p = .143, \eta^2 = .170$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Valence</td>
<td>$F(1.29, 15.49) = 0.29, p = .660, \eta^2 = .023$</td>
<td>ns</td>
</tr>
<tr>
<td><strong>MDD Group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caudality</td>
<td>$F(1.73, 50.13) = 1.96, p = .157, \eta^2 = .063$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1, 29) = 1.32, p = .260, \eta^2 = .044$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Valence</td>
<td>$F(1.37, 39.74) = 1.29, p = .277, \eta^2 = .042$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note. ns p > .05, *p < .05.*

To analyse the interaction for the control group, one-way ANOVAs were conducted on the LPPs evoked for positive and negative stimuli separately for frontal, central, and parietal caudal positions for the control participants. The alpha level was corrected to reduce inflation of Type 1 error with repeated tests to .017 (.05/3 tests).

A significant effect of valence was found for frontal sites ($F(1, 29) = 10.95, p = .003, \eta^2 = .274$), with control participants’ showing greater anterior activation for positive compared to negative words (mean difference = 1.17µV, \textit{SEM} = 0.353). No differences were found over central ($F(1, 29) = 0.49, p = .489, \eta^2 = .017$), or parietal sites ($F(1, 29) = 0.28, p = .601, \eta^2 = .010$). These results are visually presented in Figure 30.
3.3.4. Impact of SSRI Medication or Anxiety Comorbidity on the Results

The behavioural and ERP data was analysed with the exclusion of the five participants in the MDD groups who were being treated with SSRI medication or who were assessed to have a comorbid anxiety disorder (secondary diagnosis). This had a slight reduction in the strength of some of the obtained results, most likely due to the slightly reduced statistical power. However, there were no changes to significant/non-significant findings (See Appendix Q).

3.4. Discussion

Theories and experimental data suggest the existence of a negative attention bias in MDD and positive attention bias in healthy controls (Matthews & MacLeod, 2005). Given that ERPs have a high temporal resolution, Study 1 measured ERPs
continuously during participants self-referential encoding of equi-probable presentations of positive and negative personality adjectives. The aim of the study was to address the problem as to whether attention (P2) and/or enhanced cognitive expectation (P3b, N400) and elaboration (LPP) are involved in attention biases to negative stimuli. The study provided evidence that addressed this aim. The pattern of these experimental results, along with the pattern of questionnaire data, suggests internal validity for the clinical status of the three participant groups. The following sections of this chapter will outline and interpret these results. Specifically, each experimental hypothesis is interpreted sequentially, with behavioural data presented first followed by the interpretation of the electrophysiological results. Limitations, avenues for future research and clinical applications of the findings are discussed in the General Discussion (Chapter 7).

3.4.1. Cognitive Schema Activation

In the context of Beck’s (1967) schema model and Bower’s (1981) mood-congruency model, hypothesis 1 predicted that individuals with MDD would hold more negative self-schemas and thus endorse more negative adjectives as self-descriptive. Control participants were expected to hold more positive self-schema and thus, endorse more positive adjectives as self-descriptive. Given that a mood priming procedure was not used in the study, it was expected that remitted-depressed participants should show a pattern of results similar to that of the control group. Further during self-referential encoding, MDD participants—due to their negative schemata—was predicted to have a higher expectation for negative stimuli in their environment (Beck, 1967). This would result in smaller magnitude of the P3b and N400 to negative than to positive stimuli in the MDD sample (hypothesis 2). A summary of data testing Hypothesis 1 and 2 are displayed in Table 17.
Table 17

*Summary of Self-Evaluation and Expectation Data: Study 1*

<table>
<thead>
<tr>
<th>Hypothesis</th>
<th>Cognitive Assay</th>
<th>Between Subjects Differences</th>
<th>Within Subjects Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothesis 1</td>
<td>Behavioural subjective ratings of schema congruency</td>
<td>Control and remitted participants endorsed significantly more positive words as self-descriptive compared to MDD. No differences existed between the control and remitted participants. MDD participants endorsed significantly more negative words as self-descriptive compared to control and remitted participants. No differences existed between the control and remitted participants.</td>
<td></td>
</tr>
<tr>
<td>Hypothesis 2</td>
<td>P3b</td>
<td>Hypothesis 2(a) MDD participants exhibited greater P3b activation for positive compared to negative words over the right hemisphere.</td>
<td>Hypothesis 2(b) Reduced P3b amplitude for negative compared to positive stimuli over the right hemisphere was associated with greater self-reported rumination and sustained (one-month) depression levels, with a trend ((p = .052)) observed for current depression levels.</td>
</tr>
<tr>
<td></td>
<td>N400</td>
<td>Hypothesis 2(c) Not supported (null). MDD participants exhibited similar N400 activation for positive and negative words.</td>
<td></td>
</tr>
</tbody>
</table>

The MDD participants endorsed more negative words as self-descriptive compared to the controls and remitted participants. Whereas, control and remitted participants endorsed more positive words as self-descriptive compared to MDD participants. These results are consistent with suggestions that depression is
associated with negative self-schemata; while euthymic mood is associated with positive self-schemata (see Flett, Krames, & Vrendenburg, 2009; Ruehlman, West, & Pasahow, 1985). The pattern of these results supports the predictions of hypothesis 1. They are largely consistent to Bower’s (1980) associative memory theory. It is also supportive of Beck’s (1967, 1979) depressive schema model. Beck’s model suggests that depressed mood should skew information processing towards the negative. In remitted depression however, these depressive schemas are said to lie latent. Based on this premise, the detection of depressive cognitions in remitted depression is dependent on these latent depressive schemas becoming primed, such as by stressful events or sad mood induction (see Ingram, Miranda, & Segal, 1998; Miranda & Persons, 1988; Segal & Ingram, 1994). In times of euthymic mood—as in the current study’s remitted sample—it is theorised that the remitted depressed individual will engage in suppression over negative thoughts that are consistent with their latent negative schemata (Wegner, 1994). This serves to maintain their positive state. The results for the remitted group replicate previous findings using self-descriptive encoding tasks (e.g., Blackburn & Symth, 1985; Dobson & Shaw, 1987; Moretti et al., 1996). However, it contradicts the findings of Fritzche et al. (2010). It is likely that the results of Fritzhe et al. (2010) do not readily generalise to the current remitted depressed participants. For instance, Fritzhe et al.’s sample included a mixed gender, medicated community sample of significantly older (mean = 39.95 years) participants. This contrasts to the current sample’s female, unmedicated, younger (26.64 years) and university cohort. That is, the participants used in the current study were relatively high functioning and intelligent and might not be reflective of less functional populations who may be sitting at home (i.e., those in Fritsche et al.). Alternatively, due to their advanced age greater recurrences of depressive experiences might be
expected in Fritsche et al.’s sample in contrast to the current sample. Recall Beck’s model that suggests with repeated activation, core negative schemas become hypersensitive and gain a more rigid, coherent, and elaborated organisation (morph into *depressive modes*: see Section 2.1 and Beck et al., 1996; Clark & Beck, 1999). The negative cognitive schema therefore becomes more readily accessible, thereby increasing the probability that they will be activated and negatively skew information processing. Thus, greater depressive recurrence in Fritsche et al.’s remitted sample may result in more pervasive negative biases in information processing and self-endorsement. Whereas, just over 50% of the current study’s young remitted-depressed group reported to have experienced more than one depressive episode, suggesting that their latent negative schemas are unlikely to have morphed into depressive modes as yet.

Contrary to hypothesis 2, control and remitted participants showed similar P3b amplitude for the analysis of positive and negative stimuli. The null P3b results for the remitted depressed group are inconsistent with the results of Blackburn et al. (1990), who did not observe P3b differences between their remitted participants with their MDD sample. However, Blackburn et al.’s remitted participants were still being treated for depression issues. Thus, it is hard to argue that they were in complete depression remission, which questions the generalisation of their work to other remitted samples. It may be possible that the null differences in the remitted-depressed group may have been caused by insufficient statistical power given its small sample size; especially since the grand average data appeared to suggest heightened P3b amplitude for negative than positive stimuli in the remitted participants (see Figure 26). Although these waveforms suggest greater error variability, no statistical outliers or violations of normality were identified in this dataset (see Appendix L). Rather, the
ERP variability might also be a result of remitted participants being a heterogeneous group by nature (see Just et al., 2001 for a review). Replication of the remitted participants’ results in larger samples is warranted to determine if increased power will elucidate any latent negative processing biases of the P3b for the remitted group.

Consistent with hypothesis 2, participants diagnosed with MDD were found to exhibit the expected reduced P3b amplitude to positive stimuli than negative stimuli. This effect was specific to the right hemisphere electrode sites, which is consistent with Heller’s (1990, 1993) theory of hypoactive right parieto-temporal cortex in depression. Broca’s work localised the left hemisphere as the heart of the language centre. However, it is now widely accepted that the right hemisphere plays a larger role in the perception and expression of emotional language (see review by Beeman & Chiarello, 1998), especially in the case of depression (see Borod et al., 1998; Atchley et al., 2003). Davidson (2003) has shown the right hemisphere to plays an important role in the processing of negative stimuli. He suggests that deficiencies in this region would likely result in negative attention. Thus, the present results suggest that a specific deficit of the right hemisphere in depression may be associated with observed negative schema activation. However, it is important to note that previous work has also shown right-sided parietal activation to be associated with elevations in anxiety (Heller, Nitschke, & Miller, 1998). Given that the MDD participants had elevations in anxiety as part of their symptom profile, this right hemisphere effect may reflect interactions with anxiety. Enhanced right P3bs may not have been observed in the control and remitted participants purely because anxiety comorbidity was screened out in these groups. However this is unlikely as subsequent analysis that excluded all participants with identified secondary anxiety disorder from the dataset found no significant changes to the observed ERP results. It would be of benefit for future
investigations to further disentangle the impact of depression and anxiety on these ERP profiles.

Across all participants, smaller right P3b for negative than positive stimuli correlated with heightened rumination and current and sustained depression severity. The correlations appeared to be similar in magnitude to those observed in previous investigations (e.g., Blackburn et al., 1990; Yang et al., 2011). Although inferences of causation cannot be made from correlational data, it lends support towards Beck’s (1967) theory. Specifically, that negative schemas are associated with sustained depression and recurrent negative thoughts. However, it might also be possible that these correlations are the result of an un-measured variable, such as personality constructs like neuroticism (Lanciano, Bianco, Curci, & Cozzoli Poli, 2009; Lieberman, 2000). Future work using experimental manipulation of schema activation in a prospective longitudinal or in cross-lagged design is needed here. Only these types of designs will be able to specify if expectations for negative information (P3b effects) is a casual factor in sustained depression, rather than just a factor associated with these outcomes. These future investigations should also explore the impact of moderating effects, such as trait rumination, has on the data.

It is important to outline that other ERP studies have reported an opposite pattern of P3 data to that observed in the current study. These prior studies have found MDD participants, but not remitted or control participants, to exhibit significantly larger P3s to negative stimuli relative to neutral stimuli (Ilardi et al., 2007; Nandrino et al., 2004). Based on the theory that enhanced P3 amplitude is an index of greater attentional allocation (Kok, 1997), these researchers have interpreted this effect as indicative of negative attention biases. It is possible that these divergent findings and interpretations of the P3b to that employed in the current study are due to
methodological differences. For instance, both Ilardi et al. and Nandrino et al. utilised an emotional oddball paradigm, with the negative words acting as the rare/target stimulus that required a specific response (20% occurrence in both studies). In contrast, the current study utilised a self-referential encoding task in which no such oddball or response manipulations were made. Thus, the result of the current study more likely reflect that, due to their greater negative schemata, individuals with MDD naturally expect negative stimuli in their environment (as evidenced by their greater endorsement of negative stimuli as self-descriptive) rather than basic oddball effects. This means that the positive stimuli would act as the unexpected material, and thus in the theory of context-updating, would evoke larger P3bs. Similar interpretations were made by Blackburn et al. (1990) and Yang et al. (2011) for their observation of reduced P3b amplitudes to negative relative to positive stimuli in their depressed patients.

Further, both Ilardi et al. (2007) and Nandrino et al. (2004) utilised neutral stimuli as their control/baseline condition. It is argued that neutral stimuli do not reflect an effective control condition as it cannot be matched with valanced material on arousal dimensions (see Section 2.2.2. for additional explanation of these arguments). This can create confusion in the research results as the ERP, especially the P3b, is known to be differentially affected by the arousal of stimuli. For instance, see the experimental results of Codispoti et al. (2006), Delplanque et al. (2006), Dolcos and Cabez (2002), who all noted enhancements of the P3 ERP for more arousing stimuli. Another criticism of the use of neutral stimuli as the supposed “control condition” is the finding that depressed patients have been observed to misinterpret neutral stimuli as negative, as seen in Leppanen, Milders, Bell, Terriere, and Hietanen (2004). Given that word norm lists are based on responses from healthy control samples it might be
erroneous to assume that neutral words derived from these lists would be equally interpreted as neutral for depressed samples. Thus, an alternative explanation for the results of Illardi et al. and Nandrino et al. was that their depressed samples were sensitive to stimuli arousal characteristics, which resulted in the increased P3b for negative rather than neutral words. In the current study, results for positive and negative stimuli were matched on arousal dimensions, thereby reducing any interpretation confounds here.

Further, Ilardi et al. (2007) and Nandrino et al. (2004) failed to differentiate the P3a and P3b. This is a significant limitation as the P3b shows marked reductions in depression samples (see Bruder et al., 2012 for a review). The P3a on the other hand, has been shown in previous work to be decreased in depressed samples but increased in anxious samples (Bruder et al., 2002). Given that the majority of previous ERP studies using depressed samples have had comorbid anxiety issues, it is unknown whether their reported enhanced P3 results are the manifestation of anxiety or depression processes, or both. Thus, the exact ERP changes involved in emotional word processing in depressed and non-depressed states remain controversial. It requires further investigation.

The P3 morphology differences between the current study to Ilardi et al. (2007) and Nandrino et al. (2004) might also be due to divergent participant profiles. The current study employed an all-female sample, whereas both Ilardi et al. and Nandrino et al. included mixed samples. Previous work reports gender-specific lateralisation differences in ERP tests of verbal (Hill et al., 2005; Ortigue et al., 2005; Walla et al., 2001), pictorial (Lithari et al., 2010), and facial stimuli (Guillem & Mograss, 2005). These gender differences are observed to be most pronounced during emotional information processing (Killgore & Yurglun-Todd, 2001; Lee et al., 2002; Schirmer &
Kotz, 2003; Schneider et al., 2000). Specifically, relative to males, females show greater ERP amplitudes to unpleasant and less arousing stimuli (Lithari et al., 2010; Yang et al., 2009). Campanella et al. (2004) suggested that gender differences in emotional appraisal and elaboration appears to originate at an early stage of the information processing system, where attention most likely filters gate sensory inputs. That is, females have been found to show finer-tuned emotional recognition processes, reacting to emotional stimuli earlier in the processing stream than males. It is thus highly probable that some of the mixed P3 data in the depression emotional information processing literature is due to the use of mixed gendered samples. Other sample difference between the current and previous studies that might account for these divergent results is participant age of depressive onset and depression severity.

In the current experiment it is unlikely that participant age had an effect on the P3 data. This is based on prior findings that almost identical topographical distributions and amplitudes of the P3a and P3b have been observed across age group of well-functioning individuals (20-44 years, 45-69 years, and 70-90 years; Fjell & Wallhovd, 2003). Further, Ilardi et al. utilised a relatively young university sample, similar to that employed in the current study. However, the P3b data might have been differently influenced by differences in depression onset between the current sample and that used in Nandrio et al. According to the SCID-I/NP the current sample of MDD and remitted participants all reported pre-adult onset depression, occurring at approximately 15 years old ($SD = 2.56$). The onset of depression in the Ilardi et al. and Nandrino et al. samples are not specified. The MDD patients in the Nandrino et al. study were much older than the current sample (~38 to 43 years). Epidemiological research suggests that the prevalence rate of pre-adult onset depression to be approximately 22-40% (see Alpert et al., 1999; Fava, Alpert, Borus, & Nierenberg,
1996; van Noorden et al., 2011; Zisook et al., 2007). Thus, it may be speculated that only about 22-40% of their sample included pre-adult onset depression, meaning the majority of their sample likely represented adult onset depression. This is an important consideration, as some researchers have questioned whether pre-adult and adult-onset depression represents different subtypes of the disorder (see Kaufman, Martin, King, & Charney, 2001). Thus, Nandrino et al.’s results may not apply to the current findings. Given the similar participant age in Ilardi et al. (~18.88 years) it is possible that this sample had a similar age of depression onset. However, all of Ilardi et al.’s MDD participants presented with only mild severity of depressive symptoms at the time of ERP testing (average BDI-II score = 18.3), and thus may not generalise to those with severe levels of depressive symptomology like those in the current data set (MDD’s average BDI-II score at ERP testing = 29.9). Thus, the depression scores in the patient sample in Ilardi et al. were not stable. Further, their participants were further not assessed for comorbid conditions. This raises concerns over the external validity of their depression classifications.

Fabiani and Donchin (1995) reported that greater distinctiveness to one’s expectations evoke larger N400s. Given that negative items are less characteristic and more frequent with regard to depressive cognitive schemata, larger N400s were thus expected in the MDD group for positive stimuli (hypothesis 2c). Previous work has found this effect to occur over anterior regions (Berman et al., 2011; Dietrich et al., 2000). However, the current data did not support this prediction, with the MDD participants showing similar frontal N400 amplitudes for positive and negative stimuli. This contrasts with previous work by Dietrich et al. (2000) who observed greater anterior N400s to positive relative to negative words in their depressed sample. The current null findings may be subsequent to insufficient statistical power to observe the
effect (e.g., analysis power = .150). Conversely, it may be due to methodological
issues. For instance, the current study did not use the standard semantic incongruence
paradigm—adapted from Taylor’s (1953) ‘cloze probability’ paradigm—that is
typically employed to study the N400 (Kutas & Hillyard, 1984). Conclusions
regarding this ERP during self-referential processing are not well-established. The
current results are consistent with a collection of studies by Deldin et al., (2006) which
also failed to observe N400 modulations in their MDD or dysthymic samples using
this semantic incongruence paradigm. Deldin et al. argued depression to be associated
with intact semantic processing, independent of symptom severity. However, it is
important to note that Deldin et al. utilised a passive paradigm. Other clinical
populations, such as individuals diagnosed with schizophrenic disorders, have shown
to have normal N400s during passive semantic processing tasks (e.g., Mitchell et al.,
1991; Andrews et al., 1993). Interestingly, when required to respond behaviourally to
incongruent and congruent sentence endings, these populations do display reduced
N400s (Nestor et al., 1997; Strandburg et al., 1997). Thus, Deldin et al.’s conclusions
may also be restricted to only cognitive processes associated with passive sentence
reading of non-emotive stimuli. Null results are difficult to interpret distinctively.
Thus, the application of the N400 as an electrophysiological marker of schema
activation in MDD, or conclusions that the current sample failed to show negative
cognitive biases here should thus be discouraged. Future work using an active and
emotional variant of the semantic incongruence task would be more revealing.

3.4.2. Emotional Attention Biases

The P2 and P3a ERP components are thought to represent ERP assays of
orientating attention, whereas the LPP appears indicative of sustained attention and
emotional regulation processes. The results for these ERPs are shown in Table 18.
Table 18

*Summary of Attention Bias Data: Study 1*

<table>
<thead>
<tr>
<th>Hypothesis</th>
<th>Cognitive Assay</th>
<th>Null Results</th>
<th>Between groups Differences</th>
<th>Within group Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orientating Attention</td>
<td>P2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypothesis 3</td>
<td></td>
<td></td>
<td>Control participants</td>
<td>exhibited larger P2 to negative stimuli over anterior sites.</td>
</tr>
<tr>
<td>Orientating Attention</td>
<td>P3a</td>
<td>✓</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sustained Attention and Emotional Regulation Processes</td>
<td>LPP</td>
<td></td>
<td>Hypothesis 4</td>
<td>Control participants exhibited larger LPP to positive stimuli than negative stimuli over anterior sites.</td>
</tr>
</tbody>
</table>

The results of the present study supported the predictions of Williams et al.’s (1997) theory that negative attention biases in depression is not observed at early (P2 or P3a), automatic stages of information processing. This is consistent with behavioural research that failed to observe attention biases in depression (e.g., Bradley et al., 1997; Hill & Dutton, 1989; Kellough, Beevers, Ellis, & Wells, 2008; MacLeod et al., 1986; Mogg et al., 1993; Mogg et al., 2000; Neshat-Doost et al., 2000), and with null ERP evidence when examining early components thought to reflect orientating attentional processes (i.e., *P1, P2*: Deldin et al., 2000; Dietrich et al., 2000; Shimizu et al., 2006). These null findings contradict Beck’s (1967) cognitive theory of depression, which emphasises the function of automatic biases towards schema-congruent negative information. They fail to replicate previous behavioural investigations that have observed such attention biases in current and remitted
depression (Ingram et al., 1994; Joormann & Gotlib, 2007; McCabe, Gotlib, & Martin, 2000; Shestyuk & Deldin, 2010). They also do not replicate Shestyuk and Deldin (2010), who found current and remitted depression to be associated with greater parietal P2 to negative than positive words during self-descriptive encoding. Similar to the above argument in interpreting the divergences in the P3b results, inability to replicate Shestyuk and Deldin’s results may be due to similar differences in participant characteristics such as age and gender. For instance, Shestyuk and Deldin’s sample comprising of both males and female participants who were on average 10 years older than the current study’s exclusively female sample. This age divergence is an important consideration here, as unlike the P3b, the parietal P2 waveform is known to increase in magnitude with age (Crowley & Colrain, 2004).

Alternatively, the divergences in the P2 data for the depressed group might represent differences in methodologies, such as the marked differences in the studies inter-stimulus intervals (ISIs). Shestyuk and Deldin utilised a presentation rate of 200 ms with a 10 000 ISI. This compared to the current study’s presentation rate of 300 ms with a 4000 ms ISI. It is unlikely that stimuli presentation rate had any effect on the ERP effects, given the minimal differences in presentation rate between the two studies (100 ms). This is supported by Kissler, Herbert, Peyk, and Junghofer’s (2007) observation of no differences in posterior ERPs elicited during the 200-300ms post stimulus epoch when positive, negative, and neural words were presented at a rate of one word per second (1 Hz) compared to three words per second (3 Hz). However, Kissler et al. did find a marked effect on these posterior ERPs by manipulating the length of ISIs. Specifically, longer ISIs were found to result in larger ERP amplitudes, suggesting ISI timing is associated with the amount of processing resources available during ERP generation (Polich, 1990). Thus, the lack of enhanced posterior P2 ERPs
for negative stimuli in the MDD sample might alternatively be due to the use of a relatively short ISI in the current study. This might have placed higher strain on cognitive load for the depressed group, thus, reducing P2 manifestation. Further investigations are warranted to investigate the potential moderating factor of ISI on emotional information processing in depression.

The study did find control participants to exhibit enhanced anterior P2 magnitude to negative than positive stimuli. This supported the predictions of hypothesis 3. The P2 component is a positive-going deflection maximally occurring at anterior and central sites around 200 ms after stimulus presentation. Studies have shown that is it sensitive to affective evaluation (Begleiter, Porjesz, & Garozzo, 1979) and has been linked to greater orientating attention towards evolutionarily threatening information (Bartholow et al., 2003). Negative self-descriptive information holds considerable evolutionary value. Humans are not fast, are not strong, and do not have particularly good auditory or visual abilities. What we do possess is remarkable skills in working together as a team. As group membership is so paramount to our survival (and thus the ability to procreate), we need to be sensitive to negative personality characteristics that may result in social rejection (e.g., laziness, rudeness, cruelty). Thus, the enhanced P2s towards negative information in the control group may be reflective of automatic attentional orientating to this potentially threatening information. Awareness of this information would motivate engagement of defensive coping. For instance, externally this may involve the activation of prosocial behaviours to prevent or repair social impasses; whereas, internally this may include activation of cognitive reappraisal processes.

It is important to note that there are reports that anterior P2 is thought to represent the initial differentiation and inhibition of sensory input from further
processing. Specifically, enhanced anterior P2 to negative stimuli has been interpreted to represent increased recruitment of frontal brain regions to inhibit its further processing (Yao et al., 2010). These results are consistent with Lang’s (1995) approach-avoidance motivational system theory, in which positively evaluated stimuli are said to activate an approach motivational system, whereas negatively evaluated stimuli activate an avoidance motivational system. It is argued that these accounts of the P2 are not generalizable to our study. However, they used a NAP task, which does not generalise to the current study’s self-descriptive encoding task. Specifically, it is tenuous to infer about the implicit avoidance motivational system from such an explicit, encoding paradigm where no instruction was given on inhibition. Further, no behavioural (error rates, reaction times) or EEG hemisphere asymmetry was observed in the dataset, which should also be indicative of avoidance attention processes (see research by Davidson, 1984, Davidson et al., 1990; Davidson et al., 2000). This places further reservation on the validity of the interpretation of the P2 as an index of avoidance. Future studies would benefit from examining the above P2 results under conditions of self-descriptive encoding whilst assessing attentional approach or attentional avoidance to stimuli using eye-tracking technology. An eye tracker assesses the changes in attentional deployment over time and continuously records the exact position of an individual’s eye gaze without requiring an explicit response (Hermans, Vansteenkoven, & Delen, 1999; Isaacowitz, 2005). Under normal viewing conditions, individuals typically direct their line of gaze towards stimuli that attract their attention (Jonides, 1981). Shifts in gaze position are guided by shifts in attentional focus. Eye tracking technology may be particularly advantageous in the investigation of depressive samples as visual attention is not confounded by slowed motor response typical of this population. To date, a number of eye tracking studies
have found that depressed participants have difficulty disengaging their attention from negative material (Caseras, Garner, Bradley, & Mogg, 2007; Eizenman et al., 2003; Sanchez, Vazquez, Marker, Lemoult, & Joormann, 2013) and spend less time attending to positive material (Kellough, Beevers, Ellis, & Wells, 2008; Sears, Newman, Ference, and Thomas, 2011). This future work might help to better validate the conceptual interpretation of the P2 for negative stimuli during encoding.

In the current experiment, the separate PCAs (see Section 3.3.1.) for the Control, Remitted, and MDD samples all clearly identified a sustained positivity between 800 – 2000 ms that appeared consistent with the LPP (Schupp, Junghofer, Weihe, & Hamm, 2004). However, this ERP showed a small magnitude (approximately 2µV), variable latency, and lacked clearly discernable peaks in the grand-average data (see Figures 24-26). In fact, negative peaks were evident in the grand average data during this epoch for the control participants. Thus, it is important to note that despite ANOVA assumptions of normality being met for the above analysis (with no identified outliers) special caution needs to be taken in the interpretation of the data. Further, in the current study, the LPP was measured only up to 2000 ms. Thus, the presence of later sensitivity of the LPP to cognitive emotional regulation cannot be evaluated. With these limitations in mind, the current study found control participants to exhibit smaller LPP to negative compared to positive words. This supported the predictions of hypothesis 4. The amplitude of the LPP decreases when participants follow reappraisal instructions to inhibit distress elicited by unpleasant stimuli (Hajcak & Nieuwenhuis, 2006; Krompinger et al., 2008; Moser et al., 2006; Moser, Most, & Simons, 2012; Moser et al., 2009). Neuroimagining studies have documented the neural bases of reappraisal (Ochsner & Gross, 2005) and mid-latency LPPs (1000-2000 ms; Denis & Hajcak, 2009) to be associated with
enhanced recruitment of prefrontal areas. Thus, this reduced mid-latency LPP towards negative stimuli at anterior sites in the control group likely represents cognitive reappraisal strategies so to reduce the impact of negative on affect. This was possibly motivated by their earlier orientating system towards negative information (P2 data outlined above). The lack of LPP difference in the remitted or MDD groups appears to hint at cognitive reappraisal deficits in these populations. As outlined by recent models (e.g., Beck, 2008; Joormann et al., 2007), these deficit prefrontal executive control processes may serve to increase an individual’s tendency to ruminate on negative material, thereby increasing their risk for depressive disorders. The current findings are preliminary and would benefit from further replication in a larger sample and for longer latency LPP epochs (e.g., see Shestyuk et al., 2005). Further, ERP extraction techniques, such as area under curve might also prove beneficial given the long latency, and thus variability of this ERP.

3.4.3. Summary of Findings

The results of this study found MDD to be associated with more negative self-evaluations resulting in greater expectation of negative than positive stimuli (P3b), which is coupled with compromised right-parietal hemisphere activation. This electrophysiological negativity bias positively correlated with the severity of depressed and anxious mood and self-reported level of rumination. Both the P2 and LPP data suggest that susceptibility to depression may also derive from a lack of an innate or learned bias of the perceptual system. Specifically, it appears that depressed and remitted-depressed individuals have difficulty automatically identifying interpersonal threatening information (P2s for negative trait adjectives). This may result in social difficulties, such that have been typically observed in depression. They also appear to show impaired ability to turn attention towards positive stimuli in an attempt to
reappraise mood in the face of stress (enhanced LPP to positive information). Due to the cross-sectional nature of the current research design (participants were selected based on the presence or absence of a depression disorder) casual inferences cannot be made from this data. Thus, it is unknown whether the absence of this potential protective effect—represented by the P2 and LPP components—reflects a cognitive vulnerability factor increasing predisposition to depression or if it represents a cognitive scar following the experience of depression. Future replication and longitudinal investigation will help decipher this important question.

The present results suggest there is a potential benefit of investigating the impact of age of depressive onset on the emotional information processing system. This may have implications in clinical treatment decision making. Study limitations and clinical implications of the experimental results are outlined in Section 7.3. and 7.4. of the General Discussion, respectively. The study represents an important step in developing a deeper and more comprehensive understanding of depression through integrating neural and cognitive models of the disorder. The next chapter of this dissertation specifically explores recent episodic memory processing for emotional stimuli in the same sample of participants utilised in the current study.
Chapter 4

Study 2 – Emotional Explicit Memory Biases in Depression

Definition of Terms

*Autobiographical memory*: Memory for an individual’s life that consists of both episodic (details of personal experiences) and semantic (general knowledge) details.

*Cognitive Schema/Schemata*: An organised mental structure of an individual’s idiosyncratic knowledge and assumptions of self, other, and the world which is used for interpreting and processing information.

*Executive Functions*: Executive functions (also known as cognitive control/ executive control/ supervisory attentional system) are defined as basic cognitive control mechanisms that contribute to more complete cognitive functioning. They are considered to be volitional and effortful. They are centrally located in the frontal lobes and maintain tight connection throughout the brain.

*Explicit Memory*: Also called declarative memory. It involves the conscious, intentional memory of experiences and information.

*Familiarity*: A feeling or sensation that a stimulus has been previously observed, but this is not accompanied by any contextual information about that experience.

*Memory Search*: Involves the effortful and controlled attempted recall of a memory trace from long term storage.

*Mood-Congruent Memory*: A memory process that selective retrieves memories that match (are congruent with) one’s mood.

*Old/New Paradigm*: An experimental task typically used to measure explicit recognition memory in which participants’ are required to learn a list of stimuli (study phase). They then perform a recognition test, in which they are presented the studied stimuli intermixed with distractor stimuli (test phase). The task requires the participants to identify if they recognise the stimuli as part of the studied list (old stimulus) or not (new stimulus).

*Recollection*: When explicit memory for contextual information can be reconstructed about the stimulus.

*Semantic Incongruence*: When the content of presented semantic information is unexpected. It evokes the N400 ERP.
4.1. Background and Hypotheses

The aim of this study was to investigate the neural processes underlying recent episodic memory biases for emotional word stimuli in a sample of MDD, remitted depressed, and healthy female participants (i.e., controls). These were the same participants that completed Study 1 (Chapter 3). Here, an emotional Old/New task was employed, in which ERPs in response to correctly recognised items from Study 1 were intermixed with new/distractor items. This task evokes the mid-frontal (FN400) effect and the late positive complex (LPC) Old/New effects (Friedman, 2000; Friedman & Johnson, 2000; Rugg, 1995; Rugg & Allan, 2000; Rugg & Curran, 2007). The FN400 is considered to represent familiarity memory processes; whereas the LPC effect is considered to represent recollection memory processes (Friedman, 1990; Rugg, 1987; Rugg & Nagy, 1989). The late parietal negativity (LPN) is another ERP related to explicit memory processing. Specifically, it appears to reflect memory search attempt (Friedman et al., 2005). Together, these ERPs were used to assess mood-congruent memory biases and their predicted correlations with rumination and sustained depression (Joormann, 2009). They were also measured to assess theorised deficits in working memory functioning in MDD, as observed by Dietrich et al. (2000). Finally, this study sought to explore the neural processing supporting the normalisation of this memory function in depressive recovery.

4.1.1. Study background. Behavioural correlates of memory processes in depression are typically measured using free-recall and recognition paradigms. Research using these techniques has shown that depressed individuals recall on average 10% more negative stimuli than positive stimuli (Matt et al., 1992). As outlined in section 2.3.2., emotional memory shows a linear relationship with depressive severity. Memory biases for positive material are more typically observed
in non-depressed samples (Deldin et al., 2001; 2009), while dysphoric and remitted samples show no memory biases (Jermann et al., 2008; Ridout et al., 2009), and clinically depressed samples more typically show memory biases for negative material (Gilboa-Schnechtman et al., 2002; Ridout et al., 2003). In the behavioural studies, however, it is difficult to distinguish whether this effect is the result of enhanced memory operations for negative stimuli or due to preferential encoding of negative information. The superior temporal resolution of the ERP has merit in locating the stage/s of information processing implicated here.

Electrophysiological correlates of memory processes have been examined using the Old/New word recognition memory task (Friedman, 1990). This task evokes the FN400, and the LPC old/new effects (Friedman, 2000; Friedman & Johnson, 2000; Rugg, 1995; Rugg & Allan, 2000; Rugg & Curran, 2007). Recall from section 2.2., in the normative literature more negative FN400 are observed for new compared to old items, while more positive LPCs are found for old compared to new items (Curran & Friedman, 2004). In the context of the Old/New task, these ERPs are hypothesised to involve neural integration of memory processing of subsystems of the medial temporal lobe structure. Particularly in the Hippocampus and Amygdala projections to the prefrontal and parietal cortex (see Gordeev, 2007; Paller & Kutas, 1992; Rugg et al., 1996; Rugg et al., 1991). Depressed patients have shown reduced old/new ERP effects in contrast to control groups, who in turn tend to show larger late potentials to processing of positive material (Deldin et al., 2001; Dietrich et al., 2000). Context integration—a major task of the central executive system of working memory (Baddeley, 2000; Baddeley & Hitch, 1974)—is required for apt memory processing. Thus, the overall reduction of the ERP old/new effect in the depressed participants might be the result of global deficits in working memory functioning in the disorder.
This premise underpins both Beck (2008) and Joormann et al.’s (2007) contemporary models of depression (see Section 2.1.). Supporting Beck’s (1967) original model, the old/new ERP effect has not been found to normalise after clinical remission (Dietrich et al., 2000).

Another ERP associated with memory processes is the LPN (600-2000ms). This ERP appears to comprise subcomponents sensitive to retrieval ease (600 – 1200 ms) and post-retrieval judgments (1200 – 1900 ms; Herron, 2007). The magnitude of the LPN has been found to be larger when search conflict is higher (Curran, DeBuse, & Leynes, 2007). Thus, it appears to reflect memory search attempt (Friedman et al., 2005). To this PhD candidate’s knowledge, there are no prior ERP studies of MDD that have examined the LPN during old/new judgments. However, based on Bower’s (1980) associative network theory (see Section 2.1.) it would be anticipated that positive items would be more readily encoded and recalled by those in positive or euthymic moods. Thus, for non-depressed individuals, negative items would result in shallower encoding, thereby requiring a greater search attempt during episodic memory judgments. This would result in larger LPN evoked for negative than positive old items for control participants. The opposite effect, larger LPNs for positive than negative old items would be expected for those in negative mood states, such as for those with MDD.

Another ERP associated with cognitive expectation—irrespective of stimuli being old or new in nature—is the N400. As outlined in Section 3.1., the N400 is said to be evoked in response to semantic incongruence (Kutas & Federmeier, 2000). Thus, the higher congruency of positive words presented to predominately positive thoughts of control participants would predict a reduced N400 for positive compared to negative words. In reverse, a reduced N400 for the negative than positive words in
the MDD group would be expected given that negative items are less distinctive and more frequent with regard to their depressive schemata (Dietrich et al., 2000).

### 4.1.2. Study hypotheses.

Thus, the following hypotheses were predicted:

**Replication hypotheses.** The following replication hypotheses were measured to validate the sample and methodological procedure. The data from these predictions helped to test the hypotheses measured in Study 3 (Chapter 5) and 4 (Chapter 6).

*Hypothesis 1(a)* predicted that control individuals would exhibit greater free-recall for positive words relative to negative words, while MDD participants would recall more negative words relative to positive words. This was tested with planned \( t \)-test comparisons. No recall differences between positive and negative words were expected for the remitted depressed participants.

*Hypothesis 1(b)* predicted that for positive stimuli, control participants would free-recall more words than the MDD participants. For negative stimuli, MDD participants would free-recall more words than the control and remitted participants. This was analysed using a between-subjects ANOVA, with planned post-hoc \( t \)-test comparisons (\( \alpha = .05 \)).

*Hypothesis 1(c)* predicted that across all participants, greater recall of negative than positive stimuli (negative recall bias scores) was expected to be positively correlated with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and negatively correlate with emotional regulation scores (as measured by the ERQ).

*Hypothesis 2(a)* predicted that control individuals would exhibit greater recognition memory for positive words relative to negative words, while MDD participants would recognise more negative words relative to positive words. These predictions were tested with planned \( t \)-test comparisons. No recognition memory
differences were expected between positive and negative stimuli for the remitted depressed participants.

*Hypothesis 2(b)* predicted that control participants would recognise more positive words than the MDD participants. Also, MDD participants would recognise more negative old words than the control and remitted participants. This was analysed using a between-subjects ANOVA, with planned post-hoc *t*-test comparisons (*α* = .05).

*Hypothesis 2(c)* predicted that across all participants, greater recognition of negative than positive stimuli (negative recognition memory bias scores) was expected to be positively correlated with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and negatively correlated with emotional regulation scores (as measured by the ERQ).

**Electrophysiological hypotheses**.

*Hypothesis 3 (a)* predicted a novelty-by-caudality-by-valance-by-group interaction for the N400 data. Simple main effect analyses would show both the control and remitted participants to exhibit significantly larger N400 waves over frontal sites for old relative to new items. This would be reflective of the FN400 old/new effect and apt memory functioning. No frontal N400 effects were expected for the MDD participants, suggestive of deficits in explicit autobiographical recognition processes, and poor executive control. These predictions were analysed

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6 In regards to the ERP data it is important to note that the hypotheses pertain to two N4 waves: the FN400 old/new effect and the N400. The FN400 old/new effect tends to be manifested over frontal regions and is larger for old then new stimuli; it tends to assay familiarity memory (Curran & Friedman, 2004; Dietrich et al., 2000; Kayser et al., 2010; Rugg & Curran, 2007). It was predicted in Hypothesis 3. On the other hand, a N400 that fails to distinguish between novelty conditions is interpreted as a function of word expectancy, rather than familiarity (Khateb et al., 2010; Kutas & Hillyard, 1980, 1982; Lau et al., 2008). It was predicted in Hypothesis 6.
with a 2 (novelty) × 3 (caudality) × 2 (valence) × 3 (group) factorial ANOVA. The expected simple main effect for the old stimuli will be explored using separate 2 (valence) × 3 (group) ANOVAs at frontal, central, and parietal caudal locations. Planned *t*-tests will be used to analyse the differences between positive and negative stimuli for each group.

*Hypothesis 3(b)* predicted that across all participants, larger FN400 magnitude to negative than positive stimuli (negative bias scores) was expected to negatively correlate with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and positively correlate with emotional regulation scores (as measured by the ERQ). It was further expected to negatively correlate with greater negative biases in free-recall and recognition memory.

*Hypothesis 4 (a)* predicted a novelty- by-caudality-by-valance-by-group interaction for the LPC data. Reflective of apt memory functioning, simple main effect analyses would show both the control and remitted participants to exhibit significantly larger LPC waves for old relative to new items, maximum at parietal and central sites. Suggestive of deficits in explicit autobiographical recognition processes, and poor executive control, no LPC effects were expected for the MDD participants. The hypothesis will be analysed with a 2 (novelty) × 3 (caudality) × 2 (valence) × 3 (group) factorial ANOVA using a similar protocol outlined in Hypothesis 3.

*Hypothesis 4(b)* predicted that across all participants, larger parietal LPC magnitude to negative than positive stimuli (negative bias scores) was expected to positively correlate with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and negatively correlate with emotional regulation scores (as measured by the ERQ). It
was further expected to positively correlate with greater negative biases in free-recall and recognition memory.

_Hypothesis 5(a)_ was exploratory in nature. Based on Bower’s (1980) associative network theory, hypothesis 5 (a) predicted a novelty-by-valance-by-group three way interaction for the LPN data. Specifically, control participants were expected to show enhanced LPN for negative old words compared to positive old words, reflective of greater search efforts for this mood-incongruent material. Depressed individuals were expected to show enhanced LPNs for positive as compared to negative old words. This hypothesis was analysed using a 2 (Novelty) x 2 (Valence) x 3(Group) ANOVA. The expected simple main effect for the old stimuli was be explored using Bonferonni-correct post hoc analyses ($\alpha /3$ test =.017) for each group, thus, analysing valance differences.

_Hypothesis 5(b)_ predicted that that across all participants, greater LPN to negative than positive stimuli (negative expectation bias scores) was expected to positively correlate with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and negatively correlate with emotional regulation scores (as measured by the ERQ).

_Hypothesis 6 (a)_ predicted that given their predominantly negative schemata, participants with MDD would exhibit significantly larger parietal N400 for positive than negative words, while control participants would show larger parietal N400s for negative than positive words. No valence effects were predicted for the remitted participants. To test this hypothesis, the N400 data was averaged across novelty conditions and a 2 (Valence) x 3(Group) ANOVA was completed at partial electrodes. The expected interaction was analysed with planned $t$-test analyses.
Hypothesis 6(b) predicted that across all participants, smaller parietal N400 to negative than positive stimuli (negative expectation bias scores) was expected to negatively correlate with self-reported depressed mood (as measured by the BDI-II), and rumination and suppression scores (as measured by the RRS and ERQ), and positively correlate with emotional regulation scores (as measured by the ERQ). It was further expected to negatively correlate with endorsement of negative stimuli as self-descriptive in a self-referential encoding task (data derived from Study 1), and with greater negative biases in free-recall and recognition memory.

4.2. Method

4.2.1. Participants

Participants of this study consisted of the same sample of 73 female university students who were recruited in Study 1 (see Section 3.2.1.). As in Study 1, participants were placed into one of three groups according to their SCID-I/NP diagnoses:

1. Current depressed group (MDD; n = 30).
2. Remitted depressed group (remitted; n = 13).
3. Healthy control/never depressed group (control; n = 30).

See Section 3.3.1. for data on participant characteristics (Table 4).

4.2.2. Materials

Clinical and psychometric assessments. The self-report questionnaires and structured clinical interviews utilised in this study are outlined in Section 3.2.2.

Experimental stimuli. Experimental stimuli included the same pool of 135 positive and 135 negative words that were selected from standardized databases (Anderson, 1968; Kirby & Gardener, 1972) and utilised in Study 1. These 270 adjectives were then evenly distributed into three lists: List A, B, and C (Appendix J),
ensuring that no list included variations of the same word (e.g., admirable, admired).

Each list included 45 positive adjectives (e.g., considerate, friendly, nice), and 45 negative adjectives (e.g., selfish, vulgar, worthless). The three lists were counterbalanced as the old or new list across participants. Further details on the selection process of experimental stimuli and their properties (e.g., length, familiarity and valence ratings) can be found in Section 3.2.2 of this dissertation.

**EEG recording apparatus.** The sample EEG recording apparatus utilised in Study 1 and explained in detail in Section 3.2.2 of this dissertation was employed.

### 4.2.3. Procedures

**EEG recording and data reduction.** The same EEG recording and data reduction procedures utilised in Study 1 and explained in detail in Section 3.2.3. of this dissertation was employed.

**Experimental tasks.**

**Free recall memory task.** As outlined in Section 3.2.3, after completion of the self-referential encoding task in Study 1 (see Table 7 for a flowchart of experimental procedures, Section 3.2.3.), participants were asked to free recall as many words from this presentation as they could within a five-minute time frame. The researcher scored the number of correctly recalled positive and negative adjectives. After a short break (of approximately three minutes) participants then proceeded to complete the Old/New task to assess memory processing (FN400, LPC, LPN) and schema activation (N400).

**Recognition memory: the Old/New paradigm.** To test recognition memory processes, participants were presented with a list of previously presented 90 adjectives—the old list (encoded in Study 1) which included 45 positive, 45 negative words. This list was randomly mixed with 90 never-seen adjectives—the new list which also included 45 positive, 45 negative words. Following each word presentation,
participants were required to answer the question “Do you recognize this word from the first task?” by pressing either the Yes or No key on the computer keyboard. The same stimulus presentation timing as the self-referential encoding task used in Study 1 was employed. A schematic depiction of stimulus presentation rate and ISI for the Old/New task is shown in Figure 31. The Old/New task took approximately 15 minutes to complete. Dependent variables were frequency of recognition of positive or negative adjectives, error rates, reaction times, the FN400, LPC, and LPN old/new ERP effects, and N400. The instructions for the task (see Appendix R) was visually presented to participants via the computer screen and verbally explained by the PhD candidate to ensure understanding.

Figure 31. Schematic depiction of the stimulus presentation rate and ISI for the Old/New task.
Statistical Analyses

The same statistical analysis protocol utilised in Study 1 and outlined in Section 3.2.3.5. of this dissertation was employed.

4.3. Results

4.3.1. Behavioural Data

To reduce clutter and inflation of Type I error, only results that pertain to the research hypothesis or to the key variable group are presented.

Free recall task. Table 19 summarises mean percentage free-recall of positive and negative stimuli following the self-referential encoding task for the three participant groups. To test the predictions of hypothesis 1(a), planned *t*-tests were conducted between participants’ recall rates for positive and negative words (see Table 19). Control participants were found to recall significantly more positive than negative words. No significant valence differences in recall existed for the remitted or for the MDD participants.

Table 19

*Mean Percentage Free Recall (SD in parentheses) of Positive and Negative stimuli for Control, Remitted, and MDD Participants: Study 2*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>15.93 (5.53)</td>
<td>13.68 (4.33)</td>
<td>11.78 (6.07)</td>
</tr>
<tr>
<td>Negative</td>
<td>9.04 (4.81 )</td>
<td>11.11 (5.37)</td>
<td>12.44 (5.39)</td>
</tr>
<tr>
<td>Test of Difference</td>
<td><em>t</em>(29) = 6.80, <em>p</em> &lt; .001</td>
<td><em>t</em>(12) = 1.59, <em>p</em> = .137</td>
<td><em>t</em>(29) = -0.67, <em>p</em> = .510</td>
</tr>
</tbody>
</table>

To test the predictions of hypothesis 1(b), univariate ANOVAs were conducted on between-subjects recall rates for positive and negative stimuli separately. For the positive stimuli, a significant main effect of group was observed, \( F (2, 70) = 4.14, p = \)
Planned comparisons found control participants recalled more positive words than MDD participants (mean difference = 4.15%, \( p = .016 \)); no differences existed between control and remitted participants (\( p = .687 \)), or between remitted and MDD participants (\( p = .929 \)). For the negative stimuli, a significant simple main effect of group was also observed, \( F(2, 70) = 3.30, p = .043, \eta^2 = .086 \). Planned comparisons found MDD participants recalled more negative words than control participants (mean difference = 3.41%, \( p = .038 \)); no differences existed between control and remitted or between remitted and MDD participants.

To test the predictions of hypothesis 1(c), the association between participants’ percentage free-recall of positive and negative stimuli, and their respective difference scores (negative bias scores), with their self-report questionnaire results were analysed using Pearson’s correlation coefficient. The results of these correlations are shown in Table 20. As can be seen, greater free-recall for negative stimuli was positively correlated with current and sustained self-reported depression and anxiety levels, and self-reported rumination. It was negatively correlated with self-reported emotional regulation as measured via the ERQ.
Table 20

Correlations between Psychometric scores and Percentage Free Recall of Positive and Negative Stimuli: Study 2

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>n</th>
<th>Free Recall for Positive</th>
<th>Free Recall for Negative</th>
<th>Bias Score (Negative - Positive)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI-II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At testing</td>
<td>73</td>
<td>-.391***</td>
<td>.300**</td>
<td>.598***</td>
</tr>
<tr>
<td>Follow-up (1-month)</td>
<td>70</td>
<td>-.401***</td>
<td>.209*</td>
<td>.533***</td>
</tr>
<tr>
<td><strong>RRS-T Total</strong></td>
<td>73</td>
<td>-.391***</td>
<td>.218*</td>
<td>.530***</td>
</tr>
<tr>
<td>Reflective</td>
<td>73</td>
<td>-.192</td>
<td>.239*</td>
<td>.369***</td>
</tr>
<tr>
<td>Brooding</td>
<td>73</td>
<td>-.439**</td>
<td>.185</td>
<td>.546***</td>
</tr>
<tr>
<td>Depressive</td>
<td>73</td>
<td>-.403**</td>
<td>.189</td>
<td>.517***</td>
</tr>
<tr>
<td><strong>ERQ</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regulation</td>
<td>73</td>
<td>.252*</td>
<td>-.080</td>
<td>-.293**</td>
</tr>
<tr>
<td>Suppression</td>
<td>73</td>
<td>-.069</td>
<td>.094</td>
<td>.139</td>
</tr>
<tr>
<td><strong>STAI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At testing</td>
<td>73</td>
<td>-.301**</td>
<td>.328**</td>
<td>.540***</td>
</tr>
<tr>
<td>Follow-up (1-month)</td>
<td>70</td>
<td>-.284**</td>
<td>.277**</td>
<td>.482***</td>
</tr>
</tbody>
</table>

*Note.* *p > .05  **p > .01  ***p < .001

The Old/New task (recognition memory task). Table 21 summarises correct recognition rates for the Old/New task for the three experimental groups. As can be seen, error rates did not exceed 40% across conditions. The results showed no significant differences in mean errors between control, remitted, and MDD groups in participants’ identification of old ($F(2, 70) = 1.75, p = .180, P\eta^2 = .048$) or new stimuli ($F(2, 70) = 0.180, p = .835, P\eta^2 = .005$). However, across all groups, greater errors were made for the new compared to the old stimuli, $t(72)=-2.12, p = .037$. To test the predictions of hypothesis 2(a), planned $t$-tests were conducted between recognition rates for positive and negative words for each group. These results are
shown in Table 21. All participants were found to correctly recognise significantly more positive than negative old words.

Table 21

*Mean Percentage Correct Recognition Hit Rates (SD in parentheses) and Error Rates for the Old/New Task for Control, Remitted, and MDD Participants: Study 2*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Correctly Identified Old Words (Recognition)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>80.74 (9.75)</td>
<td>87.00 (7.79)</td>
<td>78.00 (12.46)</td>
</tr>
<tr>
<td>Negative</td>
<td>64.07 (15.47)</td>
<td>70.59 (13.04)</td>
<td>73.48 (12.11)</td>
</tr>
<tr>
<td>Test of Difference</td>
<td><em>t</em>(29)= 6.71, <em>p</em> &lt;.001</td>
<td><em>t</em>(12)= 4.95, <em>p</em> &lt;.001</td>
<td><em>t</em>(29)= 2.54, <em>p</em> =.016</td>
</tr>
<tr>
<td>Overall Error Rate</td>
<td>13.63 (5.53)</td>
<td>10.47 (4.43)</td>
<td>11.76 (5.81)</td>
</tr>
<tr>
<td><strong>Correctly Identified New Words</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>62.22 (14.26)</td>
<td>61.05 (9.96)</td>
<td>63.92 (15.89)</td>
</tr>
<tr>
<td>Negative</td>
<td>78.37 (9.58)</td>
<td>76.41 (8.54)</td>
<td>76.07 (14.67)</td>
</tr>
<tr>
<td>Test of Difference</td>
<td><em>t</em>(29)= -7.97, <em>p</em> &lt;.001</td>
<td><em>t</em>(12)= -4.56, <em>p</em> =.001</td>
<td><em>t</em>(29)= -5.18, <em>p</em> &lt;.001</td>
</tr>
<tr>
<td>Overall Error Rate</td>
<td>14.72 (5.41)</td>
<td>15.74 (3.54)</td>
<td>14.29 (7.05)</td>
</tr>
</tbody>
</table>

To test the predictions of hypothesis 2(b), univariate ANOVAs were conducted on participant’s percentage recognition rates for positive and negative old stimuli. For the positive stimuli, a significant simple main effect of group was observed, *F* (2, 70) = 3.22, *p* = .046, *Pη²* = .084. Planned comparisons found remitted participants correctly recognised more positive words than MDD participants (*mean difference* = 9.00%, *p* = .040); no differences existed between control and remitted (*p* = .245), or
between the control and MDD participants ($p = .972$). For the negative stimuli, a significant group difference was also observed, $F (2, 70) = 3.74, p = .029, P\eta^2 = .097$. Planned comparisons found MDD participants to correctly recognise more negative words than control participants (mean difference = 9.41%, $p = .026$); no differences existed between control and remitted ($p = .450$) or between remitted and MDD participants ($p > .999$). No significant group differences existed for percentage hit rates for positive or negative new stimuli.

To test the predictions of hypothesis 2(c), correlations between participants’ recognition of positive and negative old stimuli, and their respective difference scores, their scores on the self-report questionnaires were analysed using one-tailed Pearson’s coefficient. The results of these correlations are shown in Table 22. As can be seen, positive correlations were observed between negative memory bias scores and depression, rumination, and anxiety scores. Greater use of emotional reappraisal strategies was associated with less memory for negative than positive stimuli.
Table 22

**Correlations between Psychometric Test Results and Participants Recognition of Positive and Negative words and their respective Negative Memory Bias Scores**

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>n</th>
<th>Positive</th>
<th>Negative</th>
<th>Bias Score (Negative - Positive)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI-II</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>At testing</em></td>
<td>73</td>
<td>-.275**</td>
<td>.322**</td>
<td>.575***</td>
</tr>
<tr>
<td><em>Follow-up (1-month)</em></td>
<td>70</td>
<td>-.299**</td>
<td>.230*</td>
<td>.497***</td>
</tr>
<tr>
<td>RRS- Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Reflective</em></td>
<td>73</td>
<td>-.289**</td>
<td>.208*</td>
<td>.464***</td>
</tr>
<tr>
<td><em>Brooding</em></td>
<td>73</td>
<td>-.156</td>
<td>.141</td>
<td>.282**</td>
</tr>
<tr>
<td><em>Depressive</em></td>
<td>73</td>
<td>-.240*</td>
<td>.228*</td>
<td>.445***</td>
</tr>
<tr>
<td>ERQ</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Regulation</em></td>
<td>73</td>
<td>.307**</td>
<td>-.052</td>
<td>-.313**</td>
</tr>
<tr>
<td><em>Suppression</em></td>
<td>73</td>
<td>-.192</td>
<td>.146</td>
<td>.316**</td>
</tr>
<tr>
<td>STAI-State</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>At testing</em></td>
<td>73</td>
<td>-.175</td>
<td>.349**</td>
<td>.519***</td>
</tr>
<tr>
<td><em>Follow-up (1-month)</em></td>
<td>70</td>
<td>-.203*</td>
<td>.208*</td>
<td>.393***</td>
</tr>
</tbody>
</table>

*Note.* *p > .05 ** *p > .01 *** *p < .001

**Reaction time data.** Table 23 shows participants’ mean reaction time (RT) data for correct and incorrect recognition responses for positive and negative stimuli during the Old/New task. A 3 (group) × 2 (novelty) × 2 (response: Correct versus incorrect) × 2 (valence) ANOVA found no significant effects (main or interaction) involving the key variable group.
Table 23

*Mean Reaction Time (ms; SD in parentheses) for Correct and Incorrect Responses for Positive and Negative stimuli during Old/New Memory Task*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Correct</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Old <strong>Positive</strong></td>
<td>976.16 (415.81)</td>
<td>968.41 (197.22)</td>
<td>1045.36 (233.17)</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>981.74 (168.74)</td>
<td>1039.27 (218.61)</td>
<td>1056.39 (219.15)</td>
</tr>
<tr>
<td>New <strong>Positive</strong></td>
<td>990.24 (159.40)</td>
<td>1079.47 (212.10)</td>
<td>1100.96 (231.68)</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>940.61 (157.97)</td>
<td>1027.61 (222.86)</td>
<td>1046.86 (199.85)</td>
</tr>
<tr>
<td></td>
<td>Incorrect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Old <strong>Positive</strong></td>
<td>1009.43 (308.11)</td>
<td>1223.87 (454.99)</td>
<td>1243.92 (434.50)</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>1016.94 (192.89)</td>
<td>1114.86 (219.22)</td>
<td>1171.52 (283.43)</td>
</tr>
<tr>
<td>New <strong>Positive</strong></td>
<td>1002.79 (211.86)</td>
<td>1052.94 (235.16)</td>
<td>1111.28 (286.44)</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>1127.72 (274.95)</td>
<td>1132.52 (263.31)</td>
<td>1127.40 (281.15)</td>
</tr>
</tbody>
</table>
4.3.2. Electrophysiological Data

4.3.2.1. Preliminary Analyses to Identify Relevant ERP Components

See Section 2.2.3. of this dissertation for an outline of the PCA protocol and Section 3.3.3.1. for an outline of the PCA procedures used for the study. The PCA analysis was completed for the experimental groups separately, given that ERP differences are expected between the groups.

**Control Group.** The PCA for the ERP data consisted of rows of participants \((n = 30)\), the four experimental conditions \((\text{valence [positive, negative]} \times \text{novelty [old, new]})\), and nine electrode sites \((F_3, F_Z, F_4, C_3, C_Z, C_4, P_3, P_Z, P_4)\). Data was originally recorded at 1000 points per second (Hz). To conform to PCA analysis assumptions, the original data was reduced to represent a sampling rate of 200 points per second, which reduced the number of variables (columns) from 2000 to 400 by averaging every five sequential data points (across 2000 ms). That is, the matrix included a total of \(30 \times 4 \times 9\) rows for the 400 columns of time samples of EEG. The PCA for the old/new memory task data indicated that six components should be retained, and these accounted for 73.13% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated these components are shown in Appendix S. In temporal sequence, the approximate peak latency of loadings for each component was: Component 5 and 6 at 350 ms; Component 4 at 600 ms; Component 1 at 800 ms; Component 2 at 800 ms; Component 3 at 1000 ms.

Component 2, 4, 5, and 6 all represented negative peak latency between 300-500 ms. Inspection of the grand average ERP waveforms (see Figure 37) elicited for the Control participants for the experimental conditions between this time window suggest that this extracted negativity in these components might represent the N400 and/or FN400. Specifically, the N400 for the new experimental stimuli (Kutas &
Hillyard, 1983) and the FN400 Old/New effect for the old experimental stimuli (Curran & Friedman, 2004; Dietrich et al., 2000; Kayser et al., 2010; Rugg & Curran, 2007). The latency of the positive peak in Component 1, 2, and 4 appeared to be similar to the grand average ERP waveforms between 600-1200 ms post-stimulus at the parietal and central electrode sites. Thus, this component appeared consistent with the LPC—an ERP often associated with recollection processes (Paller & Kutas, 1992). A late, sustained negativity was identified in Component 3 between 600 and 1400 ms. This may represent late posterior distributed negative-going slow wave (LPN; see Section 4.3.1). Consistent with the parietal maximum of this ERP, the grand average waveforms do not appear to show any discernible sustained negativity in the 600-1400 ms epochs in frontal electrodes for the Control participants (see Figure 37). Rather, it seemed to be exclusively evident at the central and parietal sites.

Thus, for the Control participants, the 300-500 ms temporal window was deemed a suitable epoch to measure the negative peak of the FN400 and N400. The 500 – 1200 ms was selected to measure the positive peak of the LPC. The 1200 – 1600 ms temporal window was selected to measure the negative peak of the LPN. The epoch selected for the LPN is later than that identified by the PCA output. However, the pooled data for all participants suggested that this later epoch more reliably captured the LPN waveform (see Figure 35, Section 4.3.2.2.). The 1200-1600 ms window also replicates that used in previous research (e.g., the 1400-1600ms epoch was used to measure the LPN in Leynes, Crawford, Radebaugh, & Taranto, 2013). The six extracted components and the selected epochs for each of the above ERPs are shown in Figure 32.
**Component 6**
*Peak latency: 350 ms*
3.39% explained variance

**Component 5**
*Peak latency: 350 ms*
4.61% explained variance

**Component 4**
*Negative peak latency: 350 ms*
*Positive peak latency: 600 ms*
5.33% explained variance

**Component 2**
*Negative peak latency: 350 ms*
*Positive peak latency: 1000 ms*
14.98% explained variance

**Component 1**
*Peak latency: 800 ms*
33.94% explained variance

**Component 3**
*Peak latency: 1000 ms*
10.87% explained variance

*Figure 32.* ERP epochs for the control group for study 2


**Remitted group.** The PCA for the ERP data consisted of rows of participants ($n = 13$), the four experimental conditions (valence [positive, negative] × novelty [old, new]), and nine electrode sites ($F_3$, $F_Z$, $F_4$, $C_3$, $C_Z$, $C_4$, $P_3$, $P_Z$, $P_4$). Data was originally recorded at 1000 points per second (Hz). To conform to PCA analysis assumptions, the original data was reduced to represent a sampling rate of 200 points per second, which reduced the number of variables (columns) from 2000 to 400 by averaging every five sequential data points (across 2000 ms). That is, the matrix included a total of $13 \times 4 \times 9$ rows for the 400 columns of time samples of EEG. The PCA for the old/new memory task data indicated that four components should be retained, and these accounted for 83.31% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated with these components are shown in Appendix T. In temporal sequence, the approximate peak latency of loadings for each component was: Component 3 and 1 at 450 ms; Component 4 at 650 ms; Component 2 at 780 ms.

It appeared that Component 3 primarily represented negative peak latency. Inspection of the grand average ERP waveforms elicited for the Remitted participants (Figure 37) during this time window suggest that this extracted negativity might represent both the N400 and FN400. The peak latency of 2 and 4 appeared to be similar to the grand average ERP waveforms between 500-1200 ms post-stimulus at the parietal and central electrode sites. Thus, this component appeared consistent with the LPC Old/New effect (Paller & Kutas, 1992). A sustained negativity was identified in Component 1 between 600 and 1400 ms. This negativity appeared consistent with the LPN Old/New effect. This ERP is not easily discernable in the grand average data for the Remitted group (Figure 38), most likely due to the small magnitude of this effect and the heterogeneous nature of this sample.
Thus, for the Remitted participants, the 300-500 ms temporal window was deemed a suitable epoch to measure the negative peak of the FN400 and N400. The 500 - 1200 ms window was used to measure the positive peak of the LPC. Again, the 1200 – 1600 ms temporal window was selected to measure the negative peak of the LPN. This is a later epoch than suggested by the PCA output. However, as argued for the control participant’s data, the pooled data for all participants and previous research suggested this later epoch more reliably captured this ERP. This later epoch was also utilized so to better distinguish the LPC form the LPN when using peak-detection methods. The four extracted components and the selected epochs for each of the above ERPs are shown in Figure 33.
Figure 33. ERP epochs for the remitted group for study 2
**MDD group.** The PCA for the ERP data consisted of rows of participants \( n = 30 \), the four experimental conditions (valence [positive, negative] \( \times \) novelty [old, new]), and nine electrode sites (F\(_3\), F\(_Z\), F\(_4\), C\(_3\), C\(_Z\), C\(_4\), P\(_3\), P\(_Z\), P\(_4\)). Data was originally, recorded at 1000 points per second (Hz). To conform to PCA analysis assumptions the original data was reduced to represent a sampling rate of 200 points per second, which reduced the number of variables (columns) from 2000 to 400 by averaging every five sequential data points (across 2000 ms). That is, the matrix included a total of 30\( \times \)4\( \times \)9 rows for the 400 columns of time samples of EEG. The PCA for the old/new memory task data indicated that six components should be retained, and these accounted for 77.27\% of the variance for the promax rotation. The scree plot displaying the percentage of variance associated with these components are shown in Appendix U. In temporal sequence, the approximate peak latency of loadings for each component was: Component 2 and 6 at 350 ms; Component 4 at 500 ms; Component 1 at 750 ms; and Component 5 and 3 at 800 ms.

Inspection of the component loadings suggested that Component 2, 4, 5, and 6 represented a negative peak between 300 and 500 ms post-stimulus. Similar to that of the Control and Remitted participant groups, it is understood that this extracted negativity represents both the N400 and FN400. The positive peak latency in Components 2 and 4, appeared similar to the positive peak between 500-1200 ms seen in the grand average waveforms for the MDD participants at the parietal and central electrode sites (Figure 39). Based on this latency, topography, amplitude and experimental task, this component appeared consistent with the LPC—an ERP often associated with recollection processes (Paller & Kutas, 1992). Last, a sustained negativity was identified in Component 3 between 600 and 1400 ms. This negativity
appeared consistent with the LPN Old/New effect and can be clearly seen in the MDD grand average data (see Figure 39).

Thus, for the MDD participants—similar to their Control and Remitted counterparts—the 300-500 ms temporal window was deemed a suitable epoch to measure the negative peak of the FN400 and N400. The 500 - 1200 ms window was used to measure the positive peak of the LPC. Again, the 1200 – 1600 ms temporal window was selected to measure the negative peak of the LPN. This is a later epoch than suggested by the PCA output. However, as argued for the control participant’s data, the pooled data for all participants and previous research suggested this later epoch more reliably captured this ERP. This later epoch was also utilized so to better distinguish the LPC form the LPN when using peak-detection methods. The six extracted components and the selected epochs for the above ERPs are shown in Figure 34.
Component 6
Peak latency: 350 ms
3.08% explained variance

Component 2
Negative peak latency: 350 ms
Positive peak latency: 850 ms
20.71% explained variance

Component 4
Negative peak latency: 350 ms
Positive peak latency: 500 ms
5.59% explained variance

Component 5
Negative peak latency: 350 ms
Positive peak latency: 800ms
3.92% explained variance

Component 1
Positive peak latency: 750 ms
Sustained negativity: 1200-2000ms
33.75% explained variance

Component 3
Peak latency: 800 ms
10.22% explained variance

Figure 34. ERP epochs for the MDD group for Study 2
4.3.2.2. Validation of the Old/New ERPs: Impact of Novelty

Across all participants, a significant effect of novelty was observed for the FN400 (data averaged across all frontal electrode sites), $F(1, 68) = 7.84, p < .001, \eta^2 = .788$. As can be seen in Figure 35 (a), more negative FN400 were evoked for new compared to old stimuli. At central-parietal electrodes, a significant effect of novelty was also observed for the LPC, $F(1, 68) = 2.963, p = .045, \eta^2 = .084$. As can be seen in Figure 35 (b), more positive LPC were evoked for old compared to new stimuli. A significant effect of novelty was also observed for the LPN at central-parietal electrodes, $F(1, 68) = 2.845, p = .044, \eta^2 = .086$. More negative LPN was evoked for old compared to new stimuli (see Figure 35(b)).

Figure 35. [A] Participants grand average FN400 ERPs (averaged over group and valence) for old (black line) and new (grey line) stimuli at electrode site Fz. [B] Participants grand average LPC and LPN ERPs (averaged over group and valence) for old (black line) and new (grey line) stimuli at electrode site Pz.
4.3.2.3. ERP Results

Grand average ERP waveforms for positive and negative old and new stimuli during recognition task for the control, remitted, and MDD participants are shown in Figures 36, 37, and 38, respectively. Visual inspection suggests that control participants showed greater LPN for old relative to new stimuli. Remitted participants appeared to exhibit larger FN400 old stimuli at central sites and larger LPC for negative old stimuli at central and parietal sites. Limited differences for the impact of valence were visible in the grand average data for the MDD participants.
Figure 36. Grand average ERPs for positive (grey tones) and negative (blue tones) old (dark lines) and new (light lines) stimuli for control participants during the Old/New recognition memory task. Stimulus onset 200ms (dotted line).
Figure 37. Grand average ERPs for positive (grey tones) and negative (blue tones) old (dark lines) and new (light lines) stimuli for remitted participants during the Old/New recognition memory task. Stimulus onset 200ms (dotted line).
Figure 38. Grand average ERPs for positive (grey tones) and negative (blue tones) old (dark lines) and new (light lines) stimuli for MDD participants during the Old/New recognition memory task. Stimulus onset 200ms (dotted line).
N400 (300-500ms). To analyse the predictions of hypotheses 3(a) and 6(a) peak average N400 amplitudes were subjected to a 2 (novelty) × 3 (caudality) × 2 (valence) × 3 (group) factorial ANOVA. As shown in Table 24, the results of this analysis found a significant Valence×Group interaction.

Table 24

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 1.05, p = .357, P\eta^2 = .029$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Group</td>
<td>$F(2, 70) = 0.70, p = .500, P\eta^2 = .020$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Group</td>
<td>$F(2.45, 85.61) = 0.65, p = .553, P\eta^2 = .018$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 70) = 4.02, p = .022, P\eta^2 = .103$</td>
<td>*</td>
</tr>
<tr>
<td>Novelty×Caudality×Group</td>
<td>$F(2.67, 93.56) = 0.49, p = .668, P\eta^2 = .014$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Valence×Group</td>
<td>$F(2, 70) = 0.01, p = .999, P\eta^2 &lt; .001$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence×Group</td>
<td>$F(2.55, 89.34) = 0.20, p = .866, P\eta^2 = .006$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Caudality×Valence×Group</td>
<td>$F(3.22,112.68 ) = 1.22, p = .303, P\eta^2 = .034$</td>
<td>ns</td>
</tr>
</tbody>
</table>

Note. * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$, ns $p > .05$.

To identify the source of the Valence×Group interaction, separate one-way ANOVAs were conducted on the N400 data (averaged over novelty and caudality) for each group, thereby analysing the impact of valence. A trend was observed for control participants, $F(1, 29) = 3.41, p = .075, P\eta^2 = .105$, with them showing slightly larger N400s for negative compared to positive stimuli ($mean$ difference = -.521µV, $SEM$ = .282). As displayed in Figure 39, MDD participants exhibited significantly larger N400s for positive than negative words, $F(1, 29) = 4.29, p = .047, P\eta^2 = .129$, $mean$ difference = -.549µV, $SEM$ = .265). No differences were found for the remitted group.
To test the predictions of hypothesis 6(b), planned (one tailed) Pearson’s correlations were conducted to test the predicted associations between enhanced N400 for negative stimuli compared to positive stimuli (and their negative bias scores: N400s for negative stimuli minus N400s for positive stimuli) with participant self-reported psychometric scores. These results are shown in Table 25. As can be seen in the table, N400 bias scores were negatively correlated with greater depression, anxiety, rumination scores, and less emotional regulation. Participants’ N400 negative bias scores also negatively correlated with endorsement of negative stimuli as self-descriptive \((r = .322, p = .006)\), with participants’ free recall \((r = .330, p = .005)\) and recognition memory bias scores\((r = .274, p = .021)\).
Table 25

*Correlations between Psychometric scores and N400s evoked for Positive and Negative Stimuli and the Negative Bias scores*

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>n</th>
<th>Positive Stimuli</th>
<th>Negative Stimuli</th>
<th>Negativity Bias Score (Negative stimuli – Positive stimuli)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI-II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At testing</td>
<td>71</td>
<td>-.013</td>
<td>.154</td>
<td>-.293**</td>
</tr>
<tr>
<td>Follow-up (1-month)</td>
<td>69</td>
<td>-.041</td>
<td>.134</td>
<td>-.363**</td>
</tr>
<tr>
<td><strong>RRS- Total</strong></td>
<td>71</td>
<td>-.100</td>
<td>.077</td>
<td>-.316**</td>
</tr>
<tr>
<td>Reflective</td>
<td>71</td>
<td>-.146</td>
<td>-.005</td>
<td>-.259*</td>
</tr>
<tr>
<td>Brooding</td>
<td>71</td>
<td>.001</td>
<td>.155</td>
<td>-.268*</td>
</tr>
<tr>
<td>Depressive</td>
<td>71</td>
<td>-.103</td>
<td>.071</td>
<td>-.312**</td>
</tr>
<tr>
<td><strong>ERQ</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regulation</td>
<td>71</td>
<td>.022</td>
<td>-.153</td>
<td>.306**</td>
</tr>
<tr>
<td>Suppression</td>
<td>71</td>
<td>.002</td>
<td>-.015</td>
<td>-.031</td>
</tr>
<tr>
<td><strong>STAI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At testing</td>
<td>71</td>
<td>.063</td>
<td>.111</td>
<td>-.308**</td>
</tr>
<tr>
<td>Follow-up (1-month)</td>
<td>69</td>
<td>.000</td>
<td>.149</td>
<td>-.306**</td>
</tr>
</tbody>
</table>

Note. *p > .05 **p > .01 ***p < .001

Contradicting the predictions of Hypothesis 3(b), participants’ FN400 ERPs evoked for positive and negative old stimuli, and their difference scores did not correlate with any of the psychometric questionnaires or behavioural free-recall or recognition results.

**The LPC old/new effect (500-1200 ms).** To analyse the predictions of hypothesis 4(a), peak average LPC amplitudes were subjected to a 2 (novelty) × 3 (caudality) × 2 (valence) × 3 (group) factorial ANOVA. This analysis did not find any
significant main effect or interactions involving the key variable group. These results are presented in Table 26.

Table 26

*LPC Old/New Effect Factorial ANOVA Results: Study 2*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 0.38, p = .682, P\eta^2 = .011$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Group</td>
<td>$F(2, 70) = 1.19, p = .308, P\eta^2 = .033$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Group</td>
<td>$F(2.75, 96.33) = 1.17, p = .321, P\eta^2 = .033$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 70) = 1.25, p = .294, P\eta^2 = .034$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Caudality×Group</td>
<td>$F(3.13, 109.83) = 1.20, p = .312, P\eta^2 = .033$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Valence×Group</td>
<td>$F(2, 70) = 0.49, p = .610, P\eta^2 = .014$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality×Valence×Group</td>
<td>$F(2.57, 89.94) = 1.02, p = .378, P\eta^2 = .028$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Caudality×Valence×Group</td>
<td>$F(2.89, 101.22) = 0.26, p = .848, P\eta^2 = .007$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05.$

Contradicting the predictions of Hypothesis 4(b), participants’ LPC ERPs evoked for positive and negative old stimuli, and their difference scores did not correlate with any of the psychometric questionnaires or behavioural free-recall or recognition results.

**LPN (1200-1600 ms).**

To examine the predictions of hypothesis 5(a), a 2 (Novelty) x 2 (Valence) x 3(3Group) ANOVA was conducted on participants’ LPN data. As seen in Table 27, the three-way Novelty×Valence×Group interaction was significant. No effects involving caudality were observed.
Table 27

LPN Factorial ANOVA Results: Old/New Recognition Task: Study 2

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(1, 68) = 0.37, p = .693, \eta^2 = .011$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Group</td>
<td>$F(2, 68) = 2.37, p = .102, \eta^2 = .065$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 68) = 1.08, p = .347, \eta^2 = .031$</td>
<td>ns</td>
</tr>
<tr>
<td>Novelty×Valence×Group</td>
<td>$F(2, 68) = 3.48, p = .037, \eta^2 = .091$</td>
<td>*</td>
</tr>
</tbody>
</table>

Note. * $p \leq .05$, ns $p > .05$.

To analyse the interaction, separate Group×Valence ANOVAs were conducted on LPN data for the Old and New conditions separately. The results of these analyses are presented in Table 28. As can be seen in the table, a significant simple main effect of Valence×Group was found for the old stimuli. No effects was found for new stimuli.

Table 28

Breakdown of LPN Valence-by-Novelty-by-Group Interaction: Study 2

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Old Stimuli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>$F(2, 68) = 0.14, p = .873, \eta^2 = .004$</td>
<td>ns</td>
</tr>
<tr>
<td>Valance</td>
<td>$F(2, 68) = 0.67, p = .455, \eta^2 = .011$</td>
<td>ns</td>
</tr>
<tr>
<td>Group×Valence</td>
<td>$F(2, 68) = 3.47, p = .036, \eta^2 = .093$</td>
<td>*</td>
</tr>
<tr>
<td>New Stimuli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>$F(2, 68) = 1.08, p = .359, \eta^2 = .033$</td>
<td>ns</td>
</tr>
<tr>
<td>Valance</td>
<td>$F(2, 68) = 1.53, p = .221, \eta^2 = .022$</td>
<td>ns</td>
</tr>
<tr>
<td>Group×Valence</td>
<td>$F(2, 68) = 0.82, p = .439, \eta^2 = .024$</td>
<td>ns</td>
</tr>
</tbody>
</table>

Note. * $p \leq .05$, ns $p > .05$. 
Bonferonni-correct post hoc analyses ($\alpha/3$ test = .017), found control participants showed significantly larger LPNs for negative compared to positive old stimuli ($F (1, 29) = 7.47, p = .011, \eta^2 = .204, \text{power} = .752$). These results are displayed in Figure 40 ($\text{mean difference} = -1.03\mu\text{V}, \text{SEM} = .377$). No differences for valence were found for the remitted ($F (1, 12) > 0.01, p = .986, \eta^2 > .001, \text{power} = .050$) or MDD groups ($F (1, 27) = 0.98, p = .332, \eta^2 = .035, \text{power} = .159$).

Correlation analyses were completed to test the predictions of hypothesis 5(b). Participant’s LPN bias scores (LPNs for negative minus LPNs for positive Old stimuli) was found to correlate with greater recognition of positive words during the old/new task ($r = .501, p < .001$), greater free recall of positive material ($r = .373, p = .001$), and enhanced N400s for negative relative to positive material ($r = -.264, p = .027$). As can be seen in Table 29, it was also associated with greater self-reported
depressive, rumination, and anxiety scores. It was positively associated with reduced self-reported use of emotional reappraisal strategies as measured by the ERQ.

Table 29

_Correlations between Psychometric scores and LPN amplitude for Positive and Negative Old Stimuli, and the Negative Bias Score: Study 2_

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>Positive</th>
<th>Negative</th>
<th>Negative Bias Score (Negative stimuli – Positive stimuli)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI-II</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td>-.070</td>
<td>.258**</td>
<td>.357***</td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>-.140</td>
<td>.131</td>
<td>.298**</td>
</tr>
<tr>
<td><strong>RRS- Total</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Reflective</td>
<td>-.227</td>
<td>-.056</td>
<td>.176</td>
</tr>
<tr>
<td>- Brooding</td>
<td>-.048</td>
<td>.256**</td>
<td>.327**</td>
</tr>
<tr>
<td>- Depressive</td>
<td>-.052</td>
<td>.184</td>
<td>.258*</td>
</tr>
<tr>
<td><strong>ERQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Regulation</td>
<td>.032</td>
<td>-.157</td>
<td>-.205*</td>
</tr>
<tr>
<td>- Suppression</td>
<td>.183</td>
<td>.229*</td>
<td>.058</td>
</tr>
<tr>
<td><strong>STAI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td>-.143</td>
<td>.134</td>
<td>.296*</td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>-.144</td>
<td>.091</td>
<td>.240*</td>
</tr>
</tbody>
</table>

*Note. *p >.05 **p >.01

4.3.3. Impact of SSRI Medication and Comorbid Anxiety on the Obtained Results

The data was analysed with the exclusion of the four participants in the MDD groups who were being treated with SSRI medication or having a secondary anxiety diagnosis (n = 5). There were no changes to significant/non-significant findings (See Appendix Q).

4.4. Discussion

Increase in memory for negative information when it is encoded in relation to the self has been found consistently in studies of depression (Austin, Mitchell, &
Goodwin, 2001; Burt, Zembar, & Niederehe, 1995; Tavares, Drevets, & Sahakian, 2003). Memory processes in the disorder have been typically measured using free-recall and recognition behavioural task, and the Old/New ERP paradigm. The aim of the present study was to investigate the neural processes underlying recent episodic memory biases for emotional word stimuli in a sample of MDD, remitted depressed, and healthy control participants using an emotional Old/New task. This task evokes the ‘Old/New ERP effects’, specifically the FN400 and LPC. Larger amplitudes of the Old/New ERP effects (more negative for the FN400 and more positive for the LPC) are typically observed for old compared to new stimuli (Friedman, 2000; Friedman & Johnson, 2000; Rugg, 1995; Rugg & Allan, 2000; Rugg & Curran, 2007). The LPN appears after the old/new LPC. It is said to represent sustained evaluation memory processing, such as memory search. It is highest when stimulus details are not readily recovered during retrieval (see Johansson & Mcklinger, 2003 for a review). These ERPs were used to assess neural evidence of explicit episodic recognition mood congruent memory biases in the participant groups. The study also assessed whether these mood-congruent memory biases were associated with self-reported rumination, sustained depression (Joormann, 2009), and negative stimuli expectation (e.g., N400 ERPs and the P3b results of Study 1). The evoked components allowed for the testing of the theorised deficits in working memory functioning in depression and the process of normalisation of this function in depressive recovery.

Recall that the experimental hypotheses pertained to two N4 waves, the FN400 Old/New effect (hypothesis 3) and the semantic incongruence N400 effect (hypothesis 6). The FN400 Old/New effect tends to be manifested over frontal regions and is larger for old than new stimuli and it tends to assay familiarity memory (Curran & Friedman, 2004; Dietrich et al., 2000; Kayser et al., 2010; Rugg & Curran, 2007). The
N400 that fails to distinguish between novelty conditions, on the other hand, is interpreted as a function of semantic incongruence. Thus, in the context of depression it is expected that larger N400s would emerge following the less anticipated positive word stimuli (Khateb et al., 2010; Kutas & Hillyard, 1980, 1982; Lau et al., 2008).

The study achieved some of its experimental aims. It replicated previous behavioural evidence of mood-congruent memory effects in the behavioural data. However, no divergent group ERP data was found for the FN400 or the LPC Old/New effect, despite the task reliably evoking these components (see Section 4.3.2.2.). It is likely the relative ease of the recognition task might have contributed to the lack of group differences. That is, the task may not have required excessive involvement of the central executive system, which might be needed to expose the predicted cognitive deficit in the disorder (Hertel, 1997). The ERP data was indicative of schema expectancy effects. Specifically, the N400 results showed MDD participants had greater expectation for negative word stimuli compared to positive stimuli. Also, the LPN data demonstrated that control participants experienced greater amplitudes for negative old stimuli, suggesting greater search attempts were needed here.

The following sections of this discussion will summarise and interpret the study’s results. Specifically, each experimental hypothesis is interpreted sequentially, with behavioural information presented first following by the interpretation of the electrophysiological results. Limitations, avenues for future research and clinical applications of the findings are discussed in the General Discussion (Chapter 7).

**4.4.1. Emotional Memory Biases**

**Behavioural evidence.** A summary of resultant behavioural data for Hypothesis 1 (free recall) and 2 (recognition memory) is shown in Table 30. Overall, the behavioural data suggested that the effects of depression on memory vary as a
function of (1) clinical status: current versus remitted depression. (2) The valence of the stimuli: positive versus negative words. (3) The nature of the memory task: free-recall versus recognition.

Table 30

**Summary of the data for Recall and Recognition Memory (Hypothesis 1-4): Study 2**

<table>
<thead>
<tr>
<th>Cognitive Process</th>
<th>Between Group Differences</th>
<th>Within Participant Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypothesis 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free recall memory</td>
<td><strong>Hypothesis 1(a)</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control participants recalled more positive words than negative words.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Hypothesis 1(b)</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control participants recalled more positive words than the MDD participants. MDD participants recalled more negative words than control participants.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Hypothesis 1(c)</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Overall recall memory bias (greater recall of negative stimuli compared to positive stimuli) was found to positively correlate with level of self-reported depression, anxiety, and rumination, and to negatively correlate with use of emotional regulation.</td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recognition Memory</td>
<td><strong>Hypothesis 2 (a)</strong></td>
<td></td>
</tr>
<tr>
<td>(Error rates)</td>
<td>Contrary to mood-congruency predictions, all participants correctly recognised more positive than negative old words.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Hypothesis 2 (b)</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Remitted participants recognised more positive old stimuli compared to MDD participants. MDD participants recognised more negative old stimuli compared to controls.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Hypothesis 2 (c)</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recognition memory bias (greater recognition of negative compared to positive stimuli) was found to positively correlate with depression, anxiety, and rumination, and to negatively correlate with emotional reappraisal.</td>
<td></td>
</tr>
</tbody>
</table>

Largely consistent with the behavioural expectations of hypothesis 1, the results evidenced a preferential processing of positive stimuli in free-recall in control participants. Specifically, the control group showed enhanced free-recall for positive words over negative words. The remitted and MDD participants showed no valence
differences in free-recall rates. As predicted, the control participants recalled more positive words than the MDD participants. The MDD participants, in turn, recalled more negative words than the control participants. These findings generally support the idea of mood-congruent memory (Beck, 1967; Bower, 1981; Matt et al., 1992); the theory that individuals have enhanced memory for information that is concordant with their current mood state. The observation of no statistically divergent group valence recall rates with remitted participants supports the hypothesis that emotional memory shows a linear relationship with depressive severity. Memory biases for positive materials are more typically observed in non-depressed samples (Deldin et al., 2001; 2008), while dysphoric or remitted samples show no memory biases (Jermann et al., 2008; Ridout et al., 2009), and clinically depressed samples more typically show memory biases for negative materials (Gilboa-Schnechtman et al., 2002; Ridout et al., 2003). Furthermore, consistent with Joormann et al.’s model (2007), greater accessibility of negative than positive stimuli during free-recall was associated with greater current and sustained depression and anxiety levels, greater rumination and less use of emotional reappraisal.

Overall, recognition memory error rates were very low for each of the participant groups (e.g., all > 16%), with no differences observed between them. However, across all participants, greater errors were seen for new stimuli (errors of commission) compared to old stimuli (errors of omission). This provides evidence that word stimuli were encoded effectively into memory in the study phase (study 1) for all participant groups. It is also suggestive that participants followed the instructions of the experimental task, supporting its internal validity. Contrary to the predictions of hypothesis 2 (a), all participants recognised more positive than negative words; with remitted participants recognising more positive words than the MDD participants.
These results are inconsistent with previous theories of mood-congruent memory processing (Beck, 1967; Bower, 1981). This unanticipated finding may be a result of the recognition memory task being too insensitive to produce the mood-congruency effect. Several other studies using recognition tasks have also failed to produce significant mood-congruent results (e.g., Danion, Kauffmann-Muller, Grange, Zimmerman, & Greth, 1995; Dietrich et al., 2000; Nikendei, Dengler, Wiedemann, & Pauli, 2005). This result may alternatively reflect participant’s use of pleasant memories as an emotional regulation strategy. That is, participants may be actively retrieving pleasant memoires to maintain euthymic moods (e.g., non-depressed participants) or to regulate or reverse unpleasant mood states (e.g., MDD participants; Josephson, Singer, & Salovery, 1996). This is supported by the correlation data of hypothesis 2 (c) that found greater use of self-reported emotional reappraisal strategies to be associated with less recognition of positive relative to the negative old stimuli.

Positive correlations were also observed between negative recognition memory bias scores and depression, rumination, and anxiety scores. At first glance, these associations appear interesting given the lack of expected negative memory biases for the MDD group in hypothesis 2 (a). As in previous work (e.g., Blackburn et al., 1990), all participants were tested together to test the correlation predictions. It is likely that given the correlational data was completed for all participants together, the resultant larger sample size improved the power to identify this association compared the smaller samples used in the group analysis. However, it is important to note that given the heterogeneous nature of the sample (MDD, remitted depressed, and never-depressed individuals), the value of the calculated correlations may be biased. For instance, consistent with the predictions of hypothesis 2(b), depressed participants were found to recognise significantly more negative words than never-depressed
participants, supporting Beck’s (1967) assumption of greater activation of negative cognitions in depression. Remitted depressed participants were found to exhibit greater recognition of positive stimuli compared to the MDD group. This latter effect likely reflects the remitted participants’ employment of emotional regulation strategies, such as greater activation of positive memories in an attempt to maintain their euthymic mood. Given these between group differences for the recognition memory effects, when all participants are analysed together, three distinctive “clouds” of results may have ensued in the scatterplot. Subsequently, a high correlation may result that is entirely due to the arrangement of the three groups, but which does not represent the true relation between the two variables (e.g., depression level and recognition memory biases). Thus, these results need to be interpreted with caution (as with all correlational data). Although desirable, the small size of the experimental groups unfortunately rendered group-based correlations unreliable and therefore, they were not conducted. It is suggested for future work to focus on examination group correlations in larger samples of participants. These results should be further verified with use of regression based analyses.

**Electrophysiological evidence.** A summary of resultant ERP data for Hypothesis 3 (ERP effects for familiarity: FN400), 4 (ERP effects for recollection: LPC) and 5 (ERP effects for memory search: LPN) is shown in Table 31.
Table 31

Summary of the data for Old/New ERP effects (Hypothesis 3-5): Study 2

<table>
<thead>
<tr>
<th>Cognitive Process</th>
<th>Between Group Differences</th>
<th>Within Participant Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypothesis 3</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FN400 effects</td>
<td>Null results.</td>
<td>Null results.</td>
</tr>
<tr>
<td>(familiarity)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 4</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPC effects</td>
<td>Null results.</td>
<td>Null results.</td>
</tr>
<tr>
<td>(recollection)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 5</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LPN effects</td>
<td>Control participants had greater LPN for negative than positive Old stimuli.</td>
<td>Greater LPN for negative than positive Old stimuli was found to correlate with greater recognition of positive words, greater free recall of positive material and with greater self-reported depressive, rumination, and anxiety scores.</td>
</tr>
<tr>
<td>(Memory search)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Old/New ERP components evoked in the present study were comparable with other studies using similar paradigms (e.g., Deldin et al., 2001; Dietrich et al., 2000; Rugg et al., 1996). Specifically, when averaged across all participants, significantly larger FN400s, LPCs, and LPNs were observed for old compared to new items. Contrary to expectations of Hypotheses 3 and 4, however, these FN400 and LPC ERPs did not appear modulated by clinical status or stimulus valence. These results failed to replicate the work by Dietrich et al. (2000) who observed an increased positivity beginning approximately 250 ms post stimulus for old relative to new items in their control participants but not in their MDD participants. The absence of negative mood-congruent recognition memory bias and of the predicted reduced Old/New ERP effect in the MDD might be due to the influence of comorbid anxiety disorders in the group. Anxiety issues typically do not present with explicit negative memory biases (Coles & Heimberg, 2002; Heinrichs & Hofmann, 2001; Rinck & Becker, 2005; Williams et al., 1997). It was decided to incorporate participants with...
comorbid anxiety disorders in the study in an effort to increase the ecological validity of the results (Beuke et al., 2003; Ingram, 1989; Ingram & Hamilton, 1999).

Alternatively, the null Old/New ERP results got the FN400 and LPC might be due to the influence of medication influence in the participant sample, with data showing improvement of memory functioning with SSRIs (Levkovitz, Caftori, Avital, & Richter-Levin, 2002). Specifically, administration of SSRI antidepressant medication appears to reduce Amygdala activation to negative stimuli in both clinical (Fu et al., 2004; Sheline et al., 2001) and nonclinical samples (Harmer, Mackay, Reid, Cowen, & Goodwin, 2006). Similar to the decision to include participants with comorbid anxiety disorder in the study, those currently taking SSRI medication were also included to increase the ecological validity of the results. The National Institute of Clinical Excellence (2004) guidelines recommend a combination of CBT and SSRI mediation as the first-line treatment for severe depression. The MDD participants in the current study self-reported severe levels of depression. Thus, the results of the study generalise to understanding the severely depressed patient, who would most likely be medicated. In an attempt to evaluate the moderating effect of these potential confounders, two subsequent analyses were conducted on the ERP data: in which (1) participants on antidepressant medication were excluded; and (2) participants with secondary anxiety disorders diagnoses were excluded. These subsequent analyses—based on a MDD sample of 25 participants—did not lead to significant differences to the original results for the LPC or N400 data. Given that the reduced MDD sample size in these later analyses continue to be larger than that used in Dietrich et al. (2000), who were able to find the predicted group ERP differences, it is unlikely that these null effects are due to insufficient experimental power.
The lack of an association between depression status and the FN400 and LPC ERP effects might lastly be due to the recognition task occurring almost immediately after the self-referential encoding and free-recall task. As a result, this might have reduced opportunity for memory decay; thereby contributing to the relatively high recognition accuracy rates. However, this explanation is inconsistent with the evidence of ERP differences in Dietrich et al.’s study which had a much shorter delay (employed a continuous word Old/New paradigm) than that used in the current study. Alternatively, as argued in the behavioural evidence section, it is likely that the relative ease of the recognition task might have also contributed to the lack of group differences, in that it did not require excessive involvement of the central executive which would be required to produce the expected reduced ERPs (Heller, 1993).

Alternatively, the contradictory findings of this study to Dietrich et al. might be due to result sample differences between the experiments. Dietrich et al. used a mixed sample of participants (63% female) as opposed the all-female sample employed in the current study. Behavioural data suggest that females show enhanced language-based abilities, especially during episodic memory tasks of word stimuli (Herlitz, Airaksien, & Nordstrom, 1999; Herlitz, Nilsson, & Backman, 1997; Maitland, Herlitz, Nyberg, Backman, & Nilsson, 2004). Relative to males, females are known to evoke greater ERP amplitudes to unpleasant and high arousing stimuli (Lithari et al., 2010). Further, all of Dietrich et al.’s MDD participants were currently being treated with “psychotherapy” in an inpatient facility. Lastly, it is possible that the self-referential encoding strategy (outlined in Study 1) may have resulted in more efficient retrieval, which in turn improved source memory accuracy in the MDD group (Dulas, Newsome, & Duarte, 2011). Dietrich et al. did not employ a self-referential study phase in their Old/New task. This may have contributed to the observation of
impairment in their MDD group, as it might be argued that stimuli were more superficially encoded (Dulas et al., 2011). Thus, the implementation of effective encoding strategies, like self-referential encoding used in the current experiment appears to result in more efficient memory functioning in depression. Although requiring more thorough examination, this strategy suggests treatment implications for depressed female patients.

The results of the study partially supported the predictions of hypothesis 5 for the LPN data. Recall, the LPN represents sustained evaluation of task-relevant attributes and reconstruction of memory. It is largest when stimulus details are not readily recovered during retrieval processes (Johansson & Mcklinger, 2003). Based on predictions of mood-congruent memory processing (Bower, 1981), it was anticipated that larger LPN would be evoked for negative than positive old items in the control participants. The data supported this expectation. Supportive of the memory search account of this ERP, this effect was not evident for the new stimuli (Johansson & Mecklinger, 2003; Johnson et al., 2004). Across all participants, larger LPN for negative than positive old stimuli was found to correlate with greater recognition and free recall of positive material and with greater self-reported depressive, rumination, and anxiety scores. However—similar to the FN400 and LPC data—no significant LPN effects were found in the MDD or remitted groups.

The fact that memory biases for the MDD participants were observed for the behavioural data and not the electrophysiological (FN400, LPC, LPN) may be due to the possibility that the ERP only analysed correct recognition responses, whereas, the behavioural data measured errors of commission and omission. This is a potential limitation in ERP studies as most Old/New studies, are based on analysis of successful source memory only. The present study only analysed correct responses so as to
replicate previous work (e.g., Dietrich et al.), and also to reduce the testing time of the experimental task (as this study was the body of three ERP tasks: thereby reducing potential fatigue effects on the data, Luck, 2005). Unfortunately error rates were too few to support supplementary analyses here. Future experiments could examine this possibility by increasing error rates or test trials.

4.4.2. Self-schema Activation/N400 Semantic Incongruence Effects

A summary of resultant data for hypothesis 6 is shown in Table 32. It is important to note that the N400 results presented here were obtained from correctly classified stimuli during the Old/New task. It is argued that this ERP does not represent the neural correlate of the well-studied FN400 Old/New effect. However, unlike the FN400 Old/New effect, which tends to be manifested over frontal regions and is larger for old then new stimuli, the distribution of the present N400 was primarily at central and parietal sites and failed to distinguish between novelty conditions. Therefore, it is argued that the extracted ERP has more in common with the N400 elicited in traditional semantic incongruence paradigms (see Section 2.2., Table 1). It is thus interpreted as a function of semantic expectancy/incongruence.

Table 32

Summary of the data for Self-Schema Expectancy Effects (Hypothesis 5): Study 2

<table>
<thead>
<tr>
<th>Cognitive Process</th>
<th>Between group Differences</th>
<th>Within participants Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypothesis 6</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semantic Congruency (N400)</td>
<td><strong>Hypothesis 5(a)</strong></td>
<td>Smaller N400s (less negative µV) for negative compared to positive stimuli.</td>
</tr>
<tr>
<td></td>
<td>The MDD participants exhibited significantly larger N400s for positive than negative stimuli.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control participants exhibited a trend of larger N400s for negative compared to positive stimuli.</td>
<td></td>
</tr>
</tbody>
</table>
As predicted in hypothesis 6, MDD participants exhibited larger N400s for positive than negative stimuli. These results are consistent with those of Dietrich et al. (2000) who also observed smaller N400 ERPs for negative words compared to positive words in their depressed participants. It may represent a neural correlate of depressed participants’ negative rumination. Supportive of this, this N400 effect correlated with participants’ self-reported rumination levels. It was also associated with greater endorsement of negative stimuli as self-descriptive during self-referential encoding (Study 1 data), greater free-recall and recognition memory bias for negative stimuli, and greater severity of current and one-month depression and anxiety. This pattern of results supports the traditional models of depression (Beck, 1967; Joormann et al., 2007). However, this effect is purely correlational and thus, no causational claims can be made. It would be beneficial to explore this ERP during experimental manipulations of rumination.

Neither LPN nor N400 were modulated by the valence of the stimuli for the remitted participants. It is possible that the LPN and N400 amplitudes are less sensitive to slight differences in emotional incongruence (Deveney & Pizzagalli, 2008; Schirmer, Kotz, & Friederici, 2002; Schirmer, Tang, Penney, Gunter, & Chen, 2005). That is, the remitted participants possess less emotionally skewed cognitive schemas compared to the control (less positive) and MDD (less negative) participants. Thus, one may anticipate that they would show similar encoding, memory recall (LPN) and expectation (N400) towards both positive and negative stimuli. Alternatively, it may be the small sample size of this group rendered experimental power too low to expose these subtle ERP effects. Future replication of the above findings in a larger sample of remitted depressed participants is thus required.

4.4.3. Summary of Support for Emotional Memory Biases in Depression
In summary, the results of the current experiment replicated previous behavioural evidence of mood-congruent memory effects in free-recall. Unexpectedly, all participants recognised more positive than negative words; with remitted participants also recognising more positive words than the MDD participants. This might reflect use of pleasant memories as an emotional regulation strategy to maintain positive moods (e.g., control, remitted participants) or to regulate or reverse unpleasant mood states (e.g., MDD participants). Control participants showed greater LPNs for negative relative to positive words, suggestive of greater difficulty recalling negative information in this group. No LPN valence effects were found for the remitted participants. This may be the result of their less emotionally skewed cognitive schemas resulting in equal encoding of positive and negative stimuli, or low experimental power. No divergent group ERP data were found for the FN400 or LPC. These null electrophysiological findings likely indicate a Type II error due to methodological issues, such as reduced opportunity for memory decay or ease of the recognition task. Alternatively, it is possible that the self-referential encoding strategy (Study 1) may have resulted in more efficient retrieval, which in turn improved source memory accuracy in the MDD participants. The MDD participants exhibited larger N400s for positive than negative stimuli, which may represent a neural correlate of negative rumination. Contemporary models predict that these emotional memory biases and negative event expectations in depression are the result of a deficiency in prefrontal inhibitory control (Joormann et al., 2007). The next chapter of this dissertation specifically explores working memory inhibitory control processing for emotional stimuli in the same sample of participants utilised in the current study.
Chapter 5

Study 3 – Impaired Working Memory Inhibitory Control in Depression

Definition of Terms

Cognitive Control: An executive process defined as identifying and evaluating the multiple responses during task completion and subsequently resolving such conflict and guiding behaviour towards intended goals.

Cognitive Inhibition: The ability to restrict or remove stimuli or mental processes from working memory that are irrelevant to the task at hand.

Conflict Monitoring: An early sub-component of cognitive inhibitory control that involves the identification and evaluation of multiple responses during task completion. It is has been associated with enhanced Nogo-N2 ERPs.

Conflict Regulation: A sub-component of cognitive inhibitory control in which centres responsible for top-down inhibitory cognitive control (e.g., ACC) activate to reduce the impact that distracting/conflicting information has on task performance. Specifically, it is the process by which this conflict is resolved and behaviours are guided towards intended goals. It has been associated with enhanced Nogo-P3 ERPs.

Go/Nogo Task: An experimental task typically used to assess behavioural and/or cognitive inhibitory control. It involves the presentation of a series of Go and Nogo cues. Participants are required to respond as quickly as possible to Go cues and to inhibit responses to Nogo cues.

Negative Affective Priming Task: An experimental task typically used to assess emotional information processing. In the NAP task, two consecutive trials are presented to participants; each contains a target and a distracter stimulus (e.g., positive, negative, or neutral facial expressions). For each display, participants are required to indicate the emotion of the target and ignore the distracter. A negative bias is indicated by faster response latencies to negative targets that follow negative distracters on a previous trial.

Resistance to Distractor Interference: Denotes a form of cognitive inhibitory control that resists or resolves interference from information in the external environment that is irrelevant to the current task.

Resistance to Proactive Interference: Denotes a form of cognitive inhibition, which involves the ability to resist or remove memory intrusions that were once relevant, but have since become irrelevant to the current task (Friedman and Miyake, 2004).

Rumination: Focused attention and thoughts about the causes and consequences of one’s distress.

Sternberg Working Memory Task: An experimental task used to index working memory storage and updating processes. Participants encode a number of items into
short term memory. A probe item is subsequently presented to which participants are to indicate if it was or was not presented in the preceding memory set.

**Stroop Task:** An experimental task typically used to measure attention control and flexibility. It involves the presentation of a series of colour names in different coloured ink. Participants are required report ink colour of each word; that is, they are required to manage their attention to inhibit or override one response (reading) in order to perform the other (report ink colour). The task takes advantage of the observation that word reading is an automatic ability that interferes with colour naming when a colour word is printed in a different colour (i.e., word red printed in green).

**Working Memory:** A system responsible for the temporary holding and processing of information. This can include new and already stored data. It includes three main components, (1) the central executive, which acts as a supervisory system and controls the flow of information to and from its slave systems the (2) phonological loop and (3) visuospatial sketchpad. These slave systems are said to be short-term storage systems dedicated verbal and visuospatial domains, respectively. The central executive is similar to the construct of a supervisory attentional system regulating thought and goal setting and to the construct of attentional control.

**Working memory Updating:** The ability to manipulate and create new representations in working memory to reflect task demands.

### 5.1. Background and Hypotheses

There were three aims in this study. The first was to explore current and depressed participant’s ERP profiles during early (N2) and late (P3, LPP) cognitive inhibition of emotional material during a modified Sternberg working memory Go/Nogo task. The second sought to test if participant profiles differed in the context of the inhibitory control process examined: resistance to distractor interference versus resistance to proactive interference. The third was to test the correlated predictions of Joormann et al.’s (2007) impaired cognitive control model of depression, such as the associations between participants’ inhibitory control deficits with their self-reported depression severity, rumination, and emotional regulation. The findings of this study may better help inform how working memory control and emotional regulation deficits interact in depression disorders, and how they are/or if they are amended with remission. This may help inform treatment interventions and future research avenues.
5.1.1. **Study background.** The current study examined inhibitory control in the context of both (1) resistance to distractor interference inhibitory control: Controlling the access of information in working memory; and (2) resistance to proactive interference inhibitory control: Updating the contents of working memory once information is no-longer deemed relevant (Miakaye & Friedman, 2004).

Joormann et al. (2007) outlined a model of depression that implicates impaired ability to exert these two forms of inhibitory control over the contents of working memory. They argue that this deficit results in sustained activation of irrelevant negative stimuli in consciousness (working memory), leading to rehearsal of this material and its enhanced long-term memory. Poor inhibitory control is thereby held to deplete the depressed individual’s cognitive resources, limiting their ability to reappraise or recall positive information to regulate their affect. Together this serves to sustain and exacerbate depressed mood. The current empirical evidence of working memory inhibitory control in the context of current and remitted depression is outlined in Section 2.3.3. This data is briefly reviewed in the following section.

Researchers have developed a number of experimental tasks to measure cognitive inhibition. Details of some of these key tasks and their ERP components are outlined in Table 2 (see Section 2.2.1). It is important to note that previous work has found that depressive deficits are more readily observed when cognitive load is low in these tasks (Joormann et al, 2010; Hertel, 1997). According to Hertel (2004), low demand tasks enable rumination and mind wandering and, therefore, are sensitive to individual differences in cognitive control. For instance, Berman et al. (2011) found fMRI evidence of ineffective frontal activity in their MDD group during working memory updating under conditions of low task demand.
5.1.1.1. Resistance to distractor interference inhibitory control. Previous work suggests that current and remitted depression involves deficits in the inhibition of irrelevant information from entering working memory. This effect appears specially enhanced for negative information (see Section 2.3.3.1. for a thorough literature review). Vanderhasselt and De Readt (2009) provided ERP evidence that distractor interference inhibitory control deficits are moderated by depressive history. The researchers used a word-colour Stroop task. Vanderhasselt and De Readt observed larger N450d ERPs were observed for incongruent compared to congruent trials in the sample of never depressed participants. The authors argued that this represented normal working memory inhibitory functioning. However, this N450 effect was not observed in their remitted depressed participants. This appeared reflective of remitted depressed participants’ deficits inhibiting negative information from entering working memory. This ERP finding correlated with number of participant’s previous depressive episodes ($r = .51$), but not with age of depressive onset or duration of remission. These results suggest that deficits in the ability to keep irrelevant material from entering working memory are enhanced with each successive depressive episode. Interestingly, the researchers found no behavioural evidence for this ERP effect. This might reflect the unique advantage of ERP to detect rapid cognitive control processes that are not always evident in overt behaviour.

In their fMRI investigation of remitted depression, Kerestes et al. (2012) found remitted depression to be related to greater neural activity in the dorsal-lateral PFC (DLPFC) during completion of an n-back task with negative emotional distractors. The DLPFC appears implicated in directing attention away from task-irrelevant emotional distractors (Dolcos & McCarthy, 2006; Kerestes et al., 2012), a key task of emotional regulation (Phillips, Ladouceur, & Drevets, 2008). These abnormalities
were found predominately in the left hemisphere and occurred in the absence of any distinct group differences in performance. Thus, it is possible that remitted depressed participants need to call on greater neuro-inhibitory resources for the purposes of stopping a prepotent response to negative stimuli with the same level of skill as the control participants.

The Stroop task (Etkin, Egner, & Peraza, 2006), affective interference task (Siegle, Ingram, & Matt, 2002), negative affective priming task (Yao et al., 2010), and other emotional conflict tasks (Fales et al., 2008) are well-validated protocols for measuring cognitive inhibition (see Table 2, Section 2.2.1.). However, they are limited in their ability to extract effects of early and late inhibitory processes. To address this issue, a modified version of the emotional Sternberg working memory task with Go/Nogo responses was created (see Section 5.1.2. for an overview of this task).

The Go/Nogo task reliably evokes the Nogo-N2 and Nogo-P3 ERPs (Falkenstein et al., 1999). The Nogo-N2 is said to reflect early inhibitory processes, most likely signifying the conflict monitoring subsystem. The Nogo-P3 on the other hand is said to reflect a later cognitive inhibition subsystem, namely conflict regulation processing (Krompinger & Simons, 2011). Research in the normative population suggests that affective and arousing information in Go/Nogo tasks show similar demands on inhibitory control in healthy samples (Chiu et al., 2008). As a result, similar Nogo-N2 and Nogo-P3s have been observed for positive and negative stimuli (Chiu et al., 2008). To this PhD candidate’s knowledge, there are no prior ERP studies of current or remitted MDD using an emotional Go/Nogo task. Below is an outline of the current research from non-emotional Go/Nogo tasks.

The ERP literature for distractor interference inhibition deficit in current and remitted depression shows mixed results. Using a Go/Nogo paradigm of neutral
auditory tones, Kaiser et al. (2003) found depressed patients to make more errors of commission for Nogo stimuli in comparison to the non-depressed participants. They also exhibited early frontal-temporal negativity in the N2 time window, whereas the control participants exhibited a polarity-inverted N2. The researchers suggested that this ERP profile was indicative of deficits in the early inhibitory detection system in the depressed participants (impaired conflict monitoring). The polarity-inverted N2 is not compatible with previous reported morphology of the Nogo-N2, which tends to show negative amplitude (Falkenstein et al., 1999). It is possible that the differing results are due to different reference points, with studies that show the typical negative Nogo-N2 using either linked mastoid or earlobe reference points (e.g., Zhang et al., 2007). Kaister et al. (2003) on the other hand, used an average reference procedure. No group differences were found for the P3 time-window for the Nogo or Go stimuli.

In a sample of partially remitted depressed patients, Ruchsow et al. (2008) found no differences in the Nogo-N2 component but reductions in the frontal Nogo-P3 during completion of a Go/Nogo task. They argued that this indicated that remitted depression shows no abnormalities in early inhibitory (conflict monitoring) processing. However, they show cognitive inhibitory deficits in conflict regulation processing. It is important to note that the participants in Ruchsow et al.’s study were only in partial remission from depression. Thus, it is problematic to generalise their data to completely remitted depressed samples.

Theory and research suggests that poor inhibitory control over irrelevant negative information sustains depression as a result of increased rumination on this stuck pessimistic content (Joormann et al., 2007; Joormann & Gotlib, 2008). It would thus be anticipated that ERPs reflective of sustained elaboration or cognitive reappraisal deficits would be enhanced for negative relative to positive interference.
material in MDD samples. The LPP might be a key component here; it indexes increased sustained attention towards emotional stimuli (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Foti & Hajak, 2008; Schupp, Junghofer, Weike, & Hamm, 2003). Enhanced LPP amplitudes (during the 1000 – 2000 ms epoch) have been associated with distracting aversive items in non-clinical samples (MacNamara, Ferri, & Hajack, 2011). To date, no research has explored the LPP using the Go/Nogo task.

5.1.1.2. Resistance to proactive interference inhibitory control. People with depression show specific difficulties expelling irrelevant negative information from working memory (see Section 2.3.3.2. for a thorough literature review). For instance, Joormann and Gotlib (2008) utilised a modified Sternberg working memory task (Oberauer, 2001, 2005a, 2005b) to assess resistance to proactive interference for emotional information in depression. Their findings suggested that clinical depression was associated with specific difficulties in removing irrelevant negative information from working memory. Hierarchical regression analysis found that these negative proactive inhibitory control intrusion effects predicted self-reported rumination levels. Berman et al. (2011)’s fMRI study suggested that individuals with depression are associated with poor recruitment of the prefrontal regions to inhibit negative proactive interference stimuli. They also found this cognitive control deficit to be associated with rumination scores. These patterns of results are consistent with Joormann et al.’s (2007) impaired cognitive control model of depression.

Reduced ability to inhibit goal-irrelevant negative information has been found in remitted depression (Bhagwagar & Cowen, 2008; Joormann & Gotlib, 2007; Vanderhasselt et al., 2012). Using a cued emotional conflict task, Vanderhasselt et al. (2012) found their remitted depressed participants exhibited slower reaction times to cognitive inhibition for sad facial expression compared to positive facial expressions.
using a cued emotional conflict task. This effect was complemented by contrasting ERP data with remitted participants exhibiting reduced N450 difference scores when asked to disengage from sad facial stimuli. This effect was not evident for the happy stimuli. Enhanced N450 amplitudes in the context of this task has been interpreted to reflect the appropriate recruitment of cognitive control processing to overcome interference from conflicting mental representations (Vanderhasselt et al., 2013; West & Alain, 2000). Source localisation analyses identify regions within the ACC as the potential neurogenerators of this N450 effect (Miniussi, Ruzzoli, & Walsh, 2010). These cognitive deficits emerged in the absence of a mood priming manipulation. This supports the notion that individuals in complete remission from depression have relatively poor cognitive control in response to negative information, which may make them at increased risk for future depressive episodes (Berman et al., 2011; Joormann et al., 2007; Vanderhasselt et al., 2012).

To this PhD candidate’s knowledge, no previous ERP study has examined MDD in the context of proactive interference inhibitory control for emotional material. Further, no prior investigations have directly compared MDD participants with remitted depressed participants for proactive interference inhibitory control. None have examined these processes using the Go/Nogo task in remitted or MDD samples. Thus, the associated electrophysiological indices of conflict monitoring (Nogo-N2) and conflict regulation (Nogo-P3) processes for proactive inhibitory control deficits are relatively unknown. To fill these gaps, the present study investigated cognitive control for negative and positive proactive interference in a sample of participants with current and remitted depression, and never depressed controls.

### 5.1.2. Overview of methodological task.

In an attempt to measure distractor interference and proactive interference inhibitory control within the one task, a
modified version of the Sternberg working memory task employed in Joormann and Gotlib (2008; see section 2.3.3.2.) was used. It was tailored so that it could be conducted with ERP methods. Similar to the protocol of Joormann and Gotlib, participants were required to study two lists of words that both included a mix of positive and negative adjectives. Participants were then told to remove one list from memory (requiring working memory updating) whilst holding the other list in memory. Participants then completed a recognition test, in which they were required to indicate if a series of presented words were included in the list that they were informed to keep in memory. If the word was from this relevant list, they were told to press a Yes key as fast as possible. They were required to inhibit responses to all other stimuli. Nine stimuli were presented here: three Go stimuli, three distractor interference Nogo stimuli, and three proactive interference Nogo stimuli. Recall from Study 2 (Chapter 4) that the relative ease of the recognition ERP task did not require excessive involvement of the central executive which likely resulted in the null ERP findings. Thus, it was important that the current study did not exhibit the same methodological issue by effectively engaged this system (Hertle, 2004). Given that the present modified working memory Sternberg task required participants quickly update the content of their working memory every 30 seconds and make quick responses, it is argued to that this required moderate cognitive load. Table 33 presents the operational definitions for these three stimuli conditions. As ERP investigations require a number of stimulus trials to obtain averages with good signal-to-noise ratios (Luck, 2005), the task was completed 20 times. This allowed for 30 positive and negative stimuli trails per Go, distractor interference Nogo, and proactive interference Nogo conditions.
Table 33

Operational Definitions for Go Stimuli, Distractor Interference Nogo Stimuli, and Proactive Interference Nogo Stimuli in the Modified Sternberg Go/Nogo ERP Task

<table>
<thead>
<tr>
<th>Stimuli Type</th>
<th>Operational Definition</th>
<th>Response Demand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Go Stimuli</td>
<td>Stimuli that participants had just encoded during a learning task, and were subsequently informed to maintain in working memory. They were informed to respond to these stimuli in later recognition testing.</td>
<td>Yes – participants were required to press the Yes key in response to presentation of these stimuli during the recognition task.</td>
</tr>
<tr>
<td>Distractor Interference Nogo Stimuli</td>
<td>Stimuli that participants had not previously encoded into working memory (never seen before stimuli). They were not required to make key press responses to presentation of this stimulus during recognition testing.</td>
<td>No – participants were required to inhibit response to presentation of these stimuli during the recognition task.</td>
</tr>
<tr>
<td>Proactive Interference Nogo Stimuli</td>
<td>Stimuli that participants had just encoded during a learning task, and were subsequently informed to remove from working memory. They were informed to not respond to these stimuli in later recognition testing.</td>
<td>No – participants were required to inhibit response to presentation of these stimuli during the recognition task.</td>
</tr>
</tbody>
</table>

5.1.3. Study hypotheses. The following hypotheses were made.

5.1.3.1. Resistance to distractor interference inhibitory control.

Hypothesis 1(a) predicted a group-by-valence interaction. Here MDD participants were expected to show significantly larger error rates for distractor interference Nogo stimuli compared to the control and remitted group. This group effect was expected to be specifically enhanced for the negative stimuli. It was tested with a 3 (group) × 2 (valence) ANOVA. Planned comparisons between positive and
negative stimuli were conducted for each group. Hypothesis 1(b) further predicted MDD participants would show greater errors for negative than positive distractor interference Nogo stimuli. This was tested with a planned $t$-test.

Hypothesis 1(c) predicted that across all participants, greater errors for negative than positive distractor interference stimuli (distractor interference Nogo error bias scores) would positively correlate with self-reported depressed mood, rumination, and suppression scores, and negatively correlate with emotional regulation scores. Consistent with Joormann et al.'s (2007) model, positive correlations were also expected between the distractor interference negative error bias scores with participants P3b negative bias score (data derived from Study 1). It would also positively correlate with the N400 negative bias score, free-recall negative bias score, and their recognition memory negative bias scores (data derived from Study 2).

Hypothesis 2 predicted a group-by-valence-by-laterality interaction for the distractor interference Nogo-N2 data. Simple main effects should be observed for the remitted and MDD participants, with both expected to exhibit greater Nogo-N2s for negative than positive stimuli. For the remitted group, this effect would be localised to the left electrodes. No valence effects were expected for the control participants. The hypothesis was tested with a 3 (laterality) × 2 (valence) × 3 (group) factorial ANOVA. The predicted three-way interaction will be analysed by looking at the interacting effects of valence and group at each lateral position separately. Post-hoc analysis used Bonferoni correction to reduce the inflation of Type I error with multiple comparisons. Hypothesis 2(b) predicted positive correlations between the greater Nogo-N2 for negative relative to positive distractor interference stimuli (Nogo-N2 DI bias scores) and participants’ level of rumination and sustained depression.
Hypothesis 3 (a) expected a group-by-valence-by-laterality effect for the distractor interference Nogo-P3 data. Simple main effects should find the remitted group to have significantly larger Nogo-P3s for negative relative to positive stimuli over left and midline sites. A same statistical analysis protocol explained in Hypothesis 2 was employed. Hypothesis 3(b) predicted positive correlations between the greater Nogo-P3 for negative relative to positive distractor interference stimuli (Nogo-P3 DI bias scores) and participants’ self-reported level of rumination and sustained depression.

Hypothesis 4 was exploratory in nature. It expected a group-by-valence effect for the distractor interference Nogo-LPP data. (a) Reflective of cognitive reappraisal strategies to maintain their euthymic mood, simple main effects should find the remitted group to have significantly smaller Nogo-LPPs for negative material relative to the MDD participants. (b) While reflective of their poor emotional reappraisal, the MDD group will show larger Nogo-LPPs following negative relative to positive distractor interference stimuli. These predictions were analysed with a 2 (Valence) x 3 (Group) ANOVA, with Bonferroni corrected post-hoc comparisons between valence conditions for each group separately.

5.1.3.2. Resistance to proactive interference inhibitory control.

Hypothesis 5(a) predicted a group-by-valence interaction. Here MDD participants were expected to show significantly larger error rates for proactive interference stimuli compared to the control and remitted groups. This group effect was expected to be specifically enhanced for the negative stimuli. It was tested with a 3 (group) × 2 (valence) ANOVA, followed by planned comparisons of group for positive and negative stimuli separately. Hypothesis 5(b) further predicted MDD
participants to show greater errors for negative than positive proactive interference stimuli. This was tested with a planned \( t \)-test.

*Hypothesis 5(c)* predicted that across all participants, greater errors of commission for negative than positive proactive interference stimuli (proactive interference negative error bias scores) was expected to be positively correlated with self-reported depressed mood, rumination, and suppression scores, and negatively correlated with emotional regulation scores. Consistent with Joormann et al.’s (2007) model, positive correlations were also expected between the distractor interference negative error bias scores with participants P3b negative bias score (data derived from Study 1). It would also positively correlate with the N400 negative bias score, free-recall negative bias score, and their recognition memory negative bias scores (data derived from Study 2).

*Hypothesis 6(a)* predicted a group-by-valence interaction for the proactive interference Nogo-N2 data. A simple main effect should be observed for the MDD participants for the negative stimuli, with them showing larger Nogo-N2 to the negative relative to the positive stimuli. *Hypothesis 6 (b)* further predicted MDD participants to show significantly larger Nogo-N2 than the remitted or control participants. These predictions were analysed with a 2 (valence) \( \times \) 3 (group) ANOVA, followed by planned comparisons of group for positive and negative stimuli separately. *Hypothesis 6(c)* predicted positive correlations between the greater Nogo-N2 for negative relative to positive proactive interference stimuli (Nogo-N2 PI bias scores) and participants’ self-reported level of rumination and sustained depression.

*Hypothesis 7(a)* predicted a group-by-valence interaction for the proactive interference Nogo-P3 data. A simple main effect should be observed for the MDD participants for the negative stimuli, with them showing larger Nogo-P3 to the
negative relative to the positive stimuli. *Hypothesis 7 (b)* further predicted MDD participants to show significantly larger Nogo-P3 than the remitted or control participants. These predictions were analysed with a similar protocol outlined in Hypothesis 6. *Hypothesis 7(c)* predicted positive correlations between the greater Nogo-P3 for negative relative to positive proactive interference stimuli (Nogo-P3 PI bias scores) and participants’ self-reported level of rumination and sustained depression.

*Hypothesis 8* was exploratory in nature. It expected a group-by-valence effect for the proactive interference Nogo-LPP data. *(a)* Reflective of cognitive reappraisal, simple main effects should find the remitted group to have significantly smaller Nogo-LPPs for negative material relative to the MDD participants. While reflective of their poor emotional reappraisal strategies, *(b)* the MDD group were expected show larger Nogo-LPPs following negative relative to positive distractor interference stimuli. These predictions were analysed with a 2 (Valence) x 3 (Group) ANOVA, with Bonferoni corrected post-hoc comparisons between valence conditions for each group separately.

### 5.2. Method

#### 5.2.1. Participants

In the pilot study, medium effect sizes were observed for the hypothesized Nogo-N2 and Nogo-P3 data using a sample of 20 participants per group (see overview of these results in Appendix D). In their Go/Nogo task, Kaiser et al. (2003) observed reduced Nogo-N2 ERPs in a sample of 16 depressed participants (*p* <.01). They analysed their results using a 3-way Factorial ANOVA. Vanderhasselt et al., (2012) observed reduced N450 amplitudes on trials requiring disengagement from negative distractor items in their sample of 15 remitted-depressed participants. Their results
were also analysed using a mixed ANOVA approach, in which this significant valence-by-group interaction showed a large effect size ($\eta^2 = .20$). Thus, a sample of approximately 15 participants per experimental group was deemed to be sufficient to detect the hypothesized effects using mixed ANOVA.

Participants of this study consisted of the same sample of 73 female university students who were recruited in Studies 1 and 2 (see section 3.2.1). As in both of these studies, participants were placed into one of three groups according to their SCID-I/NP diagnoses:

1. *Current depressed* group (MDD; $n = 30$).
2. *Remitted depressed* group (remitted; $n = 13$).
3. *Healthy control/never depressed* group (control; $n = 30$).

See Section 3.3.2. for data on participant characteristics.

5.2.2. Materials

**Clinical and Psychometric assessments.** The utilised self-report tests and structured clinical interviews are outlined in Section 3.2.2. of this dissertation.

**Experimental stimuli.** The study used the original 45 positive and 45 negative adjectives presented in Study 1 and Study 2 to form the 60 (30 positive and 30 negative) proactive interference Nogo stimuli. The remaining 30 items made up 50% of the Go stimuli. These previously viewed words were not used for the distractor interference Nogo list. Instead, the study included another 45 positive and 45 negative adjectives that participants had not seen prior (i.e., they were not used in Study 1 or 2). These new words formed the 60 (30 positive and 30 negative) distractor interference Nogo stimuli. The remaining 30 items here made up the other 50% of the Go stimuli. These new words were not used for the proactive interference Nogo list. Further
details on the selection process of experimental stimuli and their properties (e.g., familiarity and valence ratings) can be viewed in Section 3.2.2. of this dissertation.

**EEG recording apparatus.** The same EEG recording apparatus utilised in Study 1 and 2, and explained in detail in Section 3.2.2. of this dissertation was employed.

### 5.2.3. Procedures

**EEG recording and data reduction.** The same EEG recording and data reduction procedures utilised in Study 1 and 2, and explained in detail in section 3.2.3. of this dissertation, was employed.

**Statistical analyses.** The same statistical analysis protocol for the ANOVA and correlation analyses utilised in Studies 1 and 2, outlined in Section 3.2.3.5. of this dissertation, was employed to test the main effect, interaction, and correlation predictions.

**ERP task: the modified Sternberg Go/Nogo task.** A modified version of the Sternberg working memory task developed by Oberauer (2001, 2005a, 2005b) that required Go/Nogo responses was used. Appendix V outlines participant task instructions. The task was modified so that it could be conducted with ERP methods. Participants were presented with a *learning display* of two lists of three words each (both presented simultaneously as two vertical lists on the screen). One list was printed in red ink and the other list in blue ink. Participants had 7.8 seconds to memorise each of the word stimuli (1.3 seconds per word; Joormann & Gotlib, 2008). The lists then disappeared, and the screen went black for 800 ms. A coloured frame then appeared around the screen. The frame was either red or blue in colour (randomised). The frame was used to cue participants as to which list to remember and respond to in the following task. If it was blue, the participant was required to
remember and respond to the words that were just previously presented in the blue list in the learning display. If it was red, they were required to remember and respond to the words that were just previously presented in the red list in the learning display. Shortly after the coloured famed appeared, a Bernoulli presentation of the nine stimuli were individually shown, all words were now printed in white ink. These included three Go stimuli, the three proactive interference Nogo stimuli, and three distractor interference Nogo stimuli (the never-seen before items). Prior to each word, a small white dot appeared on the computer screen for 1500 ms, which would cue participants that a new stimulus trial was about to begin. A word was then presented for 300 ms, after which the screen went black for 2200 ms. Like in other ERP Go/Nogo investigations, participants were required to press the ‘Yes’ key for the cued words, which constituted the Go trials. They are required to inhibit their responses to the Nogo words (e.g., Chiu et al., 2008; Ruchsow et al., 2008). This included inhibiting responses to words which participants just encoded then told to remove from working memory: Proactive interference Nogo stimuli, and to words which were presented for the first time: Distractor interference Nogo stimuli. A visual illustration of this task, the stimulus presentation rate and ISI are shown in Figure 41.
Nogo Proactive Interference Condition
(Word from Learning List which was required to be removed from memory; participants were not required to respond to this word during the recognition task)

Go Condition
(Word from Learning List which was required to be held in memory; participants were required to make a key press response to this word during the recognition task)

Nogo Distractor Interference Condition
(Word not previously encoded into memory; participants were not required to respond to this word during the recognition task)

Figure 41. Schematic depiction of presentation timings in for the modified Sternberg working memory Go/Nogo task. Note. Nine presentations of word stimuli were shown during the recognition task. This included 3 Go stimuli, 3 proactive interference Nogo stimuli, and 3 distractor interference Nogo stimuli.
To reduce any cortical activation due to the motor responses, participants completed the first half of the investigation with one hand and the second half with the other hand. This task was repeated 20 times, giving a total of 180 trials, allowing 30 trials per condition and valence category. Each word was presented only once during the task; taking approximately 25 minutes to complete.

**Statistical Analyses**

The same statistical analysis protocol for the ANOVA and correlation analyses utilised in Study 1 and 2, outlined in Section 3.2.3.5. was used.

**5.3. Results**

To reduce inflation of Type I error, only results that pertain to the research hypotheses or to the key variable group are presented.

**5.3.1. Behavioural Data**

**Participant performance (error rates).** Table 34 outlines the error rates for go (errors of omission), and the distractor and proactive interference nogo stimuli (errors of commission) for positive and negative words across the experimental groups. As can be seen, error rates were relatively low across all conditions.

*Errors of omission: Go condition.* A 3 (group) × 2 (valence) ANOVA was conducted on participants’ percentage errors for the go condition. No group differences were found for overall errors, or interactions with valence. The effect of valence was also not significant.
Table 34

Mean Percentage of Error Rates (SEM in parentheses) for the Go/Nogo Task

<table>
<thead>
<tr>
<th>Errors of Omission: Go Condition</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>25.56 (2.67)</td>
<td>20.33 (2.46)</td>
</tr>
<tr>
<td>Remitted</td>
<td>19.49 (2.74)</td>
<td>22.05 (2.22)</td>
</tr>
<tr>
<td>MDD</td>
<td>23.56 (1.92)</td>
<td>21.56 (2.34)</td>
</tr>
</tbody>
</table>

Errors of Commission: Distractor Interference Nogo Condition

<table>
<thead>
<tr>
<th>Control</th>
<th>2.11 (0.44)</th>
<th>1.22 (0.47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remitted</td>
<td>3.59 (1.22)</td>
<td>2.31 (1.16)</td>
</tr>
<tr>
<td>MDD</td>
<td>2.56 (0.63)</td>
<td>2.67 (0.67)</td>
</tr>
</tbody>
</table>

Errors of Commission: Proactive Interference Nogo Condition

<table>
<thead>
<tr>
<th>Control</th>
<th>17.00 (1.70)</th>
<th>15.78 (1.75)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remitted</td>
<td>16.67 (2.26)</td>
<td>15.13 (1.98)</td>
</tr>
<tr>
<td>MDD</td>
<td>17.89 (1.62)</td>
<td>22.44 (2.06)</td>
</tr>
</tbody>
</table>

Note. Maximum score for errors possible is 100.

Errors of commission: Nogo distractor interference condition. To test the predictions of hypothesis 1(a), a 3 (group) × 2 (valence) ANOVA was conducted on participants’ percentage errors for the nogo distractor interference stimuli. The Group×Valence interaction, $F (2, 70) = 1.06, p = .320, \eta^2 = .029$ and the main effects for group and valence were both not significant.

Testing the predictions of hypothesis 1(b), t-test analysis did not find MDD participants to exhibit more errors for distractor interference nogo stimuli for negative than positive stimuli, $t(29) = -0.17, p = .869$. To test the predictions of hypothesis 1(c), Pearson correlation coefficient analyses found greater errors for negative compared to positive distractor interference stimuli (distractor interference negative bias score) did not correlate with the self-reported depression, anxiety, rumination, and emotional regulation or suppression scores. It also failed to correlate with participants’
N400 negative bias scores or their recognition or free recall memory negative bias scores. However, a significant correlation was found between distractor interference negative error bias scores and greater right hemisphere P3b amplitudes for negative than positive stimuli during self-descriptive encoding, $r = .242, p < .05$.

**Errors of commission: Nogo proactive interference condition.** To test the predictions of hypothesis 5 (a), a 3 (group) × 2 (valence) ANOVA was conducted on participants’ percentage errors for the nogo proactive interference stimuli. The Valence × Group interaction was significant, $F(2, 70) = 3.06, p = .027, \eta^2 = .080$, whereas the main effects of valence and group were both not significant. To identify the source of the interaction, univariate ANOVAs were conducted for the positive and negative stimuli, thus, examining the impact of group. No simple main effects were found for the positive proactive interference stimuli. For the negative proactive interference, a simple main effect of group was observed: $F(2, 70) = 4.19, p = .019, \eta^2 = .019$. Planned analyses ($\alpha = .05$) found MDD participants displayed more errors inhibiting negative stimuli compared to both the control ($mean$ difference $= 6.67, p = .018$), and remitted participants ($mean$ difference $= 7.32, p = .046$). No differences were found between the control and remitted participants.

To test the predictions of hypothesis 5(b), $t$-test analyses found MDD participants to exhibit significantly more errors for negative than positive stimuli proactive interference stimuli, $t(29)= 2.24, p = .033$ ($mean$ difference $= 4.56\%$).

To test the predictions of hypothesis 5(c) Pearson correlation coefficient analyses found that greater errors for negative compared to positive proactive interference stimuli (*proactive interference negative bias scores*) positively correlated with current and one-month depression levels, rumination, and self-reported anxiety levels. These results are displayed in Table 35. The proactive interference negative
bias scores was also found to positively correlate with greater recognition memory for negative compared to positive stimuli, \( r = .336, \ p = .004 \), but not with their P3b, N400, or free recall memory negative bias scores.

Table 35

**Correlations between Psychometric scores and percentage Proactive Interference**

**Error Rates for Positive and Negative Stimuli and Negative Bias Scores**

<table>
<thead>
<tr>
<th>Psychometric</th>
<th>n</th>
<th>Positive Stimuli</th>
<th>Negative Stimuli</th>
<th>Negativity Bias Score (Negative-Positive)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI-II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td>73</td>
<td>-.019</td>
<td>.353***</td>
<td>.365***</td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>70</td>
<td>-.045</td>
<td>.218*</td>
<td>.249**</td>
</tr>
<tr>
<td><strong>RRS- Total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Reflective</td>
<td>73</td>
<td>-.122</td>
<td>.140</td>
<td>.237*</td>
</tr>
<tr>
<td>- Brooding</td>
<td>73</td>
<td>.084</td>
<td>.377***</td>
<td>.284**</td>
</tr>
<tr>
<td>- Depressive</td>
<td>73</td>
<td>-.062</td>
<td>.278*</td>
<td>.281**</td>
</tr>
<tr>
<td><strong>ERQ</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Regulation</td>
<td>73</td>
<td>.063</td>
<td>-.174</td>
<td>-.203*</td>
</tr>
<tr>
<td>- Suppression</td>
<td>73</td>
<td>.039</td>
<td>.169</td>
<td>.100</td>
</tr>
<tr>
<td><strong>STAI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- At testing</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Follow-up (1-month)</td>
<td>73</td>
<td>-.102</td>
<td>.177</td>
<td>.283*</td>
</tr>
<tr>
<td></td>
<td>70</td>
<td>-.083</td>
<td>.156</td>
<td>.245*</td>
</tr>
</tbody>
</table>

*Note. * \( p > .05 \) ** \( p > .01 \) *** \( p < .001 \)*

**Reaction time data.** Table 36 shows participants’ mean RT data for correct go stimuli responses for positive and negative stimuli during the modified Sternberg
working memory inhibition task. A 3(group) × 2 (valence) ANOVA found null results for the effect of group, valence, and their interaction.

Table 36

Mean Reaction Times (ms; SEM in parentheses) for Correct Go Stimuli Responses during the modified Working Memory Sternberg Go/Nogo Task: Study 3.

<table>
<thead>
<tr>
<th>Experimental Group</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>661.00 (27.26)</td>
<td>661.24 (30.01)</td>
</tr>
<tr>
<td>Remitted</td>
<td>702.67 (41.41)</td>
<td>668.04 (45.59)</td>
</tr>
<tr>
<td>MDD</td>
<td>748.70 (27.26)</td>
<td>740.84 (30.01)</td>
</tr>
</tbody>
</table>

5.3.2. Electrophysiological Data

5.3.2.1. Preliminary analyses to identify relevant ERP components

See Section 2.2.2. of this dissertation for an outline on the PCA protocol and Section 3.3.1. for an outline of the PCA procedures used for the study. Nogo ERPs differ from go ERPs in terms of both cognitive process and neural generators (Bokura et al., 2001; Pfefferbaum & Ford, 1988). Thus, go and nogo stimuli were analyzed in separate PCAs. Cognitive inhibition engages frontally distributed executive processing; thus, only fronto-central electrode sites were analyzed for nogo stimuli processing. The PCA analysis was completed for the experimental groups separately given that ERP differences are expected between the groups.

Nogo Stimuli PCA

Control group. The PCA consisted of rows of 28 participants (two participants were lost from the original sample of 30 due to error rates >50%), four experimental conditions (valence, Nogo condition), and the six frontal-central electrode sites (six: F3, FZ, F4, C3, CZ, C4). That is, the matrix included a total of 28×4×6 rows existed for
the 400 columns of time samples of EEG. A scree plot indicated that four components should be retained, which accounted for 60.46% of the variance for the Promax rotation. The scree plot displaying the percentage of variance associated with the principal components is shown in Appendix W. In temporal sequence, the approximate peak latency of loadings for each component was: Component 3 at 350 ms; Component 1 at 750 ms; Component 4 at 800 ms; Component 2 at 1600 ms.

The negative peak in the 200-600 ms for Component 2 and 4 appeared consistent with the hypothesized Nogo-N2 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010). This ERP is evident at frontal-central midline sites in the grand average Nogo distractor interference and proactive interference data for the Control participants (see Figures 49 and 53). The positive peak in between 400 – 800 ms extracted in Component, 3, and 4 seems consistent with the hypothesized Nogo-P3 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010). Again, this ERP is visible in this time epoch for the Nogo stimuli at frontal and central sites (see Figure 49 and 53). Components 2 and 4 appeared to represent the sustained positivity of the LPP between 1200 and 2000ms (Hajcak et al., 2010).

Thus, based these PCA results and previous work, the 200-400 ms epoch was used to measure the Nogo-N2, and the 401-800 ms epoch was used to measure the Nogo-P3, and 801– 2000ms to measure the Nogo-LPP. The extracted components are shown in Figure 42.
Figure 42. ERP epochs for Nogo stimuli for the control group.
**Remitted group.** The PCA consisted of rows of 13 participants, four experimental conditions (valence, Nogo condition), and the six frontal-central electrode sites (six: F₃, F₂, F₄, C₃, C₂, C₄). To conform to PCA analysis assumptions the original data was reduced to represent a sampling rate of 400 points per second, reducing the number of variables (columns) from 2000 to 200 by averaging every 10 sequential data points (across 2000 ms)\(^7\). That is, the matrix included a total of 13×4×6 rows for the 200 columns of time samples of EEG. A scree plot indicated that six components should be retained, which accounted for 66.1% of the variance for the Promax rotation. The scree plot displaying the percentage of variance associated with the principal components is shown in Appendix X. In temporal sequence, the approximate peak latency of loadings for each component was: Component 6 at 250 ms; Component 1 at 600 ms; Component 3 at 800 ms; Component 5 at 1000 ms; Component 2 and 4 at 1200 ms.

The negative deflections between 200 ms and 400 ms evident Components 3 and 6 appeared consistent with the Nogo-N2 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010), which seemed evident at frontal-central midline sites in the grand average data for both the distractor interference (Figure 50) and proactive interference (Figure 54) stimuli. The positive peak between 400 -800 ms in Component 1, 3 and 6 appeared consistent with the Nogo-P3 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010). Components 5 and 2 appeared to show correlates of inverted Nogo-P3 during this epoch. Component 2 appeared to represent the sustained positivity of the LPP between 1200 and 2000 ms (Hajcak et al., 2010). Thus, similar to the Control

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\(^7\) Given the smaller sample of remitted participants, for the PCA matrix to be valid, the original data for these the remitted sample was reduced to 200 EEG samples. This is smaller than the adequate number (300) recommended by Tabachnick and Fidell (2005). Thus, care is specifically taken to only analyse extracted components whose morphology is consistent with hypothesized epochs for robust ERPs (e.g., N2, P3, LPP). When more stringent extraction protocols are followed like this previous work has shown that smaller PCA samples will continue to produce statically valid results (Bentler, 2000; de Winter, Dodou, &Wieringa, 2009; Sapan & Zeller, 2002; Zeller, 2005).
participants, the 200-400 ms epoch was used to measure the Nogo-N2, and the 401-800 ms epoch was used to measure the Nogo-P3, and 801 – 2000ms to measure the LPP. The extracted components are shown in Figure 43.

**Component 1**
- Positive peak latency: 600 ms
- 28.78% explained variance

**Component 2**
- Negative peak latency: 700 ms (possible inversion of Nogo_P3)
- Sustained positivity: 1200 - 2000 ms
- 15.67% explained variance

**Component 3**
- Negative peak latency: 420 ms
- Positive peak latency: 800 ms
- 3.99% explained variance

**Component 4**
- Peak latency: 1200 ms
- 5.92% explained variance

**Component 5**
- Negative peak latency: 600 ms (possible inversion of Nogo_P3)
- Positive peak latency: 1000 ms
- 4.61% explained variance

**Component 6**
- Negative peak latency: 250 ms
- Positive peak latency: 400 ms
- 3.55% explained variance

*Figure 43. ERP epochs for Nogo stimuli for the remitted group.*
**MDD group.** The PCA consisted of rows of 30 participants, four experimental conditions (valence, Nogo condition), and the six frontal-central electrode sites (six: F₃, F₂, F₄, C₃, C₂, C₄). That is, the matrix included a total of 30×4×6 rows for the 400 columns of time samples of EEG. A scree plot indicated that six components should be retained, which accounted for 64.87% of the variance for the Promax rotation. The scree plot displaying the percentage of variance associated with the principal components is shown in Appendix Y. In temporal sequence, the approximate peak latency of loadings for each component was: Component 3 and 6 at 350 ms; Component 5 at 400 ms; Component 1 at 600 ms; and Component 2 and 4 at 1200 ms.

The small negative peaks in the 200-400 ms epoch for Component 3, 5, and 6 appeared to represent the Nogo-N2 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010), which seemed evident at frontal-central midline sites in the grand average data (see Figures 51, 55). The positive peak in Component 1, 3 and 4 appeared between 400 - 800ms appears consistent with the Nogo-P3 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010). Components 2 and 4 appeared to represent the sustained positivity of the LPP between 1200 and 2000ms.

Thus, similar to the control and remitted participants, the 200-400 ms epoch was used to measure the Nogo-N2, and the 401-800 ms epoch was used to measure the Nogo-P3, and 801 – 2000ms to measure the LPP. The extracted components are shown in Figure 44.
Component 3
Negative peak latency: 350 ms
Positive peak latency: 650 ms
9.97% explained variance

Component 6
Negative peak latency: 200 ms
Positive peak latency: 350 ms
3.66% explained variance

Component 5
Negative peak latency: 400 ms
Positive peak latency: 780 ms
3.79% explained variance

Component 1
Positive peak latency: 600 ms
29.23% explained variance

Component 4
Positive peak latency: 600 ms
Sustained positivity: 1600-2000 ms
5.12% explained variance

Component 2
Sustained positivity: 1600-2000 ms
13.09% explained variance

Figure 44. ERP epochs for Nogo stimuli for the MDD group.
Go Stimuli PCA

The PCA for the Go stimuli in the inhibition task was completed on the full sample of 71 participants for statistical validity purposes. Specifically, experimental groups could not be analyzed in separate PCAs as the smaller number of experimental conditions resulted in fewer rows than columns, thereby violating statistical assumptions (Tabachnick & Fidell, 2005). The research hypotheses did not pertain to the Go-ERP results; rather Go-ERP results are reported for replication and internal validity purposes. Thus, given that the groups are not expected to produce different factors, pooling their results for the PCA is deemed desirable due to increase in sample size (Tabacknick & Fidell, 2005).

The go stimuli in the inhibition task rows consisted of all participants, two experimental conditions (valence), and the six central-parietal electrode sites (C3, Cz, C4, P3, Pz, P4). That is, the matrix dimensions included a total of 71×2×6 rows for the 400 columns of time samples of EEG. A scree plot indicated that five components should be retained, which accounted for 76.82% of the variance for the Promax rotation. The scree plot displaying the percentage of variance associated with the principal components is shown in Appendix Z. In temporal sequence, the approximate peak latency of loadings for each component was: Component 5, 175 ms; Component 4, 400 ms; Component 3, 800 ms; Component 2, 1150 ms; Component 1, 1600 ms. An inspection of the component loadings and the grand average ERP waveforms (see Figure 57 - 59) indicated that all components primarily represented positive activity.

Between approximately 300 and 800 ms, the PCA solution identified two components: Component 3 (10.42% explained variance), and Component 4 (4.95% explained variance). Both appeared consistent with the Go-P3, observed within this epoch at cento-parietal sites in the grand average data. It is possible that these two
components represent the P3e (early; peak latency of 317 ± 22 ms) and P3l (late; peak latency 651 ± 49 ms) subcomponents of the Go-P3 as identified in Bokura et al. (2001), respectively. Alternatively, they may represent variation in latency of the P3 ERP, possibly reflecting depression’s influence on slowing this components manifestation (P3b, mean Cohen's d = 0.85: Anderer et al., 2002; Kawasaki et al., 2004; Roschke & Wanger, 2003). Component 5 (3.64% explained variance) appeared similar to the P2, showing a positive peak within the 150-300 ms temporal window (Lijffijt et al., 2009). This component was not analyzed as it did not pertain to the study hypotheses. Component 1 appeared to represent the sustained positivity of the LPP between 800 and 2000ms (Hajcak et al., 2010). Thus, the 401-800 ms epoch was used to measure the Go-P3 ERP and 801 ms – 2000ms was used to measure the LPP. The extracted components and the analyzed epochs are shown in Figure 45.
**Component 5**
*Peak latency: 175 ms*  
3.64% explained variance

**Component 4**
*Peak latency: 400 ms*  
4.95% explained variance

**Component 3**
*Peak latency: 800 ms*  
10.42% explained variance

**Component 2**
*Peak latency: 1150 ms*  
27.89% explained variance

**Component 1**
*Peak latency: 1600 ms*  
29.91% explained variance

*Figure 45.* ERP epochs for go stimuli for study 3. Data is based on all participants.
5.3.2.2. Validation of Go/Nogo Task and their evoked ERPs

**Distractor interference stimuli.** Averaged across all participants and frontal electrode sites, a significant effect of condition (go versus nogo) was observed for the Nogo-N2 data, $F(1, 68) = 7.84, p < .001, \eta^2 = .788$. More negative N2s were evoked for distractor interference nogo compared to go stimuli. Figure 46 visually displays these results at electrode site Fz. A significant effect of condition was also observed for the Nogo-P3 data, $F(1, 68) = 2.963, p = .045, \eta^2 = .084$. Smaller frontal P3s were evoked for distractor interference Nogo stimuli compared to go stimuli (see Figure 46). The morphology of this P3 effect is dissimilar to that typically observed in standard Go/Nogo tasks (refer to Figure 10; Section 2.2.); but is similar to the reduced Nogo-P3 observed in studies using depressed and remitted depressed samples (e.g., Kaiser et al., 2003; Rushshow et al., 2008; Section 2.3.3.).

**Figure 46.** Grand average ERP results for nogo distractor interference stimuli and go stimuli (averaged over group and valence) at electrode site Fz. Dotted line refers to stimulus onset.

**Proactive interference stimuli.** Averaged across all participants and frontal electrode sites, a significant effect of condition (go versus nogo) was observed for the
Nogo-N2 data, $F(1, 68) = 7.84, p < .001, \eta^2 = .788$. More negative N2s were evoked for proactive interference nogo compared to go stimuli. Figure 47 visually displays these results at electrode site Fz. A significant effect of condition was also observed for the Nogo-P3 data, $F(1, 68) = 2.963, p = .045, \eta^2 = .084$. Smaller frontal P3s were evoked for proactive interference nogo stimuli compared to go stimuli (see Figure 47). Similar to the distractor interference Nogo data, this morphology of the Nogo-P3 is dissimilar to that typically observed in standard Go/Nogo tasks (confer to Figure 10; Section 2.2.); but is similar to the reduced Nogo-P3 observed in studies using depressed and remitted depressed samples (e.g., Kaiser et al., 2003; Rushshow et al., 2008; Section 2.3.3.).

![Figure 47. Grand average ERP results for nogo proactive interference stimuli and go stimuli (averaged over group and valence) at electrode site Fz. Dotted line refers to stimulus onset.](image)

**Go Stimuli.** Averaged across all participants and central-parietal sites, a significant effect of condition (go versus nogo) was observed for the P3 data, $F(1, 68) = 7.84, p < .001, \eta^2 = .788$. Larger parietal P3s were evoked for go stimuli compared
to the Nogo stimuli (averaged across distractor interference and proactive interference conditions). Figure 45 visually displays these results at electrode site Pz.

*Figure 48.* Grand average ERP results for go stimuli (averaged over group and valence) and nogo stimuli (averaged over group, valence, and condition) at electrode site Pz. Dotted line refers to stimulus onset.

### 5.3.2.3. ERP Results for Distractor Interference Inhibitory Control.

Grand average ERP waveforms for positive and negative distractor interference nogo stimuli for the control, remitted, and MDD participants are shown in Figures 49, 50, and 51, respectively. Visual inspection suggested little difference in frontal or central ERPs evoked for positive and negative stimuli for the control or MDD groups. The remitted group appeared to exhibit larger Nogo-P3 over midline sites for negative than positive stimuli. There is more variability evident in the remitted participants’ grand average waveforms as compared to that in the control or MDD groups. This most likely reflects the remitted groups smaller sample size (13 versus 30) and the heterogeneous nature of these samples in general (see Just et al., 2002 for a review). The LPP waveform is most identifiable in the control participant’s data; however there does not appear to be much difference in magnitude for positive and negative stimuli.
Figure 49. Grand average ERPs for correct positive (black line) and negative (blue line) distractor interference nogo stimuli for control participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 50. Grand average ERPs for correct positive (black line) and negative (blue line) distractor interference nogo stimuli for remitted participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 51. Grand average ERPs for correct positive (black line) and negative (blue line) distractor interference nogo stimuli for MDD participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
**Distractor interference Nogo-N2 (200-400ms).** To test hypothesis 2(a), peak average Nogo-N2 amplitudes were subjected to a 3 (laterality) × 2 (valence) × 3 (group) factorial ANOVA. The results of this analysis, shown in Table 37, found no significant effects.

Table 37

**Nogo-N2 Factorial ANOVA Results for Distractor Interference Stimuli: Study 3**

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 66) = 0.02, p = .978, \eta^2 = .001$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality×Group</td>
<td>$F(4, 132) = 0.29, p = .882, \eta^2 = .009$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F(2, 66) = 0.72, p = .492, \eta^2 = .021$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality×Valence×Group</td>
<td>$F(3.59, 118.66) = 0.796, p = .518, \eta^2 = .024$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note. * $p \leq .05$, ns $p > .05$.

Contradicting the predictions of Hypothesis 2(b), participants’ distractor interference Nogo-N2 ERPs evoked for positive and negative old stimuli, and their difference scores did not correlate with any of the psychometric questionnaires or behavioural free-recall or recognition results.

**Distractor interference Nogo-P3 (401 - 800ms).** To investigate the predictions of hypothesis 3(a), peak average Nogo-P3 amplitudes for distractor interference stimuli at frontal and central sites were subjected to a 3 (laterality) × 2 (valence) × 3 (group) factorial ANOVA. The results of this analysis are shown in Table 38, with the Laterality×Valence×Group interaction the focus of further analysis.
Table 38

*Nogo-P3 ANOVA Results during Go/Nogo Task for Distractor Stimuli: Study 3*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F (2, 66) = 0.24, p = .786, P\eta^2 = .007$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality×Group</td>
<td>$F (3.52, 116.09) = 2.35, p = .066, P\eta^2 = .066$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence×Group</td>
<td>$F (2, 66) = 4.52, p = .014, P\eta^2 = .120$</td>
<td>*</td>
</tr>
<tr>
<td>Laterality×Valence×Group</td>
<td>$F (4, 132) = 3.08, p = .018, P\eta^2 = .085$</td>
<td>*</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05$, **$p < .01$.

To identify the source of the significant three-way interaction, 3 (group) × 2 (valence) ANOVAs were conducted on distractor interference Nogo-P3 data for each lateral position (averaged over caudality). The results of these analyses, shown in Table 39, found significant Group×Valence interactions at left and midline electrode sites.

Table 39

*Breakdown of Distractor Interference Nogo-P3 Laterality×Valence×Group Interaction: Study 3*

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Electrode Sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>$F (2, 66) = 0.29, p = .749, P\eta^2 = .009$</td>
<td>ns</td>
</tr>
<tr>
<td>Valance</td>
<td>$F (1, 66) = 1.25, p = .268, P\eta^2 = .019$</td>
<td>ns</td>
</tr>
<tr>
<td>Group×Valence</td>
<td>$F (2, 66) = 4.96, p = .010, P\eta^2 = .131$</td>
<td>**</td>
</tr>
<tr>
<td>Midline Electrode Sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>$F (2, 66) = 0.27, p = .764, P\eta^2 = .008$</td>
<td>ns</td>
</tr>
<tr>
<td>Valance</td>
<td>$F (1, 66) = 5.21, p = .026, P\eta^2 = .073$</td>
<td>*</td>
</tr>
<tr>
<td>Group×Valence</td>
<td>$F (2, 66) = 6.21, p = .003, P\eta^2 = .158$</td>
<td>**</td>
</tr>
<tr>
<td>Right Electrode Sites</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>$F (2, 66) = 0.66, p = .519, P\eta^2 = .020$</td>
<td>ns</td>
</tr>
<tr>
<td>Valance</td>
<td>$F (1, 66) = 1.77, p = .188, P\eta^2 = .026$</td>
<td>ns</td>
</tr>
<tr>
<td>Group×Valence</td>
<td>$F (2, 66) = 1.51, p = .227, P\eta^2 = .044$</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Note.* ns $p > .05$, *$p < .05$. 
Subsequent univariate ANOVAs were conducted for the effect of valence for each participant group separately, for interactions observed over the left and midline electrode sites. At left electrode sites, a significant effect of valence was found for the remitted group, $F(1, 11) = 8.47, p = .014, \eta^2 = .435$, with larger Nogo-P3 evoked in response to the successful inhibition of negative compared to positive distractor interference words ($mean$ difference = 1.60µV). No valence differences were found for the control or MDD groups. A similar pattern of results were found over the midline electrode sites, with remitted participants exhibiting larger Nogo-P3 for negative compared to positive stimuli, $F(1, 11) = 8.11, p = .016, \eta^2 = .424$ ($mean$ difference = 2.47µV). Again, no valence differences were found for the control or MDD groups. A visual depiction these effects are shown in Figure 52.

![Figure 52](image)

_Figure 52._ Participants’ grand average distractor interference Nogo-P3 ERPs over left, midline, and right hemispheres (averaged over caudality) for positive and negative distractor interference stimuli during the modified Sternberg working memory inhibition task. _Note._ ** $p < .01$, error bars refer to standard error of the mean.
Contradicting the predictions of Hypothesis 3(b), participants’ distractor interference Nogo-P3 ERPs evoked for positive and negative old stimuli, and their difference scores did not correlate with any of the psychometric questionnaires or behavioural free-recall or recognition results.

**Distractor Interference LPP (801-2000 ms).** In order to investigate the predictions of hypothesis 4, peak average LPP amplitudes were subjected to a 2 (Valence) x 3 (Group) ANOVA. The interaction was null, \( F(2, 66) = 0.13, p = .875, \) \( P_{\eta^2} = .004, \) power = .070.

5.3.2.3. ERP Results for Proactive Interference Inhibitory Control.

Grand average ERP waveforms for positive and negative proactive interference Nogo stimuli for the control, remitted, and MDD participants are shown in Figures 53, 54, and 55 respectively. Visual inspection suggests little differences in Nogo-N2 or Nogo-P3 evokes for the positive and negative stimuli across all participant groups. Slightly larger LPP ERPs are evident for positive stimuli for the control group, with this effect maximum at mid and left frontal sites.
Figure 53. Grand average ERPs for correct positive (black line) and negative (blue line) proactive interference nogo stimuli for control participants during the modified working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 54. Grand average ERPs for correct positive (black line) and negative (blue line) proactive interference nogo stimuli for remitted participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 55. Grand average ERPs for correct positive (black line) and negative (blue line) proactive interference nogo stimuli for MDD participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Proactive Interference Nogo-N2 (200-400ms). To analyse the predictions of hypothesis 6, peak average Nogo-N2 amplitudes were subjected to a (valence) × 3 (group) factorial ANOVA. The analysis failed to find a significant interaction, $F(2, 66) = 0.29, p = .75, \eta^2 = .009$, or significant impact of group, $F(2, 66) = 0.08, p = .925, \eta^2 = .002$. No correlations between proactive interference Nogo-N2 results and the self-report measures were observed.

Proactive interference Nogo-P3 (401-800ms). To investigate the predictions of hypothesis 7, peak average Nogo-P3 amplitudes for proactive interference nogo stimuli at frontal and central sites were subjected to a 2 (valence) × 3 (group) ANOVA. The analysis failed to find a significant interaction, $F(2, 66) = 0.17, p = .844, \eta^2 = .005$, or significant impact of group, $F(2, 66) = 0.95, p = .391, \eta^2 = .028$. No correlations between proactive interference Nogo-P3 results and the self-report measures were observed.

Proactive Interference Nogo LPP (801-2000 ms). In order to investigate the predictions of Hypothesis 8, peak average LPP amplitudes were subjected to a 2 (Valence) x 3 (Group) ANOVA. The Valance × Group two-way interaction was significant, $F(2, 66) = 4.41, p = .016, \eta^2 = .118$. To analyse the significant two-way interaction further, separate ANOVAs were conducted for positive and negative stimuli. No group differences were found for positive stimuli ($F(2, 66) = 0.62, p = .541, \eta^2 = .018$). A simple main effect was found for the negative stimuli ($F(2, 66) = 4.41, p = .016, \eta^2 = .118$), with Bonferroni-corrected ($\alpha/3$ tests $= .017$) post-hoc comparisons found MDD participants exhibited significantly larger LPPs compared to remitted participants (mean difference $= 2.26\mu V, SEM = 0.762, p = .012$). No differences existed between control and remitted participants (mean difference $= 1.65\mu V, SEM = 0.774, p = .110$) or between control and MDD participants (mean
difference = 0.61µV, \( SEM = 0.592, p = .916 \). The planned \( t \)-test outlined in Hypothesis 8(b), failed to find significant differences in LPP amplitude between positive and negative stimuli in the depressed sample. A visual illustration of the LPP results for the participant groups is shown in Figure 56.

*Figure 56. Participants’ grand average LPP for positive and negative proactive interference stimuli during the modified Sternberg working memory task. *\( p < .05 \), Error bars refer to standard error of the mean.*

5.3.2.5. Attention processing: Go responses

Grand average ERP waveforms for Go stimuli for the Control, remitted, and MDD participants are shown in Figures 57, 58, and 59, respectively. Visual inspection of these waveforms suggests little variability both within and between groups as a function of stimulus valence.
Figure 57. Grand average ERPs for correct positive (black line) and negative (blue line) go stimuli for control participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 58. Grand average ERPs for correct positive (black line) and negative (blue line) go stimuli for remitted participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
Figure 59. Grand average ERPs for correct positive (black line) and negative (blue line) go stimuli for MDD participants during the modified Sternberg working memory inhibition task. Stimulus onset 200ms (dotted line).
**Go-P3 (301-800 ms).** Peak average Go-P3 amplitudes were subjected to a 2 (valence) × 3 (group) ANOVA. No significant effects involving the key variable, Group, were observed.

**Go-LPP (801-2000 ms).** In order to investigate sustained processing of positive and negative Go stimuli, peak average LPP amplitudes were subjected to a 2 (Valence) x 3 (Group) ANOVA. No significant effects involving the key variable, Group, were observed.

5.3.3. Impact of SSRI Medication, Comorbid Anxiety, and the Univariate Outlier on the obtained Results

The data was analysed with the exclusion of the five participants in the MDD group who were being treated with SSRI medication or who had a comorbid anxiety diagnosis. There were no changes to the direction of the obtained findings (See Appendix Q). However, due to the loss of experimental power the significant four-way interaction for the distractor interference Nogo-N2 data just failed to reach significance ($p = .059$). The results were also reanalysed with the exclusion of the univariate outlier (Appendix Q). No changes to significant/non-significant results were observed.

5.4. Discussion

The present study aimed to investigate the ERP correlates of inhibitory control for emotional material in working memory for current and remitted depression. A modified working memory Sternberg Go/Nogo task with emotional stimuli was developed. Both distractor interference and proactive interference inhibitory control processes were measured via error rates, and the Nogo-N2, Nogo-P3 and LPP ERPs. The second aim of the study sought to investigate if participant profiles varied in regards to the type of cognitive inhibitory assessed: resistance to distractor versus
resistance to proactive interference inhibitory control. The third was to test the correlated predictions of Joormann et al.’s (2007) impaired cognitive control model of depression, such as the associations between participants’ inhibitory control deficits with their self-reported depression severity, rumination, and emotional regulation. The study partially supported its aims. In the ERP data, MDD was found to be associated with over-mobilisation of midline anterior regions during inhibition of distractor interference stimuli, while remitted depression was associated with midline and left anterior region during conflict identification (Nogo-N2) of positive distractor stimuli. Remitted depressed participants were also found to be associated with enhanced Nogo-P3 during successful inhibition of negative distractor interference items. It is possible that this reflects a compensatory response during recovery from a major depressive episode. In regards to resistance to proactive interference, the behavioural error rates suggest that MDD was associated with difficulties removing negative information from working memory (Joormann & Gotlib, 2008). Consistent with previous research and theory, this inhibitory deficit correlated with sustained depression levels at a one-month follow-up. The present data showed no ERP effects of conflict monitoring (Nogo-N2), conflict regulation (Nogo-P3) or cognitive reappraisal (LPP) for proactive interference inhibition deficits in the clinical samples. It is possible that the high task demand and participant fatigue served to suppress the manifestation of these hypothesised cognitive control deficits on the electrophysiological level.

The following sections of this discussion will summarise and interpret the results for each of the study’s hypotheses. The behavioural data presented first following by the interpretation of the electrophysiological data. Results for the distractor interference stimuli are presented, followed by the results for the proactive
interference stimuli. Prior to this however, supportive data that validates the internal validity of the modified Go/Nogo task is discussed.

5.4.1. Validation of the Modified Working Memory Sternberg Go/Nogo Task

As far as the ERP results are concerned, the typical components evoked during the modified emotional Sternberg Go/Nogo task—the Nogo-N2 and Nogo-P3—were comparable with those identified in other ERP Go/Nogo studies (Chiu et al., 2008). The N2 component, which was validated by a PCA analysis, was elicited almost exclusively in the nogo condition with a frontal maximum. This replicates previous work (Chiu et al., 2008; Falkenstein et al., 1999; Folstein & van Petten, 2008; Kirmizialsan et al., 2006; Kopp et al., 1996; Nieuwenhuis et al., 2004). The P3 component was consistently observed in both the go and nogo trials. In the go trials, it showed a more parietal maximum, whereas in the nogo trials, it showed a more anterior maximum. This is consistent to previous findings using the standard Go/Nogo paradigm (Pfefferbaum et al., 1985). There was no hemispheric, laterality, or valence interactions between the distractor interference and proactive interference Nogo conditions across participants. These results are consistent with previous work (Chiu et al., 2008). Together, these expected ERP results support the internal validity of the current modified Sternberg working memory Go/Nogo task.

5.4.2. Resistance to Distractor Interference Results

A summary of resultant data for hypotheses 1 to 3 are shown in Table 40.
Table 40

*Behavioural and ERP Results of Resistance to Distractor Interference Inhibitory Control for the MDD, Remitted, and Control Groups: Study 3*

<table>
<thead>
<tr>
<th>Cognitive Process</th>
<th>Between Subjects Differences</th>
<th>Within Subjects Effects</th>
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</thead>
<tbody>
<tr>
<td><strong>Hypothesis 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhibitory control deficits for distractor interference material</td>
<td><strong>Hypothesis 1 (a)</strong> Null: No group differences were found for errors for negative distractor interference stimuli.</td>
<td><strong>Hypothesis 1 (c)</strong> Greater errors for negative compared to positive distractor interference stimuli (negativity bias) positively correlated with right Pb3 bias scores during self-referential encoding (Study 1).</td>
</tr>
<tr>
<td><strong>Hypothesis 1 (b)</strong> Null: MDD participants made similar errors for negative and positive distractor interference stimuli.</td>
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<tr>
<td><strong>Hypothesis 2</strong></td>
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<td></td>
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<tr>
<td>Conflict monitoring inhibitory control (Nogo-N2)</td>
<td><strong>Hypothesis 2</strong> Null results</td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 3</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conflict regulation inhibitory control (Nogo-P3)</td>
<td><strong>Hypothesis 3</strong> Remitted participants exhibited larger Nogo-P3s to negative compared to positive stimuli at left and midline electrode sites.</td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 4</strong></td>
<td></td>
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</tr>
<tr>
<td>Emotional reappraisal (Nogo-LPP)</td>
<td><strong>Hypothesis 4</strong> Null results</td>
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</table>

**Behavioural evidence.** The study failed to support hypothesis 1 (a) and 1 (b). Specifically, no differences were observed for distractor interference error rates between participants or valence conditions. The current experiment required that participants quickly update the content of their working memory every 30 seconds. It was also conducted following participants’ completion of two prior cognitive tasks on encoding and memory (see Study 1 and 2). As outlined in Section 3.2.3.3.2,
participants reported increased levels of fatigue with each of the experimental tasks. It is therefore possible that the current task required high cognitive load and was tested under conditions of heightened fatigue. This could have suppressed the manifestation of these hypothesised cognitive control deficits. This is consistent with previous work that finds depressive deficits are more readily observed when cognitive load is low (Joormann et al., 2010; Hertel, 1997).

These results contradict those of Joormann (2004) and Goeleven et al. (2006), who both found their depressed participants to show greater deficits inhibiting negative distractor stimuli during the NAP task compared to their control participants. The failure of to replicate these behavioural findings may be attributed to task differences between the studies. It is possible that the two tasks might assess different inhibition processes. Specifically, the NAP task may allow for greater measurement of conflict regulation processes; whereas the Go/Nogo task may allow for greater measurement of conflict monitoring processes. This appears consistent with Williams et al.’s (1997) integrated cognitive model of depression (see Section 2.1). Williams et al. argued that depression is related to an overactive resource allocation mechanism, resulting in difficulties disengaging from negative information once it has been made the focus of explicit attention (Bradley et al., 1997). However, they suggest that the disorder is not related to deficits in the affective decision mechanism, which controls initial interpretation of incoming information. These two mechanisms may underlie conflict regulation and conflict monitoring inhibitory processes, respectively. The current behavioural results are consistent with those of Joormann et al. (2010) who also failed to find group differences between control and MDD participants’ ability to ignore emotional material in an Ignore/Suppress task. It could be argued that
behavioural performance of this task also represented conflict monitoring inhibitory processes, more so than conflict regulation.

Thus, the behavioural result suggests that depressed participants can prevent entry of irrelevant emotional material into working memory as effectively as non-depressed individuals can during go/nogo processing. Prior research and theories suggest that they may find it difficult to ignore negative irrelevant material once they start processing its semantic content or affective valence. Further studies should examine this idea more explicitly by systematically varying task instructions to tap into both conflict monitoring and conflict regulation processing in one behavioural task. For instance, this could be achieved by conducting a Go/Nogo task of emotional stimuli in which participants complete a number of conditions in which these processes are manipulated. Here, one condition may require the inhibition of negative words but not positive words, with a subsequent block conducted for the inhibition of positive but not negative words. This condition may provide a measure of conflict regulation processes for negative and positive material, as participants are required to process the affective valence of the stimuli to complete the task. Another condition may require participants to inhibit responding to word stimuli which begin with a certain letter, say the letter W, with both negative (e.g., worthless, worried) and positive (e.g., wonderful, wise) Nogo stimuli presented. This condition may provide a measure of conflict monitoring processes for negative and positive material as they are only required to conduct initial evaluation of the visual aspects of incoming information. The error rates for the two conditions, visual versus affective processing, may therefore be better compared.

Alternatively, the null behavioural result might have been due to insufficient power in the statistical analyses to reveal group effects. Meta-analysis results show
that cognitive deficits associated with MDD are commonly observed in only 50% of depressed individuals (Veiel, 1997). It therefore requires large samples for these deficits to become statistically significant at the group level. Samples in electrophysiological and neuroimaging studies are usually small because of various constraints, such as time and funding limitations and participant exclusion criteria. This argument is supported by the observation of significant behavioural effects when the sample (i.e., power) was increased for the within-subjects analysis. It was found that greater inhibition errors for negative than positive distractor interference stimuli was positively associated with participants’ greater anticipation of negative stimuli during self-referential processing (P3b negative bias scores, Study 1). This data supports the notion that negative information processing biases may result from defective inhibitory processes, resulting in greater access and activation of negative material in working memory.

Electrophysiological evidence. For the electrophysiological data, hypothesis 2 predicted that MDD and remitted participants would show more negative Nogo-N2 for negative than positive distractor interference stimuli and in comparison to the control participants (Kaiser et al., 2003; Kerestes et al., 2012; Vanderhasselt et al., 2012; Yao et al., 2010). In the remitted group this result was expected to be localised over the frontal left hemisphere and be apparent despite comparable behavioural performance to the control participants (Kerestes et al., 2012). Contrary to the predictions of hypothesis 2, MDD and remitted participants were not found to exhibit enhanced Nogo-N2 to negative distractor stimuli compared to positive stimuli or compared to the control group. This result diverged from the evidence of inhibition deficits specific towards this information (Yao et al., 2010), and implicated in recent cognitive models of depression (Beck, 2008; Joormann et al., 2007). Hypothesis 3
predicted that remitted depressed participants would show greater frontal Nogo-P3 for negative relative to positive stimuli (Kerestes et al., 2012). Again, no valence effects were predicted for the Nogo-P3 for the control or MDD participants (Kaiser et al., 2003; Katz et al., 2010). Consistent with hypothesis 3, remitted depressed participants were found to exhibit enhanced Nogo-P3s at left and midline frontal sites negative as compared to positive distractor stimuli. These results are consistent with the fMRI findings of Kerestes et al. (2012) who found greater neural activity in the left DLPFC during performance of a working memory-load task with negative distracter items. Similar to the current results, Kerestes et al. also did not observe accompanying significant group differences in performance in their remitted depressed participants. Thus, the remitted depressed participants had to gather greater neuro-inhibitory resources for the purposes of stopping a prepotent response to negative stimuli with the same level of efficiency as the positive stimuli. These results are consistent with previous studies that have shown deficits in emotional processing and executive control persist into remission when patients are euthymic and medication free (Bhagwager & Cowen, 2008; Clark et al., 2005; Neumeister et al., 2006; Joormann & Gotlib, 2007; Kerestes et al., 2012; Preiss et al., 2009).

The finding that this effect was lateralized over the left and midline sites is consistent with previous neuroimaging evidence that the left DLPFC and rostral ACC are implicated in top-down attentional control (Banich, 2009). This may include processes such as directing attention away from task-irrelevant emotional distracters during working memory (Dolcos et al. 2006; Kerestes et al., 2012), and regulation of reactivity of the Amygdala (Disner, Bevvers, Haigh, & Beck, 2011). Thus, it appears that this left-midline frontal hyperactivation in the remitted group reflects a compensatory response during recovery from a major depressive episode. That is,
these individuals may engage greater neural activation during conflict regulation stages of successful inhibitory processes in order to suppress further processing of irrelevant negative content (Matthews & Antes, 1992; Walker et al., 2003). This interpretation is consistent with previous research by Wenzlaff et al. (2001) that suggests that cognitive biases are often difficult to observe in remitted depressed states because these participants actively try to suppress negative thoughts and information. Future research would likely benefit from longitudinal studies that examine the relationship between suppression and sustained depression remission through the process of enhanced cognitive inhibition over negative distractor stimuli.

As expected, control participants showed no differentiation of either the Nogo-N2 or Nogo-P3 to stimuli valence characteristics. This supports previous research that affective and arousing information show similar demands on inhibitory control in healthy samples, resulting in apt and similar task performance (Chiu et al., 2008). This may allow for greater working memory capacity and flexibility in the control participants (Hasher & Zacks, 2004). This may help to facilitate an individual’s ability to effectively problem-solve or evoke pleasant memories to help regulate mood in the face of external stress. This may work to inoculate the individual against emotional psychopathology.

The exploratory predictions of hypothesis 4 were not supported. Specifically, no Nogo-LPP differences were observed between control, remitted, and MDD participants during processing of distracting stimuli. The impact of valence was also not significant. The null result is consistent with previous investigations which show that high load working memory tasks to reduce the LPP elicited by distractor interference items (MacNamara et al., 2011; Wangelin et al., 2011) most likely due to enhanced activation of the DLPFC (Miller & Cohen, 2001). Thus, it is possible that
the high demand nature of the current Sternberg working memory task recruited greater DLPFC activation, which served to effectively inhibit sustained processing of distracting negative stimuli. It is important to note however, that null results are difficult to interpret. They might alternatively be the result of insufficient power, especially given the small magnitude of this effect (see Figure 49-51). Thus, further replication and study of the LPP during Nogo processing under conditions of high and low working memory load is warranted. It will also be important to investigate the mediating neural mechanisms of the DLPFC here.

5.4.3. Resistance to Proactive Interference Inhibitory Control

A summary of behavioural data for resistance to proactive interference inhibitory control processing (hypothesis 5-8) is shown in Table 41.

Behavioural evidence. As predicted in hypothesis 5, the results of the study indicated that participants diagnosed with MDD exhibited reduced abilities to remove proactive interference negative information from working memory. This is consistent with previous work (Berman et al., 2011; Joormann & Gotlib, 2008; Joormann et al., 2010). Specifically, compared to never-depressed and remitted depressed participants, MDD participants demonstrated greater error rates to negative proactive interference intrusion probes (i.e., a probe from the irrelevant list). This most likely reflected continued activation of this negative information in working memory in the MDD participants after it was declared to be no longer relevant. An important finding was that this pattern was not found for the positive proactive interference information, supporting the hypothesis that depression is associated with a specific deficit for mood-congruent stimuli (Joormann et al., 2007; Joormann & Gotlib, 2008).
Table 41

**Behavioural and ERP Results of Resistance to Proactive Interference Inhibitory Control for the MDD, Remitted, and Control Groups: Study 3**

<table>
<thead>
<tr>
<th>Cognitive Process</th>
<th>Between Group Differences</th>
<th>Within participant Effects</th>
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<tbody>
<tr>
<td><strong>Hypothesis 5</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhibitory control deficits for distractor interference material: error rates</td>
<td><strong>Hypothesis 5(a)</strong> MDD participants made significantly more errors inhibiting negative proactive interference stimuli compared to both the control and remitted participants.</td>
<td><strong>Hypothesis 5(c)</strong> Greater errors for negative compared to positive proactive interference stimuli (negativity bias) positively correlated with participants’ self-reported depression, rumination, and anxiety, and negatively correlated with emotional regulation scores. It was also found to positively correlate with behavioural measures of the negative recognition memory bias.</td>
</tr>
<tr>
<td></td>
<td><strong>Hypothesis 5(b)</strong> MDD participants made significantly more errors for inhibition of negative relative to positive proactive interference stimuli.</td>
<td></td>
</tr>
<tr>
<td><strong>Hypothesis 6</strong></td>
<td>Null results</td>
<td></td>
</tr>
<tr>
<td>Conflict monitoring inhibitory control (Nogo-N2)</td>
<td></td>
<td></td>
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<tr>
<td><strong>Hypothesis 7</strong></td>
<td>Null results</td>
<td></td>
</tr>
<tr>
<td>Conflict regulation inhibitory control (Nogo-P3)</td>
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<tr>
<td><strong>Hypothesis 8</strong></td>
<td>MDD participants exhibited significantly larger LPPs for negative stimuli compared to Remitted participants.</td>
<td>Sustained processing bias of negative proactive interference stimuli (greater LPPs for negative than positive stimuli) was found to positively correlate with level of rumination and follow-up depression levels.</td>
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<tr>
<td>Sustained processing (LPP)</td>
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</table>

The finding that the remitted participants performed similarly to the control participants suggests that, on the performance output level, this deficiency in proactive interference deficit appears to diminish following depression recovery. This result
contradicts previous findings that remitted depression also shows reduced ability to
proactive interference negative information (Bhagwagar & Cowen, 2008; Joormann &
Gotlib, 2007; Vanderhasselt et al., 2012), and that it is predictive of one-year follow-
up depression severity as mediated by greater rumination (Demeyer et al., 2012).
However, task and sample differences may help explain these studie’s differences.
First, both Vanderhasselt et al. and Demeyer et al. utilised neutral stimuli as the control
condition. Thus, it is difficult to determine if stimuli were effectively matched on
arousal dimensions as was manipulated in the current study (see Section 2.2.2.). The
present study also employed young female participants who were actively pursuing
their education; that is, one may assume that they were relatively high functioning
individuals. In contrast, both Vanderhasselt et al. and Demeyer et al. had greater age
variances and more heterogeneous samples. Vanderhasselt et al’s. behavioural
differences emerged in their response time data. However, in the current task reaction
time, data could not be measured for the proactive interference stimuli as they required
an inhibition of the behavioural response from participants (nogo responses). Thus, it
is possible that Vanderhasselt et al. measured prepotent response inhibition in their
study (Friedman & Miyake, 2004), which the current task did not measure. Future
research would benefit from directly comparing these two forms of inhibition in the
context of remitted depression.

The present study utilised a structured clinical assessment to determine clinical
group status. This process effectively ruled out any comorbid axis I disorder in the
remitted sample. Demeyer et al. also utilised a structured clinical interview to exclude
the presence of depression of other axis I conditions in their remitted depressed
participants, with the exception of anxiety disorders (38% of sample). The presence of
anxiety disorder in their remitted depressed sample is problematic because anxiety is
associated with hypervigilance towards highly arousing information (Bar-Hiam et al., 2007). Thus, the results of previous studies to assess proactive interference in remitted depression may be confounded by the possibility that observed deficits in inhibition may be due to the arousal level of the negative words rather than their negative valence alone, and these deficits may be influenced by participants’ anxiety levels, rather than, or in addition to, their remitted depression. Further, 43% of Demeyer’s remitted participants were taking anti-depressant medication—as opposed to the 0% of the remitted sample in the current study—thereby suggesting that these participants were not in complete clinical remission. This makes it difficult to generalise their information to either current or remitted depressed samples. It is important to also note that Demeyer et al. also failed to find significant associations between cognitive control, rumination, and depression symptoms at baseline measurement. Similar to the remitted sample used in the current study, at baseline testing most of Demeyer et al.’s remitted sample scored in the non-clinical levels on the BDI-II. However, one-year later 34.8% of Demeyer’s remitted sample scored in the clinical level (>19) on the BDI-II. Participants’ cognitive control impairments were predictive of their future depression severity, which was mediated by rumination. It is possible that at baseline, Demeyer’s remitted sample exhibited little variability in the depression measure, which might have reduced chances of finding significant associations at this time. It is possible that this played a part in the current data’s inability to find effects here, with the range of participant scores on the BDI-II at baseline also small (see section 3.3). Alternatively, it is possible that the relationship between mood and inhibition of negative material in working memory is nonlinear; that is, deficits are found only at very high levels of negative affect.
Consistent with the predictions of hypothesis 5(c) Pearson’s correlation coefficient analyses found that greater errors for negative compared to positive proactive interference stimuli (difference score) positively correlated with current and one-month depression levels, rumination, and self-reported anxiety levels. The proactive interference negativity bias scores were also found to positively correlate with greater recognition memory for negative compared to positive stimuli. These data are theoretically consistent with Joormann et al.’s (2007) cognitive model.

**Electrophysiological evidence.** To this PhD candidate’s knowledge, no previous ERP study has examined MDD in the context of proactive interference inhibitory control for emotional material. Further, no previous investigations have directly compared MDD participants with remitted depressed participants for proactive interference inhibitory control or have examined these processes using the Go/Nogo task. Thus, the associated electrophysiological indices of proactive interference inhibitory processing in this sample are relatively unknown. Therefore, the predictions of the ERP data for proactive interference inhibitory control were based on theory and thus, were relatively exploratory. Specifically, hypothesis 6 predicted that MDD participants would show poorer inhibitory control conflict monitoring (as evidenced by smaller Nogo-N2) for negative than positive proactive interference stimuli, and overall smaller Nogo-N2s than the control or remitted participants (Joormann 2010; Vanderhasselt et al., 2012). Hypothesis 7 expected control participants to show enhanced inhibitory control conflict regulation for negative (as evidenced by larger Nogo-P3s) than positive proactive interference stimuli and in comparison to the MDD and Remitted groups (Vanderhasselt et al., 2012).

Contrary to the expectations of hypotheses 6 and 7, the present data showed no ERP effects of the Nogo-N2 or Nogo-P3 for proactive interference stimuli in the MDD
or remitted samples. This finding is surprising, given the observation of the above mentioned behavioural performance differences. Although some investigators have demonstrated that depression is associated with an impaired ability to remove negative material from consciousness once it enters (Banich et al., 2009; Joormann & Gotlib, 2008; Joormann et al., 2010; Whitmer & Banich, 2007), most of this data has generally been obtained from studies at the behavioural level. It is possible that the current task required high cognitive load and was tested under conditions of heightened fatigue, which suppressed the manifestation of these hypothesised cognitive control deficits on the electrophysiological level. This is consistent with previous work that finds depressive deficits are more readily observed when cognitive load is low (Joormann et al., 2010; Hertel, 1997).

As argued previously, sample characteristics may account for the inability of the data producing the expected ERP effect or proactive interference inhibitory control deficits in the MDD sample. Specifically, it could be that the use of a university student sample ensured that the participants used in the current study were relatively high functioning and intelligent. Thus, the results from this sample group may not easily generalise to a less functional sample, such as inpatient groups. Alternatively, the EEG has limited spatial resolution. Thus, it is possible that individual differences in this electro-cognitive profile may have resulted in error variances that concealed this effect in the between-subjects analysis (Type II error). In fact, studies assessing the role of spatial density (i.e., number of electrodes) on source reconstructions show improved spatial resolution with high-density EEG recordings. In a simulation study, Lantz, Grave, Spinelli, Seeck, & Michel (2003) reported that the source localization accuracy increased linearly from 25 to 100 electrodes. Michel et al. (2004) found that source imaging with 128-channel EEG epileptic spike data led to correct localization
(to the order of the affected lobe) in 93.7% of the focal epileptogenic area as independently assessed through pre-surgical assessments. It is therefore possible that the 32-channel EEG used in the present study did not provide enough spatial resolution data to allow for the identification of aberrant left inferior frontal gyrus spatial variability in the current depressed sample. Therefore, it is still unclear at which temporal stage—which can be effectively measured by the ERP—of emotional information processing these neural processes are affected by updating the contents of working memory inhibitory dysfunction in depression. This knowledge would be beneficial for developing effective means of intervention. Further ERP studies with larger samples and greater density EEG may amend this shortcoming.

The finding that proactive interference inhibitory deficits for negative material were observed for the behavioural data and not the electrophysiological may be due to the possibility that the ERP only analysed correct inhibition responses, whereas, the behavioural data measured errors which can be fewer of number compared to those required in ERP methods (see section 2.2.2.). This is an inherent limitation in the ERP studies as most Go/Nogo studies are based on analysis of successful inhibition performance only. Given predictions that MDD is associated with inhibition impairment, especially for mood-incongruent stimuli, it may be possible that excluding incorrect responses from statistical analysis fails to assess the potential points of the information processing stream where these biases originate. The present study only analysed correct responses so to replicate previous work. In future, accuracy rates for all trial types were high (77.5%–84.77%), preventing examination of ERPs time-locked to errors. Future work may benefit from assessing a larger pool of stimuli and directly examining the effects of performance on the Nogo components.
Hypothesis 8 predicted a group-by-valence effect for the proactive interference Nogo-LPP data. Specifically, reflective of cognitive reappraisal, remitted depressed individuals were expected to show smaller Nogo-LPPs for negative material relative to the MDD participants. While reflective of their poor emotional reappraisal strategies, those with MDD were expected to show larger Nogo-LPPs following negative relative to positive distractor interference stimuli. The study supported these predictions. Given that the LPP is reduced under conditions of voluntary suppression of negative emotion (Moser et al., 2006), the finding that the remitted, but not control participants, exhibited significantly reduced LPPs compared to the MDD group, may suggest evidence for compensatory suppression of negative content in recovery from depression order to maintain a euthymic mood (Matthews & Antes, 1992; Walker, Skowronski, & Thompson, 2003). It is important to note that the LPP showed a small magnitude (approximately 2µV), variable latency, and lacked clearly discernable peaks in the grand-average data for each of the participant groups (see Figures 53 - 55). Thus, although ANOVA assumptions of normality were met for the above analysis, special caution needs to be taken in the interpretation of these results. The current findings would benefit from further replication in a larger sample. It would be interesting to examine the impact that remitted depressed individuals’ past treatment modalities (e.g., medication versus cognitive regimes), age of depressive-onset, and depression course (e.g., number of past episodes, average duration of illness) have on their emotional reappraisal abilities following exposure to distressing events. Further, greater understanding of the possible neural-generators underlying reappraisal in remitted samples could also be gained by analyzing LPP data alongside neural imagining techniques, such as fMRI. Last, ERP extraction techniques, such as area
under curve might also prove beneficial given the long latency, and thus variability, of this ERP (Schupp et al., 2000).

5.4.4. Summary Working Memory Inhibitory Deficits in Depression

In summary, remitted participants exhibited greater Nogo-N2s during inhibition of negative distractor interference items. They also showed enhanced Nogo-P3 and reduced LPPs during removal of negative proactive interference items from working memory. This may suggest evidence of recruitment of suppression-based processes in remitted depression in an effort to maintain euthymic mood when confronted with stressful stimuli. For the MDD group, the study failed to support its ERP hypotheses. It is possible that the high task demand and participant fatigue served to suppress the manifestation of these hypothesised cognitive control deficits on the electrophysiological level. For the behavioural data, depressed individuals showed difficulties removing once relevant but now irrelevant negative items from working memory (proactive interference). This inhibitory deficit positively correlated with participants’ self-reported depression, and rumination, and negatively correlated with emotional regulation scores. It was also found to positively correlate with negative recognition memory bias scores. These results generally support the predictions of Joormann et al.’s (2007) impaired cognitive control model of depression. The final study of this dissertation—Study 4—examines the predictive power of this model. It is presented next.
Chapter 6

Study 4 – A Discriminative Validity Analysis of Joormann et al.’s (2007) Impaired Cognitive Control Model of Depression

6.1. Background and Hypotheses

The aim of the present study was to investigate if the cognitive processes implicated in Joormann et al.’s (2007) impaired cognitive control framework could effectively predict depression status (MDD, remitted depression, and never-depressed controls). Specifically, predictive discriminant function analyses (DA) were performed using the linear combination of participants’ behavioural markers of inhibitory control deficits, including both resistance to distractor and resistance to proactive interference; markers of mood-congruent free-recall and recognition memory; and participants’ self-reported use of rumination, and emotional reappraisal and suppression techniques. The study also allowed determination of which variables contributed to the separation of group membership (Norusis, 2008).

The discriminant analysis did not include any of the ERPs analysed in Study 1-3, despite finding preliminary evidence that they mapped onto different cognitive aspects of Joormann’s et al.’s model. Many of these ERPs were found to differ between current, remitted and never-depressed samples, and thus may be useful in clinically separating their profiles. However, their inclusion in the current discriminant analysis would render the discriminant function unstable. Specifically, the robustness of classification in discriminant analysis is sensitive to multicollinerarity between predictor variables. Thus, inclusion of ERPs that have similar morphology (e.g., P3b and Nogo-P3) but that represent different aspects of Joormann’s et al.’s cognitive model (e.g., attention versus inhibitory processes) would
violate this statistical assumption. Given that these results related to clinical profiles, it is important to show caution here. Further, unlike the behavioural or psychometric measures, there continues to be marked inconsistency in the interpretation of ERP data in this literature (e.g., see Section 3.4.1). This raises the question as to the current applicability of these ERPs as clinical assays. This is particularly the case for the Nogo ERPs evoked in the never-before tested Modified Sternberg working memory task of Study 3. Rather, these ERPs need to be further validated in larger samples and then replicated prior to their inclusion in clinical classification analyses. This is an avenue for future research.

6.1.1. Study background. Joormann et al.’s (2007) cognitive control model of depression argues that impaired ability to exert top-down inhibitory control over the contents of working memory results in lack of control over the bottom-up negative self-schemas and ruminative response styles (Hertel, 1997; Joormann, 2004; Joormann, 2005; Joormann et al., 2008; Linville, 1996; Nolen-Hoeksema et al., 2008). The model implicates deficits in both resistance to distractor interference and resistance to proactive interference inhibitory control processes (Friedman & Miyake, 2004). Section 2.1. of this dissertation explains this model, and Section 2.3. provides the existing empirical evidence supportive of these predictions. Although, Joormann et al.’s model does not make specific reference to remitted depression, impaired cognitive inhibition has been found to be defective in this group (Vanderhasselt et al., 2012). Research on remitted MDD has resulted in the mapping of vulnerability factors, which are found to significantly increase the risk of subsequent major depressive episodes (see Ingram et al., 1998). This valuable clinical group does not possess the confounding influence of the presence of depressive symptoms. In the context of inhibitory control as a cognitive vulnerability mechanism in depression, this
effect is likely to be pronounced in females. Females’ tendency to engage in greater ruminative responses styles has been found to partially account for this gender difference (Nolen-Hoeksema et al., 1999). Thus, although Joormann et al.’s model does not specify effects for remitted depression, if it were to show good sensitivity and specificity of prediction specific for remitted depressed status, the model may provide an important clinical tool for identifying those vulnerable to depressive relapse. This may be particularly sensitive for the female client.

The objective of Study 1 to 3 of this dissertation—similar to most other previous studies in this area (see Section 2.3)—has been the establishment of mood-congruent cognitive biases and inhibition deficits in depression. They were also interested in examining how these processes are related to poor emotional regulation skill and sustained depressed mood. However, these studies have utilised multiple univariate tests, an approach that did not take into consideration correlations between dependent measures. This work effectively informs us about the possible noxious prognosis that each of these cognitive patterns may have on depressed individuals. However, it is unable to explain how these processes are related to each other or interacts to maintain depression. In an attempt to address this shortcoming, the present study statistically integrated the resultant behavioural data for these mood-congruent memory biases, inhibitory deficits, and emotional regulation processes obtained in the three previous ERP studies. Multivariate statistics were employed here, specially, predictive discriminant analysis (protocol outlined in Section 2.6).

6.1.2. Study hypotheses. The following predictions were made:

*Hypothesis 1* predicted a strong significant relationship would be found between predictors outlined in Joormann et al’s (2007) cognitive model and clinical group status.
Hypothesis 2 predicted higher structure loadings on the discriminate analysis for ruminative response styles, negative memory biases and emotional regulation deficits, with smaller effects for working memory inhibitory control deficits for negative stimuli.

Hypothesis 3 predicted good (> .75; Fleiss, 1981) sensitivity and specificity for the control and MDD samples based on the linear combination of the predictor variables. The remitted group was expected to show poor predictive sensitivity, given the equivocal data in the literature for the presence of cognitive deficits and emotional biases in remitted depression and the heterogeneous nature of these samples.

6.2. Method

6.2.1. Participants

Participants consisted of the same sample of 73 female university students who were recruited in Study 1 to 3 (see Section 3.2.1). Participants were placed into one of three groups according to their SCID-I/NP diagnoses:

4. Current depressed group (MDD; n = 30).
5. Remitted depressed group (remitted; n = 13).
6. Healthy control/never depressed group (control; n = 30).

See Section 3.3.2. of this dissertation for data on participant characteristics.

6.2.2. Materials

Clinical and psychometric assessments. The self-report tests and structured clinical interviews outlined in Section 3.2.2. of this dissertation were utilised.

6.2.3. Procedures

Operational definitions of the predictor variables.
Free recall memory. The procedures for obtaining this data are outlined in detail in Section 4.2 of this dissertation. The dependent variable was the frequency of recall of positive relative to negative adjectives (free recall negative bias score).

Recognition memory: The old/new paradigm. The procedures for obtaining this data are outlined in detail in Section 4.2 of this dissertation. The dependent variable was the frequency of recognition of positive relative to negative adjectives (recognition memory negative bias score).

Inhibitory Control: The modified Sternberg working memory task. The procedures for obtaining this data are outlined in detail in Section 5.2 of this dissertation. Dependent variables included the frequency of errors for positive relative to negative distractor interference nogo stimuli (distractor interference negative bias score); and errors for positive relative to negative proactive interference nogo stimuli (proactive interference negative bias score).

Self-report assessment. After completion of the modified Sternberg working memory task, participants were seated in a private room and asked to complete the RRS and the ERQ self-report questionnaires.

Statistical analysis. Bivariate and partial correlations were conducted to test the predicted associations in Joormann et al.’s model. Discriminant function analysis (DA) was performed to evaluate the predictions of the study hypotheses. The behavioural markers of inhibitory control deficits included the distractor interference and proactive interference negative bias scores. Behavioural markers of mood-congruent memory bias scores included participant’s free-recall and recognition memory negative bias scores. Markers of emotional regulation processes was measured by participants’ self-reported use of rumination on the RRS, and their self-reported use of emotional reappraisal and suppression techniques on the ERQ. Wilk’s
lambda was used to assess the significance of the discriminate function as a whole. Fisher’s LSD procedure was employed to make pairwise comparisons among three groups. Univariate analysis was employed to determine if the predictor variables differed significantly between the three participant groups. Fisher’s procedure was selected to tease out any significant differences here as it has been found to contain family-wise error at or below the nominal rate (Levin, Serline, & Seaman, 1994). Jacknifed (one case at a time deleted) quadratic classification method was used to determine if participants were correctly classified into their respective groups based on the linear combination of the predictor variables.

6.3. Results

6.3.1. Inter-correlations between ERP studies. A summary of the correlations for the ERP and behavioural assays of the variables implicated in Joormann et al.’s (2007) cognitive model derived from Study 1, 2, and 3 are shown in Figure 60. Given that many of the experimental variables were associated, partial correlations were also calculated to determine the unique relationship between each of the variables. Specifically, the unique variances were obtained whilst statically removing the impacts that the other cognitive variables had on the correlated factors. For instance, the partial relationship between proactive interference biases with RRS scores was computed while controlling for the effects that these variables had with schema congruency effects/attention biases, recognition and free-recall memory effects, and the distractor interference bias effects. These partial correlations are presented in the parentheses under each of the bivariate results.
Figure 60. Correlations (partial correlations in parentheses) between working memory inhibitory control deficits for negative stimuli, defective emotional regulation, and mood-congruent cognitive biases implicated in Joormann et al.’s (2007) cognitive control model of depression.
6.3.2. Separation of clinical groups based on Joormann et al.’s model.

Discriminant analysis was conducted on the original sample of 73 cases (30 controls, 13 remitted depressed and 30 MDD participants). One univariate outlier was identified in the data set in the recognition memory negative bias scores ($z = -3.51$). Given that the sample consisted of both clinical and non-clinical participants known to vary on this variable (see Dati et al., 2012 for a review and Chapter 4), its distribution is expected to have greater spread than normal. Thus, this participant was deemed to be part of the sample and thus was retained in the main analysis (Tabachnick & Fidell, 2007). An analysis with this outlier removed is shown in Appendix Q in which no differences to significant/non-significant effects were found. No multivariate outliers were present in the data set (largest Mahalanobis Distance = 15.92, $z = 2.70$).

Evaluation of assumptions of linearity, normality, multicollinearity or singularity (all inter-correlations $r < .55$) and homogeneity of variance-covariance ($\text{Box’s } M \ p = .791$) were all acceptable for DA. This allowed for an adequate sample size of approximately 10:1 ratio of cases to predictors (Tabachnick & Fidell, 2007).

To test the predictions of hypothesis 1, a DA was conducted to determine group prediction based on clinical status: MDD, remitted depressed, and never-depressed controls. Two discriminate functions were calculated, with a combined $\Lambda = .322$, $\chi^2(14, N = 73) = 7.91, p < .001$. After removal of the first function, there was still a strong relationship between groups and predictors, $\Lambda = .831$, $\chi^2(6, N = 73) = 12.39, p = .027$, with Canonical $R = .783$ for the first discriminant function and .411 for the second discriminate function. The two functions accounted for about 88.6% and 11.4% of the between-group variance respectively. As shown in Figure 61, the first discriminant function maximally separates the MDD group from the other two
non-depressed groups. The second discriminate function discriminates the control and remitted groups, with MDD participants falling between these two groups.

Figure 61. Discriminant functional analysis for cognitive processes implicated in Joormann et al.’s (2007) model of depression. Note. CNT refers to control group, RMT refers to remitted group, and MDD refers to MDD group. Group centroid scores indicated by the green square (for function 1: Control centroid score = -1.15, remitted centroid score = -0.72, MDD centroid score = 1.46; for function 2: Control centroid score = 0.33, remitted centroid score = -0.91, MDD centroid score = 0.07).

Testing the predictions of hypothesis 2, the structure (loadings) of correlations between predictors and discriminant functions were calculated. They are outlined in Table 42. The best predictors for distinguishing between depressed and non-depressed status (the first discriminant function) was increased self-reported use of rumination, decreased self-reported utilisation of emotional regulation, and greater mood-congruent free-recall and recognition memory for negative relative to positive stimuli.
Although only small, greater proactive interference deficits for negative than positive information also loaded on this function. The best predictors for distinguishing the control and remitted groups (the second discriminant function) was greater self-reported use of suppression strategies, with a small loading evident for distractor interference negative bias scores. Free-recall negative memory biases scores also negatively loaded (-.462) on the second discriminant function.

Table 42

*Structure Matrix between Predictors and Discriminant Functions*

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Discriminant Function</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Rumination (RRS-Total)</td>
<td>.843</td>
</tr>
<tr>
<td>Free-Recall Negative Bias</td>
<td>.473</td>
</tr>
<tr>
<td>Emotional Regulation (ERQ-R)</td>
<td>-.438</td>
</tr>
<tr>
<td>Recognition Memory Negative Bias</td>
<td>.405</td>
</tr>
<tr>
<td>Proactive Interference Negative Bias</td>
<td>.232</td>
</tr>
<tr>
<td>Emotional Suppression (ERQ-S)</td>
<td>.298</td>
</tr>
<tr>
<td>Distractor Interference Negative Bias</td>
<td>.129</td>
</tr>
</tbody>
</table>

| Eigenvalue | 1.58 | 0.20 |

*Note.* Boldface indicates largest absolute correlation between each predictor and discriminant function.

Table 40 contains the classification means for the three participant groups on the discriminant functions. Univariate analysis (all \( df = 2, 70 \)) showed that self-reported use of rumination, emotional regulation and emotional suppression strategies, and negative biases in free-recall and recognition memory differed significantly between the three participant groups (all \( p < .001 \)). There was a trend for group differences for proactive interference negative bias scores (\( p = .053 \)). No significant
Bonferroni procedure was employed to make pairwise comparisons among three groups (directional hypotheses). Significant differences are denoted in Table 42 as having different letters in their superscripts.

Table 43 suggests that on the first function, MDD participants had higher rates of rumination, recognition and free-recall for negative relative to positive stimuli, and less use of emotional regulation strategies compared to both the control and remitted participants. The control participants recalled more positive words than the remitted participants. On the second function, the remitted participants showed less self-reported use of emotional suppression strategies compared to the control and MDD participants.

Table 43

*Means (SD in parentheses) for the Discriminant Functions for the Predictor Variables*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Clinical Status</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Loading on Function 1</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Rumination (RRS-Total)</td>
<td>35.40 (11.40) a</td>
</tr>
<tr>
<td>Free-Recall Negative Bias</td>
<td>-6.89 (5.54) a</td>
</tr>
<tr>
<td>Emotional Reappraisal (ERQ-R)</td>
<td>31.33 (5.17) a</td>
</tr>
<tr>
<td>Recognition Memory Negative Bias</td>
<td>-16.41 (12.67) a</td>
</tr>
<tr>
<td>Proactive Interference Negative Bias</td>
<td>-1.22 (9.64) a</td>
</tr>
<tr>
<td><strong>Loading on Function 2</strong></td>
<td></td>
</tr>
<tr>
<td>Emotional Suppression (ERQ-S)</td>
<td>13.90 (4.28) a</td>
</tr>
<tr>
<td>Distractor Interference Negative Bias</td>
<td>-0.89 (3.02) a</td>
</tr>
</tbody>
</table>

*Note.* Within each row, means having the same letter in their superscripts are not significantly different from each other at the .05 level.
Jacknifed (one case at a time deleted) quadratic classification method found 49 of the 73 participants (67.1%) were correctly classified into their respective groups based on the linear combination of the predictor variables. This is compared with the 34 (46.18%) who would have been properly classified by chance alone. As outlined in Table 44, the DA produced correct classification (sensitivity) of 66.7% of the control participants, 7.7% of the remitted participants, and 93.3% of the MDD participants. As can in seen in Table 43, most of the remitted participants were classified as part of the control group (69.2%). Specificity ratings were high across all participant groups: Control = 76.74%, 95% confidence interval: 61.36 – 88.23%, remitted = 90%, 95% confidence interval: 79.48 – 96.22%, and MDD = 81.40%, 95% confidence interval: 66.9 – 91.8%.

Table 44

*Discriminant Analysis Jacknife Classification Results*

<table>
<thead>
<tr>
<th>Actual Group</th>
<th>Control</th>
<th>Remitted</th>
<th>MDD</th>
<th>Total n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>20 (66.7%)</td>
<td>5 (16.7%)</td>
<td>5 (16.7%)</td>
<td>30</td>
</tr>
<tr>
<td>Remitted</td>
<td>9 (69.2%)</td>
<td>1 (7.7%)</td>
<td>3 (23.1%)</td>
<td>13</td>
</tr>
<tr>
<td>MDD</td>
<td>1 (3.3%)</td>
<td>1 (3.3%)</td>
<td>28 (93.3%)</td>
<td>30</td>
</tr>
</tbody>
</table>

*Note.* Percentage correct classification is presented in parentheses.

6.3.2. Impact of SSRI Medication, Comorbid Anxiety, and the Univariate Outlier on the obtained Results

The data was analysed with the exclusion of the five participants in the MDD groups who were being treated with SSRI medication or who had a comorbid anxiety
diagnosis. There were no changes to the direction of findings (See Appendix Q). The results were also reanalysed with the exclusion of the univariate outlier (Appendix Q). No changes to significant/non-significant results were observed. However, distractor interference error scores were found to load on factor 1 rather than factor 2. Similar specificity and sensitivity results were observed.

6.3.3. Separation of Depressed Status based on Joormann et al.’s model

Given that Joormann et al.’s (2007) cognitive model of depression was designed to separate depressed and non-depressed cognitive functioning, an additional DA was performed to determine whether participants current depressed and non-depressed clinical status could be distinguished based on the linear combination of the hypothesised predictors. The results of this subsequent analysis are shown in Appendix AA as it does not pertain to any of the research predictions.

6.4. Discussion

The aim of Study 4 was to investigate if the cognitive processes implicated in Joormann et al.’s (2007) cognitive control model could effectively predict depression status. The study achieved its experimental aims. The following sections of this discussion will summarise and interpret the study results. Limitations, avenues for future research and clinical applications of the findings are briefly discussed, with Section 7.3. of the General Discussion elaborating on this further.

6.4.1. Inter-Correlations between ERP Studies

The correlations observed in the dissertation (see Figure 60) are theoretically consistent with Joormann et al.’s general predictions. Specifically, depression symptoms was associated with greater activation of negative schemas in working memory, operationalised as enhanced P3b (data derived from Study 1) and N400s (data derived from Study 2) for positive than negative stimuli. It was also significantly
correlated with greater behavioural evidence of trouble inhibiting negative proactive interference stimuli from working memory (data derived from Study 3), which was also correlated within greater emotional regulation deficits (i.e., increased susceptibility to ruminate, recollection bias scores, decreased use of emotional reappraisal techniques), and with one-month BDI-II levels. Working memory inhibitory control deficits for negative distractor information (data derived from Study 3) was correlated with schema congruency bias scores (P3b) and decreased accessibility of positive material (free recall bias score from Study 2), providing support of the belief that it is associated with activation of negative content in working memory. Distractor interference negative bias scores were not found to correlate with depression symptoms, use of rumination, emotional reappraisal deficits, or long term negative memory biases. This is inconsistent with previous research that finds positive associations between these constructs (e.g., Zetche & Joormann, 2011).

Emotional affect and regulation were measured with questionnaires, whereas the cognitive processes were measured with cognitive tasks. These questionnaire-based measures sometimes do not correlate very highly with laboratory results for emotional information processing biases (e.g., Koster, De Raedt, Leyman, De Lissnyder, 2010). Thus, it is important to note that although these questionnaires have shown external validity to predict depression and emotional regulation behaviours, they were not designed to measure the cognitive processes or brain activity pinpointed in theoretical models of depression. That is, it is possible that method variance may have decreased the ability to detect these hypothesized relationships. Podsakoff, MacKenzie, Podsakoff, and Lee (2003) describe method variance to “variance that is attributable to the measurement method rather than to the construct of interest” (pg. 879). Meta-analytic studies find that the relationship between constructs is strongly influenced by
whether their measures are obtained from the same or different sources (Lance et al., 2010). Thus, the differences in the measurement methods between distractor interference negative bias scores with self-reported depression symptoms, for instance, may have resulted in the statistical deflation of their expected bivariate linear relationships (Type II error). Future research might benefit from mitigating the potential effects of method bias when data from psychometric tests and cognitive tasks are to be examined. Podsakoff et al. (2003) outlines a number of statistical remedies that will minimise the detrimental effects of method biases. One of these is the correlation-based marked variable technique (Lindell & Whitney, 2001). This technique requires researchers to identify a ‘marker variable’ that is not expected to be related to the variables of interest. The smallest correlation between the marker variable with the variables of interest serves as an estimate of the effect of method bias. This is then statically paritaled out of the experimental/hypothesized zero-order correlations.

Given that many of the experimental variables were correlated, partial correlations were also conducted to determine the unique relationships between them. All the partial correlations were observed to be smaller than the simple correlations, suggesting shared variances between the experimental variables. This is expected given the many moderating predictions between variables. For instance, theory and previous research suggest that greater errors for negative distractor interference stimuli would be able to predict greater sustained depression levels with this link to be moderated by rumination or emotional regulation deficits (Demeyer et al., 2012). Despite this, many of the partial correlations remained significant, suggesting strong unique relationships between the variables. This is especially the case for the relationships between reduced proactive interference inhibitory control for negative
stimuli with depression and rumination scores, and with negative biases in recognition memory. However, some significant correlations disappeared when the other experimental variables were controlled. This was the case for the relationship between greater P3b bias score with BDI-II, and N400 bias scores with BDI-II scores. Both these correlations failed to remain significant while controlling the other variables. This is likely due to the P3b and N400 biases both being measures of schema activation (e.g., decreased activation towards expected, negative stimuli). Thus, shared variance between them is anticipated, validating this interpretation of these ERPs (see Study 1 and 2 discussions).

It is acknowledged that this data is correlational in nature, and although the results are consistent with theoretical expectations, no causal interpretations can be made. That is, impaired working memory inhibitory control could result in these negative attention and memory biases and emotional regulation deficits that lead to depression. However, the alternative could equally be true: depression results in mood-congruent attention and memory and poor regulation selection. Further, it might be possible that these associations are the result of an un-measured variable, such as genetics (e.g., Hasler, Drevets, Manji & Charney, 2004; Whitmer & Gotlib, 2012) or personality variables (Lanciano, Bianco, Curci, & Cozzoli Poli, 2009; Lieberman, 2000). Future research should aim to examine these hypothesised associations using experiential manipulation of cognitive control within prospective longitudinal assessment or cross-lagged designs. These studies would better determine if cognitive control deficits are a casual factor in the development of maladaptive emotional regulation and depression, rather than just a factor associated with these outcomes.

6.4.2. Interpretation of the Discriminant Analysis
Predictive DA was performed to determine whether the current MDD, remitted depressed, and never-depressed control groups could be distinguished from one another based on the linear combination of the cognitive processes implicated in Joormann et al.’s (2007) cognitive control model of depression. Linear combinations comprised of participants’ behavioural markers of inhibitory control deficits, including both resistance to distractor and resistance to proactive interference; markers of mood-congruent free-recall and recognition memory bias scores; and participants’ self-reported use of rumination, reappraisal and suppression emotional regulation techniques. Supportive of hypothesis 1, the analysis found two significant discriminative functions that when attempting to differentiate between the three groups. The first function accounted for a large proportion of the between groups variance. It appeared to maximally separate the MDD group from the other non-depressed groups (remitted depressed and control group). The second function appeared to primarily distinguish the control and remitted groups. Table 45 displays the order of predictive function and factor loading for the predictor variables.

Table 45


<table>
<thead>
<tr>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Separated MDD group from non-depressed</td>
<td>Separated MDD group from non-depressed</td>
</tr>
<tr>
<td>(remitted and control groups)</td>
<td>(remitted and control groups)</td>
</tr>
</tbody>
</table>

1. Greater self-reported use of rumination
for MDD participants  2. Less self-reported use of emotional reappraisal in control participants.

3. Greater free recall of negative than positive words recalled in a short-term memory task in MDD participants

4. Less self-reported use of emotional reappraisal for MDD participants

5. Greater recognition memory for negative than positive words in the MDD participants

6. Greater errors for negative than positive proactive interference stimuli in the MDD participants

7. Greater errors for negative than positive distractor interference stimuli in the Remitted participants

Note. Variables are numbered in order of predictive strength.

The cluster of predictors is consistent with those implicated in Joormann et al.’s (2007) cognitive control model of depression. Their relative predictive strengths are consistent with the predictions of hypothesis 2. No significant group differences were found for the negative bias distractor interference error rates. These results contradict those of Joormann (2004) and Goeleven et al. (2006), which both found their depressed participants to show greater deficits inhibiting negative distractor stimuli during the NAP task compared to their control participants.

As outlined in Section 5.4. of this dissertation, the failure to replicate these behavioural findings may be attributed to task differences between the studies, which may assess different inhibition processes. Specifically, it is argued that behavioural performance of the Go/Nogo task represented conflict monitoring inhibitory
processes—which seem to be preserved in MDD—more so than conflict regulation, which appears anomalous in the disorder.

The second function accounted for a smaller proportion of between groups variance. It appeared to maximally separate the control from the remitted depressed participants. Remitted participants exhibited greater use of suppression emotional regulation strategies and, to a smaller extent, greater distractor intrusions from positive stimuli. Both of these processes most likely represent their compensatory attempts to keep their euthymic mood in recovery from depression (see section 5.4. from Study 3).

Quadratic classification procedures found around 70% of participants were able to be correctly classified into their respective groups based on the linear combinations of the predictor variables outlined in Joormann et al.’s model. Consistent with the predictions of hypothesis 1, this was significantly higher than that which would have been accurately predicted based on chance alone. The resulting overall agreement indicated high levels of agreement between predicted and true group membership; in general, agreement of 70% or above are regarded as indicating high levels of agreement (Brennan & Prediger, 1981). Given that Joormann et al.’s model is primarily focused on the effects observed when people are in a depressed mood, rather than those in euthymic or remitted depressed moods, it is not surprising that the highest classification rates were found for the MDD group. Consistent with hypothesis 3, poorest sensitivity was observed for the remitted participants, with over half of these participants being classified as part of the control group and a fifth as part of the MDD group. Thus, this suggests potential clinical application of the predictors outlined in Joormann et al.’s model (as operationalised in the current study) for diagnostic purposes for classification of MDD and control status. However, this is not recommended in the case of the remitted depressed patients. It is necessary to note
that the variability in the remitted group is understandable given the heterogeneous nature of these samples (Just et al., 2001). For instance, the current sample was inclusive of individuals at different stages of depression recovery. Further, the present study did not manipulate the likely activating role of stress in cognitive vulnerability of this sample group (mood priming hypothesis; Back, 1967). Thus, it may be possible that the current sample were measured at a time when their cognitive biases/deficits were not operative (Just et al., 2001). Just et al. have argued that the use of remitted depressed samples is a logically “backward” subject selection strategy, in that participants are selected on the dependent variable (depression) and then are compared on the independent variable (cognitive vulnerability). Thus, this protocol makes it unclear whether negative cognitive biases observed in remitted depressed persons are representative of a cognitive vulnerability that contributed to the manifestation of the MDD episode, or due to the consequence of that depressive experience (i.e., scar hypothesis). However, this does not discredit the use of remitted depressed samples in this research, especially for studies in which researchers are interested in hypotheses regarding prediction of depressive relapse or cognitive functioning following depression. Future investigations are likely to benefit from the use of both remitted samples—in both mood-induced and euthymic conditions—as well as depression high-risk samples (e.g., those with depressed parents) to explore this cognitive vulnerability versus cognitive scar predictions.

The low classification rate for the remitted group is also understandable given Joormann et al.’s. (2007) cognitive model of depression is designed to separate depressed and non-depressed cognitive functioning rather than remitted depression status. Thus, a supplementary DA was performed (see Appendix N) to determine whether participants’ current depressed and non-depressed clinical status could be
distinguished based on the linear combination of Joormann’s cognitive predictors. The
discriminant function here found a strong link between groups and predictors. The
cluster of predictors for this subsequent DA is also consistent with those implicated in
Joormann et al.’s (2007) cognitive model. Quadratic classification procedures for this
supplementary DA found around 85% of participants were able to be properly
classified into their respective depressed and non-depressed groups based on the linear
combinations of the predictor variables outlined in Joormann et al.’s model. This was
significantly higher than that which would have been accurately predicted based on
chance alone. The resulting overall agreement indicated exceptionally high levels of
agreement between predicted and true group membership.

The cross-sectional design of the current work also makes it difficult to rule out
the possibility that the observed effect of clinical status is a consequence of elevation
in symptoms of affective distress rather than contributors to elevations in such
symptoms. It is crucial to consider the direction of these effects in prospective studies
in the future. The fact that these predictive effects could be observed within this small
sample is representative of strong associations between the different constructs.
Unfortunately, this sample size did prevent more fine-grained analyses on other
potentially interacting variables (e.g., the role of the number of episodes, treatment
history, and family history of depression). Future work would benefit from using
multivariate statistics to confirm these interactions given its potential to detect effects
that multiple omnibus univariate tests may miss.

**Summary**

In summary, depression is a highly heterogeneous disorder. Therefore, it is
unreasonable to expect that a signal casual pathway will explain a significant amount
of variability in this disorder. However, the results of this study show promising
evidence that the predictors implicated in Joormann et al.’s (2007) cognitive control model may effectively predict depressive status and MDD versus never-depressed clinical status. It does not appear to be able to predict remitted depression status using the operational definitions of the cognitive processes employed in the current dissertation. This suggests that interventions based on teaching effective emotional regulation strategies, such as problem solving instead of rumination and positive memory recall and cognitive control training to help improve updating of working memory have the ability to reduce the depressive episode in young, female samples.
Chapter 7

General Discussion

The main aim of the dissertation was to assess the validity of the cognitive model of depression proposed by Joormann et al.’s (2007). Recall from the literature review (Chapter 2), information encountered in the environment activates existing mood-congruent schemas from the person’s long-term memory (Dudai, 2002). In the context of depression, once activated, these schemas preferentially direct attention and retrieval to information that is consistent with its negative content (Beck, 1967; Bower, 1981). This may result in greater recognition of negative information (Matthews & Mcleod, 2005) and rumination on this negative material (Johnson et al., 2009; Nolen-Hoeksema, 1991). Rumination in the context of depressed affect further enhances accessibility to negative memories, by continuing to reactivate attention to core negative self-schemas (Ciesla & Roberts, 2002; Ciesla & Roberts, 2007; Joormann, 2004; Joormann, 2005, 2009; Joormann et al., 2008; Joormann et al., 2007; Lyubomirsky et al., 1988; Lyubomirsky & Nolen-Hoeksema, 1995; Robinson & Alloy, 2003). This in turn exacerbates and perpetuates the depressed mood state (Demeyer et al., 2012). On the other hand, adaptive mood repair appears to involve the alleviation of a negative mood by recalling positive material or accepting emotions (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Gross & Munoz, 1995; Kovacs, Joormann, & Gotlib, 2008; Kring & Werner, 2004). Here, the individual must activate and elaborate positive content in working memory, whilst also inhibiting connections to negative representations (Ochsner & Gross, 2005).

This dissertation consisted of three ERP studies of information processing of emotional word stimuli in a sample of female university students. Participants’ underwent diagnostic assessment using the Structured Clinical Interview for DSM-IV
Disorders, and were selected if they met criteria for one of the three experimental groups: MDD ($n = 30$), remitted depression ($n = 13$), and never-depressed healthy controls ($n = 30$). The three ERP studies aimed to examine the impact that clinical status had on processing emotional stimuli under conditions of self-referential encoding and attention (Study 1), recent episodic memory (Study 2), and working memory inhibitory control (Study 3). Based on Joormann et al.’s (2007) cognitive model of depression, the dissertation also aimed to determine whether group status could be predicted based on the linear combination of participants data obtained in these preceding experiments (Study 4). Taken as a whole, the dissertation results are theoretically consistent with Joormann et al.’s cognitive model and the research hypotheses. This chapter will review the overall study findings, with an attempt made to integrate them with each other and with existing data. It will outline the contributions that the findings make to the literature and their clinical implications. The final section specifies the dissertation limitations and future research directions.

### 7.1. Overview of Dissertation Findings

**Study 1.** The results of this study suggested MDD to be associated with more negative self-evaluations resulting in greater expectation of negative than positive stimuli (P3b), which is coupled with compromised right-parietal hemisphere activation. This electrophysiological negativity bias is positively correlated with severity of current and future depressed and anxious mood and self-reported level of rumination. This supports Beck’s (1967) cognitive theory of depression that activated negative schemas in depressed mood negatively skew data processing, which serves to maintain the negative affect. Never-depressed clinical status was associated with more positive self-evaluations. Remitted-depressed status was associated with fewer positive and negative self-evaluations compared to never-depressed and MDD status,
respectively. Thus, the less emotional internal set evident in remitted depression may explain the finding of statistically similar P3b for positive and negative stimuli in this group. It is possible that ERPs amplitudes are only sensitive to more extreme differences in incongruence between cognitive self-schemas and environmental expectations. Both the P2 and LPP data suggested that the susceptibility to depression may also derive from a lack of an innate or learned bias of the perceptual system. Specifically, it appeared that depressed and remitted-depressed individual failed to automatically identify interpersonal threatening information (P2s for negative trait adjectives). In the real world, this may create implications for social functioning. Current and remitted depressed participants also appeared to show impaired ability to turn attention towards positive stimuli in an attempt to reappraise mood in the face of stress (enhanced LPP to positive information). Consistent with previous work (e.g., Shestuk et al., 2005; Shestuk & Deldin, 2010), these ERP effects were not associated with behavioural differences in reaction time data. This suggests the unique ability of the ERP over behavioural assays in elucidating information processing biases in clinical research.

**Study 2.** The results of this study generally support the idea of mood-congruent recall memory (Bower, 1981). Consistent with Joormann et al.’s (2007) model, greater free-recall and recognition of negative relative to positive stimuli was positively associated with current and chronic depression and anxiety levels and use of rumination responses. Control participants showed greater LPNs for negative relative to positive words, suggestive of greater difficulty recalling negative information in this group. No LPN valence effects were found for the remitted participants. This may be the result of their less emotionally skewed cognitive schemas resulting in equal encoding of positive and negative stimuli, or low experimental power. No
divergent group ERP data were found for the FN400 or LPC. These results failed to replicate the work of Dietrich et al. (2000) who observed an increased positivity beginning approximately 250 ms post stimulus for old relative to new items in their control participants, which was lacking in the MDD group. The contradictory results between the two ERP studies may be a result of gender, age, and treatment differences between the experiments. Dietrich et al. used a mixed sample of participants (63% female) as opposed the all-female sample employed in the current study. Further, all of Dietrich et al.’s MDD participants were currently being treated with “psychotherapy” (the researchers did not elaborate on type of therapeutic approach) in an inpatient facility, and were an average of 7.7 years older than their control sample and 10.5 years older than the current study’s MDD participants. Given that decline in episodic memory is a common feature of aging (for a review see Buckner, 2004 and Hedden & Gabrieli, 2004) and that aging is known to reduce the Old/New FN400 and the LPC effects (Wolk et al., 2009), it is possible that Dietrich et al.’s smaller ERPs in their MDD group was confounded with ERP age effects. Further, Study 2’s null findings might also be the result of reduced opportunity for memory decay and/or the relative ease of the experimental task, in that it did not produce enough demands on the working memory system to show the predicted deficits. The impact of these variables should be further investigated in future work.

The results of the study further support the predictions that MDD is associated with more negative self-evaluations, resulting in greater expectation of negative than positive stimuli (enhanced N400; Kutas & Hillyard, 1980). This electrophysiological negativity bias is positively correlated with severity of current and future depressed and anxious mood and self-reported amount of rumination. These results are consistent with those of Dietrich et al. (2000) who also observed smaller N400 ERPs
for negative words compared to positive words in their depressed participants, which they suggested represented a neural correlate of depressed participants’ negative rumination. It is consistent with previous data that the amplitude of the N400 reflects the extent to which the self-relevant information is discrepant with a person’s self-concept (e.g., Watson, Dirtschel, Obonsawin, & Jentzsch, 2007). That is, stimuli incongruent to their prevailing mood and schemata requires greater cognitive resources compared to congruent stimuli, and as such, is associated with increased neural activity to process and respond appropriately. This result complements the P3b data observed in Study 1. It also supports Beck’s (1967) cognitive theory of depression that activated negative schemas in depressed mood negatively skew data processing, serving to maintain the negative affect. Never-depressed clinical status was associated with more positive self-evaluations resulting in greater expectation of positive stimuli. There was a trend of enhanced N400s in response to negative relative to positive stimuli in the control sample. Remitted depressed status was associated with fewer positive and negative self-evaluations compared to never-depressed and MDD status, respectively. Thus, similar to the P3b results in Study 1, the less emotional internal set evident in remitted depression may explain the finding of statistically similar N400 for positive and negative stimuli in this group.

**Study 3.** The electrophysiological results of Study 3 suggested that current and remitted depression was associated with over-mobilisation of anterior regions in the early processing stage of cognitive inhibition (conflict identification: Nogo-N2) of positive distractor stimuli. It is possible that this reflects rapid identification and avoidance of positive stimuli in the clinical groups. The finding that this effect was not present in the control group suggests further support of the hypothesis of a lack of the protective positive bias in individuals vulnerable to depressive disorders (Deldin et
al., 2000; Deveney & Deldin, 2004; Yang et al., 2011; see Taylor & Brown, 1988). These results mirror the findings of a lack of a positive protective bias in Study 1 (P2 and LPP data) and Study 2 (free-recall memory bias for positive stimuli in control participants). During later stages of cognitive inhibition, that is, during conflict regulation (Nogo-P3), individuals with remitted depression were found to exhibit hyperactive conflict monitoring during cognitive inhibition of negative distracter negative stimuli. They also showed reduced LPPs during removal of negative proactive interference items from working memory. This may suggest evidence of recruitment of suppression-based processes in remitted depression in an effort to maintain euthymic mood when confronted with stressful stimuli. Given that this data did not correlate with the performance data, this result seems to indicate that remitted depressed individuals need to recruit greater neuro-inhibitory resources for the purposes of stopping a prepotent response to negative stimuli with the same degree of efficiency as positive stimuli and as healthy controls. Thus, this electrophysiological profile may indicate active suppression of negative cognitive processes and content in the remitted sample in an effort to sustain their euthymic mood. Participants with MDD appeared to show marked difficulties disengaging from elaborative processing of once relevant but now irrelevant negative items from working memory (proactive interference deficits). Previous studies have indicated that problems in updating the content of working memory might underlie the sustained processing of negative material in ruminative responses (Joormann & Gotlib, 2008), which predicts both the onset and maintenance of depression (see Nolen-Hoeksema et al., 2008).

**Study 4.** The results of the discriminative analyses performed in Study 4 showed promising evidence that the processes implicated in Joormann et al.’s (2007) cognitive control model could effectively predict MDD versus never-depressed clinical
status. However, as measured in the current dissertation, these variables do not appear to be able to predict remitted depression status. It is crucial to note that the variability in the remitted group is understandable given its small size and the heterogeneous nature of these samples (Just et al., 2001). For instance, the sample was inclusive of individuals at different stages of depression recovery. Further, the dissertation did not manipulate the likely activating role of stress in cognitive vulnerability of this sample group (priming hypothesis; Beck, 1967). Thus, it may be possible that the current sample were measured at a time when their cognitive biases/deficits were not operative (Just et al., 2001). The overall discriminant analysis showed greater self-reported use of rumination and suppression, and less use of emotional reappraisal significantly differed between depressed and non-depressed participants. Depressed participants also reported greater free-recall, recognition memory, and proactive interference inhibitory control deficits for negative relative to positive stimuli. This cluster of predictors is consistent with those implicated in Joormann et al.’s (2007) cognitive model of depression. For the MDD group, high sensitivity (93.3%) and specificity (81.40%, 95% confidence interval: 66.59 – 91.58%) was observed. The fact that these predictive effects could be observed within this small sample is representative of strong associations between the different constructs. No significant differences were found for the negative bias distractor interference scores. The failure to replicate these behavioural findings here may be attributed to task differences between the studies, which may assess different inhibition processes. Specifically, it is argued that behavioural performance of the Go/Nogo task represented conflict monitoring inhibitory processes—which seem to be preserved in MDD—more so than conflict regulation, which appears anomalous in the disorder (Joormann, 2004; Goeleven et al, 2006). Study 4 clearly demonstrated the benefit of using multivariate
statistics to determine interactions between the cognitive processes implicated in depression, which may help to uncover effects that multiple omnibus univariate tests may miss.

7.2. Contributions the Dissertation Makes to the Literature

The dissertation’s series of studies have several academic and methodological strengths that make it a valuable contribution to the empirical literature in this area. The main strength of the dissertation is its assessment of emotional attention, explicit memory, and working memory inhibitory processing in the same sample of MDD, remitted depressed, and never-depressed individuals. It is the first study to statistically integrate these cognitive results with psychometric measures of rumination, depression, and emotional regulation using predictive discriminant analysis. The dissertation was guided by updated clinical models of depression and allowed for analysis of the clinical usefulness of Joormann et al.’s (2007) model. Further, the dissertation included a longitudinal design (re-test of depression scores over a one-month period). This allowed for the examination of which cognitive processes were associated with ongoing depression symptoms, thereby informing treatment interventions. Extending its contributions to clinical practice, the dissertation is in line with the research initiative for the latest edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). Specifically, the DSM-5 emphasizes a need to translate neuroscience research findings into a new classification system for mental illnesses based on underlying neural processes. While most behavioural and neuroimaging studies of cognition are extremely helpful, they typically only measure outcome data. Thus, they typically fail to capture the rapid sequence of information processing that occurs prior to response output. The dissertation studies capitalised on the excellent
temporal resolution of the ERP to reveal subtle activities at different stages of the information processing stream (e.g., Krompinger & Simons, 2009; Yao et al., 2010). Specifically, through careful extraction of ERPs using PCA methods the dissertation was able to distinguish vigilance from sustained attention processes (Study 1), memory recollection versus memory familiarity (Study 2), and conflict monitoring inhibition versus conflict resolution cognitive inhibition (Study 3). To this PhD candidate’s knowledge, Study 3 is the first ERP study of proactive interference inhibitory control in current and remitted depression. This information will allow for improvement of clinical interventions for female clients, which will likely result in reduced relapse rates for depressive disorders.

7.3. Limitations of the Dissertation

The results of the series of studies must be weighed in light of their limitations. First, the studies consisted of a young, female sample of university students. This reduces the ability to generalise the experimental results to low intelligence, male, or older populations. An all-female sample was selected to reduce any confounding differences in the ERP effects due to known reported gender effects (Hill et al., 2005; Ortigue et al., 2005; Walla et al., 2001;), especially in the case of emotional information processing (Killgore et al., 2001; Lee et al., 2002; Schirmer & Kotz, 2003; Schneider et al., 2000;). University students may be characterised by relatively good cognitive functioning, which might make it more difficult to observe cognitive biases and deficits due to ceiling effects. Their higher intellect and functioning are also unlikely to be truly representative of the general population, specifically to uneducated populations or those in inpatient facilities. Further, provided that only 50% of the MDD sample self-reported severe levels of depression on the BDI-II the applicability of the thesis results may not easily translate severely depressed people.
Another sample limitation involved the inclusion of participants in the MDD group who also met a secondary anxiety diagnosis. Attention biases for negative items have been reported in anxiety disorders, especially for threat-related material (see Williams et al., 1997). Specifically, clinical anxiety appears to be more associated with orientated forms of attention towards danger (Williams et al., 1997), usually evidenced by enhanced P3a amplitudes (Bruder et al., 2002) or implicit memory processes, usually evidenced by FN400 (Pauli, Dangler, & Wiedmann, 2005). Given that these effects were not observed in the current data set (see Study 1 and Study 2), and given that there were no changes to significant/non-significant effects when these participants were excluded from analysis (Appendix H), it could be argued that the secondary anxiety diagnoses were superseded by the more pronounced depression symptoms. Alternatively, these effects may not have been observed because depressive-toned (e.g., sad, loser, worthless) stimuli were selected over threat-based stimuli (e.g., scared, panicked, vulnerable). The decision to incorporate participants with comorbid anxiety disorders was made in an effort to increase the ecological validity of the results (Beuke et al., 2003; Ingram, 1989; Ingram & Hamilton, 1999), with the US National Comorbidity Survey reporting that 58% of individuals with MDD have a comorbid anxiety disorder (Kessler, Nelson, McGonagle, & Liu, 1996). However, because no such psychiatric control group was included in the study, the issues of specificity cannot be addressed.

The inclusion of participants who were currently taking SSRI medication is another sample limitation, given previous studies have found these drugs to reduce Amygdala activation to negative stimuli in both clinical (Fu et al., 2004; Sheline et al., 2001) and nonclinical samples (Harmer et al., 2006). In the current sample, medication status did not seem to have any effect on changing the direction or
significance of the experimental results (see Appendix Q). However, given the small sample of participants currently taking SSRI medication in the present study ($n = 4$) it is possible that there was not enough power to find any moderating effects here. Including participants taking SSRI medication has the benefit of enhanced ecological validity in generalizing to actual MDD population. The NICE (2004) guidelines recommend a combination of CBT and SSRI medication as the first-line treatment for severe depression. The MDD participants in the dissertation studies self-reported severe levels of depression. Thus, the result of the study may generalise to understanding the severely depressed client, who would most likely be medicated.

The heterogeneous, young, and small size of the remitted participant group reduces its generalizability to older, chronic, and medicated remitted patient groups. However, the remitted sample characteristics are argued to be a strength of dissertation, since older age (~ medium 40 years), medication use (typically SSRI), and recurrent depression (~ medium past major depressive episodes = 4) have already been shown to affect cognitive performances in remitted-depression (see Synder, 2012 for a review; Table A2; Appendix F). Further, the sample consisted of pre-adult onset depression, which may not readily correspond to profiles expected in adult-onset depression often used in previous work. This is an important consideration as some researchers have questioned whether pre-adult and adult-onset depression represents different subtypes of the disorder (Kaufman, Martin, King, & Charney, 2001). Thus, this research adds to the literature by providing novel insight into the relationship between ERP processing of emotional stimuli under conditions of encoding, episodic memory, and working memory inhibitory control in these young, medication-free remitted samples. However, these differences are still poorly understood and would benefit from further analysis. It would be interesting here to explore if differences
exist in the cognitive profiles between first-episode and recurrent depression samples. Previous work shows recurrent MDD patients have more serious cognitive impairments compared to first-episode MDD patients, such as greater negative attention biases (Chen et al., 2014), autobiographical memory (Nandrino et al., 2002), and executive function deficits (Karabekiroglu, Topcuoglu, & Gonentur, 20100). Similar to Nandrino et al. (2002), the current sample is a mix of first-episode and recurrent remitted depression. However, unlike Nandrino et al. the sample size was too small to analyse ERP differences between these two sub-groups. Differences between first-episode and recurrent depression are still poorly understood in terms of cognitive profiles and vulnerabilities, and future longitudinal investigation to explore these subsamples further would be beneficial.

The clinical consistency of the current remitted sample is uncertain as they are (most likely: Kessler et al., 1997) in an earlier phase of their illness. That is, it is unknown whether these participants with just one life-time depressive episode, will experience recurrent MDD episodes in later years, or will develop bipolar profiles. However, it may be argued that since the current remitted-MDD sample continued to reported slight elevations of depression and anxiety (as measured by the BDI-II and STAI-T: see Table 8) they presumably reflect a group susceptible to depression relapse. For instance, a substantial body of literature demonstrates that among remitted MDD patients, those with residual symptoms have a significantly higher risk of relapse compared to those without residual symptoms (Mojtabai, 2001; Judd, Akiskal, & Paulus, 1997). In fact, the presence of residual depressive symptomology appears the most accurate marker for depression relapse (Pintor, Torres, Navarro, Matrai & Gasto, 2004; Paykel et al., 1995; Judd et al., 1998). This effect appears irrespective of antidepressant or psychotherapeutic intervention (Thase et al., 1992).
Intriguingly, anxious agitation in remitted-depressed samples appears to predict MDD relapse, more so than any residual core mood symptoms (Hybels, Steffens, McQuoid, & Ramma Krishanan, 2005).

Another factor to consider when comparing the dissertation results with previous work is the fact that the current remitted depressed sample were un-medicated, whereas participants in prior work include those on antidepressant regimes (see Appendix F). Depression-related cognitive scars in the remitted group might have been more evident in the dissertation if participants in the remitted sample were allowed to continue to consult treatments, such as antidepressants or psychotherapy interventions. Research produces mixed data regarding the question as to whether cognitive biases and working memory deficits are precursors to depression. That is, are they trait-like susceptibilities (stable liability models), or whether the experience of depression creates a cognitive fault that did not exist prior to the episode (cognitive scars; Lewinsohn et al., 1981). It is true that these inconsistent results are primarily due to differences in experimental methodologies and sample characteristics, such as mediation use. However, antidepressant medications, such as SSRIs are known to positively influence emotional information processing (onset 7-14 days following medication commencement; Bhangwagar et al., 2004; Harmer et al., 2002, 2003a, 2004, 2006). Previous behavioural results have shown most cognitive impairments associated with depression resolve with recovery through medication, even when participants are continuing to consult these interventions (recovery is incomplete; Merens, Booij, & Van Der Does, 2008; Weiland-Fiedler et al., 2004). Thus, it can be argued that because the remitted-depressed patients are medication-free, their results are not confounded by potential medication effects thereby making it more likely that cognitive scars might be elucidated. Thus, medicated remitted-depressed samples
may confound observation of predictive variables, such as residual mood symptoms, given that antidepressants work to suppress these experiences.

The dissertation hypotheses pertained to main effects or interactions with stimulus valence and diagnostic classification. Thus, mixed ANOVA is the dominant statistical approach, and that suggested by Luck (2005) for analysis of ERPs. The number of participants in the remitted depressed group was significantly smaller than that in the control or MDD groups. As outlined in Appendix L, assumptions of normality were met within this group. However, one may question if this sample was too small to detect the expected ANOVA effects. The size of the remitted participants deemed necessary to detect hypothesized effects using mixed ANOVA was based on effect sizes observed in the previous literature. For instance, in a similar ERP study, Vanderhave et al., (2012) observed reduced N450 amplitudes on trials requiring disengagement from negative distractor items in their sample of 15 remitted-depressed participants. Their results were also analysed using a mixed ANOVA approach, in which this significant valence-by-group interaction showed a large effect size ($\eta^2 = .20$). The significant Nogo-N2 and Nogo-P3 effects for remitted participants during inhibition of negative distractor stimuli observed in the current dissertation (Study 3) was also in the large range ($\eta^2 = .419 - .434$; see Section 5.3). This suggests that the experiment possessed adequate power for the hypothesised analysis. Obviously, replication of experimental findings for the remitted participants will ultimately determine if effects are true representations of this population. It is wondered if the sample size was larger, would observed null results have become significant for the remitted-depressed participants (for instance, the Nogo-N2 or Nogo-P3 results for the proactive interference stimuli). This is an important question, which requires further exploration and analysis in a larger sample.
In summary, the results of the remitted-depressed participants can only be generalised to young, early-onset, undedicated female samples. Future work would benefit from investigating the influence of client variables, such as depression chronicity, age of onset, treatment modality, and residual symptoms (inclusive of anxiety) on cognitive processing of emotional stimuli in remitted-depression. It would be beneficial to compare cognitive profiles between participants who experience single versus remitted major depressive episodes. Such casual relationships can only be tested in large-scale prospective studies and not with a cross-sectional remission design. Despite this consideration, the dissertation results obtained from the remitted sample are promising in identifying potential cognitive vulnerability factors in depression which can be observed using the ERP.

Considerations of the methodological design also need to be highlighted. First, the study included self-report measures of depression and anxiety symptom severity, rumination, and emotional regulation processes. These indirect psychometric measures of emotional and cognitive function prevent casual inferences being made. Untangling the nature of the associations found in the data set will require longitudinal studies with experimentally manipulated rumination and emotional regulation protocols.

The studies involved the use of verbal stimuli and thus, cannot be generalised easily to emotional pictorial or facial stimuli. It might be argued that pictorial or facial stimuli may have more ecological validity than word stimuli; however, in terms of research internal validity they are also more complex. Specifically, arousal and valence characteristics are more difficult to control in pictorial and facial compared with verbal stimuli. The arousal and valence dimensions are known to significantly affect the morphology of ERPs (Luck, 2005), and can therefore easily result in a Type
III error (finding a statistical difference but attributing it to the wrong hypothesis). Further, both theory and research evidence indicate that idiosyncratic data consistent with one’s internal schematic set is required to generate mood-congruent information processing biases (Beck, 1967; Matthews & MacLeod, 2005). For this reason, the present study utilised a self-referential encoding task in Study 1. As opposed to facial or photographic stimuli, verbal stimuli can be easily incorporated into this procedure and therefore the choice of stimuli for the current study. This is a particular methodological strength of the dissertation, as this elaborative processing strategy ensures enhanced recall compared to structural or semantic encoding of the same material (Klein, Loftus, & Burton, 1989). This is particularly the case in clinical samples, such as depression (Shestyuk & Deldin, 2010). Thus, one can assume that the experimental stimuli were effectively encoded prior to the subsequent examinations of participants’ explicit memory (Study 2) and working memory inhibitory control (Study 3).

At an ERP design level, the limited number of trials for Study 2 and 3 reduced the study’s ability to assess ERPs for error data. As such, it is currently unknown if ERPs evoked for failures in source memory in the Old/New tasks or incorrectly inhibited nogo stimuli evoke different neural activation than the typically Nogo-N2 and Nogo-P3 waves observed for successful inhibition trials. It is also acknowledged that this information is correlational in nature, and although the results are consistent with theoretical expectations, no causal interpretations can be made. The cross-sectional design also makes it difficult to rule out the possibility that the observed interactions are the result of the consequences or symptoms of depression. However, despite these limitations, the results of the dissertation studies are exciting in that they help inform how prevention and treatment programs for depression may be benefited.
7.4. Clinical Implications of the Dissertation Findings

The dissertation findings suggest that interventions that strengthen top-down attention control and promote acceptance may help young, female depressed clients to (a) disengage from negative cognitions (Study 1, 2, and 3), (b) enhance attention to positive stimuli (Study 1 and 3), and in the context of remitted depression (c) reduce suppression tendencies (Study 3). Together, this may assist patients to better manage their mood and reduce vulnerability to relapse. This could be achieved through cognitive behavioural therapy (CBT). Extensive empirical data supports the effectiveness of CBT for depression (mean Cohen’s \(d=0.67\); Cuijpers, van Straten, Bohlmeijer, Hollon, & Andersson, 2010). Given that improvement of attention control appears to have a pivotal role in the origin, maintenance, and recurrence of depressive episodes, adjunctive therapies to CBT aimed at strengthening attentional control processes might aid in the treatment. Research shows that attention biases to emotional information can be trained and untrained (Derryberry & Reed, 2002; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). As with training to divert attention away from negative stimuli, preliminary data shows that individuals can be trained to selectively direct attention to positive stimuli (Wadlinger & Isaacowitz, 2008). These results have yet to be replicated in clinically depressed samples. Preliminary studies using this simple attention retraining practice have been shown to be successful in reducing symptoms of clinical (Papageorgiou & Wells, 2000; Siegle, Ghinassi, & Thase, 2007) and subclinical depression (Wells & Beevers, 2010), after as little as two weeks of training. Consistent with Joormann et al.’s (2007) and Beck’s (2008) models, reductions in negative attention biases using these attention training interventions have been found to be associated with reductions of
rumination (Cohen’s $d = 1.26$; Siegle et al., 2007) and to mediate improvements in depressive symptoms ($r = .52$, Cohen’s $d = 1.04$; Wells & Beevers, 2010).

The findings of Study 1 and 3 suggested a lack of a protective emotional processing bias in depressed and remitted samples. Thus, interventions that train focus on positive information in one’s environment may be beneficial. Positive psychotherapy (PPT) interventions offer a promising way to increase positive cognitions, feelings, and behaviour in depressed patients (Seligman, Rashid, & Parks, 2006). A recent meta-analysis of 51 PPT interventions with 4266 individuals found this method significantly decreases depressive symptoms (mean $r = -.31$; Sin & Lyubomirsky, 2009). Therapeutically, mental health professionals may improve their depressed patients by increasing the processing of positive material by explicitly placing the depressed patient in contact with positive reinforcing contexts. This can be achieved through external behavioural activation interventions (Syzdek, Addis, & Martell, 2010); or in-session interventions, such as by strengths-based approaches or by verbally reinforcing the depressed patients’ positive qualities until they start to become more aware and accepting of them. As with training to divert attention away from negative stimuli, preliminary data shows that individuals can be trained to selectively direct attention to positive stimuli (Wadlinger & Isaacowitz, 2008). Interestingly, this brief (one session) positive-training paradigm was observed to result in enhanced inhibition for negative stimuli in a subsequent visual stress task in a sample of non-depressed university participants (Wadlinger & Isaacowitz, 2008). These PPT techniques have been found to reduce depressive relapse (Fava & Ruini, 2003). Further, preliminary evidence suggests that increasing activation of positive material in working memory may also reduce the lack of positive affect, emotional regulation deficits, and emotional cognitive biases that characterise depressive
disorders. The non-specific nature of these approaches provides transdiagnostic applications in the treatment of psychopathology. Therefore, they may not only reduce the individual’s vulnerability to depressive relapse, but also protect them from susceptibility to other psychopathology in the face of a diverse range of triggers. This provides an ecologically valid intervention, as depression—as in the current sample—is a highly comorbid disorder (Kessler et al., 2007).

The results of Study 3 suggested that remitted depressed individual’s exhibited hyperactive conflict monitoring during cognitive inhibition of negative distracter negative stimuli. Given that this data did not correlate with the performance data, this result seems to indicate that remitted depressed individuals need to recruit greater neuro-inhibitory resources for the purposes of stopping a prepotent response to negative stimuli with the same degree of efficiency as positive stimuli and as healthy controls. Thus, this electrophysiological profile may indicate active suppression of negative cognitive processes and content in the remitted sample in an effort to keep their euthymic mood. It is well established that suppression-based techniques are ineffective in mood regulation. Rather, these clients would probably benefit from formal training in mindfulness based interventions. Mindfulness is a mental mode characterised by purposely focusing attention to present-moment experience without judgment, elaboration, or emotional reactivity (Kabat-Zinn, 1990). Mindfulness interventions may also work to improve cognitive control and emotional reactivity in depressed patients. Random control trial studies support the therapeutic value of mindfulness for the treatment of depression (Kuyken et al., 2010; Ma & Teasdale, 2004; Teasdale et al., 2000) and on decreasing the rates of depressive relapse (Bondolfi et al., 2010; Teasdale et al., 2000). It provides a strategy for patients to engage in a lived experience rather than react automatically while ruminating about
distress. Research finds lower levels of negative affect, Amygdala reactivity, and negative rumination, and higher levels of positive mood, well-being, and working memory capacity with mindfulness training (Anderson, Lau, Segal, & Bishop, 2007; Carmody & Baer, 2008; Davidson et al., 2003; Jha, Stanley, Kiyongag, Wong, & Geifand, 2010; Way, Cresswell, Eisenberger, & Lieberman, 2010; Zeidan, Johnson, Diamond, David, & Goolkasian, 2010). A recent meta-analytic review of 727 studies, of which 39 were analysed, found mindfulness interventions were associated with a relatively large effect size (Hedges’ g=0.95) for improving depressive symptoms (Hofmann, Sawyer, Witt, & Oh, 2010). Repeated engagement of cognitive control and emotional regulation during the practice of mindfulness may serve to strengthen these processes (Jha et al., 2010; Williams, 2010). As a metacognitive form of awareness, mindfulness involved the process of decentring and a shifting of cognitive sets that enable effective reappraisal of circumstances. It emphasises non-biased attention to one’s current experience, whether it be negative or positive in nature. Therefore, it also presents a strategy to reduce suppression or the avoidance of processing positive material, characteristic of current and remitted depression.

As observed in the dissertation’s series of studies, the ERP may provide a valuable clinical tool to assess disrupted cognitive information processing. The high temporal resolution of the ERP allows for more sensitive assessment of the specific cognitive components disrupted in the depressed client (i.e., ERPs can distinguish between orienting, selective, and sustained attention). These benefits might compensate for their low spatial resolution and their limited ability to detect changes occurring in deep sub-cortical structures (e.g., Amygdala). Further, compared to other neuroimaging techniques, ERPs are not expensive to use and do not require extensive training to administer and interpret. As such, there are opportunities for the clinical
utilisation of the ERP as an objective marker of cognitive processing in depression treatment (Alhaj, Wisniewski, & McAllister-Williams, 2011). Although requiring further replication, the current research suggests that the P3b (Study 1) and N400 (Study 2) may be used to provide information on schema activation and negative expectation (e.g., hopelessness) in depression. Greater Nogo-N2s during inhibition of negative distractor interference items and enhanced Nogo-P3 during removal of negative proactive interference items may constitute use of suppression-based processes (Study 3). It will be interesting to determine if future research will validate (through further analysis and replication) the use of these ERPs as clinical indicators of increased risk for depressive relapse. Given that previous work indicates that the P3 are under partial genetic control (Katsanis, Iacono, McGue, & Carlson, 1997), it might also have potential as appropriate endophenotypic indicators for depression vulnerability (Kemp et al., 2009). Thus, ERP information for emotional information processing in the depressed client may help the clinician discover and provide a more detailed treatment plan. For instance, CBT based interventions focused on testing negative schemas might be selected where biased P3b or N400 effects for negative stimuli are observed. Alternatively, attention retraining interventions or mindfulness based skills might be specifically selected where insufficient anterior P2 activation to negative stimuli and ruminative response processes are observed to be part of the client’s cognitive profile. This individualised treatment planning on the cognitive level may help to improve treatment recommendations that are based on manualised protocols developed almost 60 years ago (Beck, 1967). This more personalised and data-driven approach to clinical service may allow for faster symptom improvement for the individual client. However, it is important to note that the findings in the thesis relate to group profiles and do not necessarily indicate the appropriate treatment for an
individual. Further, to be clinically useful, experimental group results must also be relevant to the defined group of patients in a particular clinical setting. As outlined in section 7.3., the study results are limited in their applicability of the dissertation results to older, male, adult-onset, and very severely depressed patients. Directions for future investigations in attempt to extend understanding of individual and group profiles for these populations are outlined next.

7.5. Future Directions for Investigation

In future research endeavours it will be helpful to examine different subtypes of depression. It will also be beneficial to confirm the specificity of the current findings by directly comparing emotional information processing in depression with other emotional and cognitive psychopathologies, especially with anxiety disorders and disorders marked by impulsivity (e.g., substance misuse, PTSD, OCD, ADHD, and bulimia nervosa). Future studies should also explore ERP profiles across the different brain locations, particularly lateralisation effects given atypical hemisphere activation in depression (Davidson, Pizzagalli, & Nitschke, 2009). Statistical data reduction techniques, such as PCA, should also be employed in these future electrophysiological investigations to better differentiate ERP components and subcomponents, which may help reduce some of the disparity between study results (e.g., P3 data, see Section 2.3.1.). Further source localisation techniques such as the current source density (CSD) estimates using spherical spline surface Laplacian algorithm (Perrin, Perrier, Bertrand, & Echallier, 1989) could also help with identifying key neurogenerators. Given the recent push towards integration of cognitive neuroscience research in depression with neurochemical abnormalities (e.g., Beck, 2008). Future research should also consider neurotransmitter associations with ERPs and abnormal cognitive functioning. The specific effect of medication status on
the obtained results should also be the focus of further investigation. Longitudinal investigations are needed to assess individual differences in these cognitive profiles prior to the onset of depression, during a depressive episode, and in symptom remission. This future work will encourage the development for more accurate neuro-cognitive models of depression, which will support improvements in how we can treat this recurring disorder (APA, 2013).

A further problem with this literature is that there are a wide range of cognitive tasks used that tap a wide range of fundamental processes (e.g., in attention: Orienting, selective attention, disengaging attention, attention for visual vs. verbal stimuli). Relationships among these cognitive tasks are not well-established and finding significant effects appears to be dependent upon running those tasks in highly specific ways. Future research should decide which particular attention and memory tasks are most reliably associated with depression symptoms, and under what conditions individuals with depression symptoms or at risk for depression exhibit biases. Future research may also benefit from investigating the impact of clinical variables, such as chronicity, age of onset, treatment modality, on emotional information processing of remitted depressed patients. This will help to explain possible mediating factors leading to cognitive biases and deficits in MDD. The direction of these hypotheses associations might also be better investigated using cross-lagged experimental designs. In crossed-lagged design, each participant would undergo each level of the treatment conditions thereby acting as their own experimental control. For instance, in a very simple investigation, such as one that studies the impact of depressed mood on cognitive control deficits, the experiment might be conducted as follows. On the first day of the experiment, the experimental sample of depressed and non-depressed participants are divided in half with one half the sample’s completing a Go/Nogo task
following mood-induction, while the other group complete the Go/Nogo task without mood-induction procedures. Both error rates and Nogo-ERP amplitudes are taken as the measure of cognitive control deficits. On the second day the conditions are reversed; that is, individuals who underwent the Go/Nogo task following mood-induction would now complete the task again under their naturally occurring mood state and vice-versa. The size of the effect will be the difference of error rate and Nogo ERPs on the days with and without mood induction. Future investigations will also need to examine the results to determine if the cognitive effects observed in these studies similarly occur in older or male populations.

7.6. Conclusion

The extent to which depressed individuals are impaired in inhibition of emotional stimuli from working memory varies by the specific sub-component of cognitive control and clinical status. For instance, MDD appears to be more related to difficulties expelling negative information from working memory than to difficulties controlling access of negative material to working memory. Remitted depression appears to be associated more with active suppression processes, involving greater recruitment of prefrontal areas to block the entrance and to remove negative items from working memory. Data from this dissertation also suggests that when assessing for cognitive control dysfunction in depression, behavioural data appears lacking in isolation, with the ERP data providing detailed insights into abnormal cognitive control processes. Consistent with Joormann et al.’s (2007) model, the dissertation results are suggestive that poor proactive interference working memory inhibitory control results in sustained activation of irrelevant negative stimuli in consciousness, leading to rumination and facilitation of long-term memory for negative events (Hertel, 1997; Joormann, 2004; Joormann, 2005; Joormann et al., 2008; Linville,
1996; Nolen-Hoeksema et al., 2008). This exhausts their cognitive resources, limiting their ability to reappraise or recall mood-incongruent information to regulate their mood. This appears to increase the cycle of negative cognitions, sustaining depressed mood. Based on these resultant data, 67.1% of participants, with 93.3% of MDD participants, were able to be properly classified into their respective depressed and non-depressed groups based on the linear combinations of the predictor variables outlined in Joormann et al.’s (2007) model. Thus, the use of this cognitive model and ERP measures of cognitive processes during clinical assessment may serve in more detailed selection of essential clinical interventions with the depressed female patient.
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Appendix A

Glossary of Terms

**Attentional Avoidance**: Attention is preferentially allocated away from information. It appears secondary to facilitated attention and difficulty in disengagement. Research revealing evidence of attentional avoidance has typically employed the dot probe paradigm with stimulus duration of 500 ms (Chen, Ehlers, Clark & Mansell, 2002; Koster et al. 2006; 2005).

**Autobiographical memory**: Memory for an individual’s life that consists of both episodic (details of personal experiences) and semantic (general knowledge) details (Conway & Pleydell-Pearce, 2000).

**Bottom-up Processing**: The fast, automatic, implicit, and preconscious processing of information based on the salient features of the stimulus and/or idiosyncratic memory associations. Also known as data-driven or associative information processing (Clark & Beck, 2010; Ochsner & Gross, 2005).

**Cognitive Control**: An executive process defined as identifying and evaluating the multiple responses during task completion and subsequently resolving such conflict and guiding behaviour towards intended goals (Botvinick et al., 2001; Botvinick et al., 2004; Botvinick, 2007; Desimone & Duncan, 1995).

**Cognitive Inhibition**: The ability to restrict or remove stimuli or mental processes from working memory that are irrelevant to the task at hand (MacLeod, 2007).

**Cognitive Schema/Schemata**: An organised mental structure of an individual’s idiosyncratic knowledge and assumptions of self, other, and the world which is used for interpreting and processing information (Beck, 1967).

**Conflict Monitoring**: An early sub-component of cognitive inhibitory control that involves the identification and evaluation of multiple responses during task completion. It is has been associated with enhanced Nogo-N2 ERPs (Botvinick et al., 2001; Krompinger & Simons, 2010).

**Conflict Regulation**: A sub-component of cognitive inhibitory control in which centres responsible for top-down inhibitory cognitive control (e.g., ACC) activate to reduce the impact that distracting/conflicting information has on task performance. Specifically, it is the process by which this conflict is resolved and behaviours are guided towards intended goals. It has been associated with enhanced Nogo-P3 ERPs (Botvinick et al., 2001; Krompinger & Simons, 2010).

**Context Updating**: Updating one’s mental representations of the current environment (Donchin, 1981). For instance, updating one’s world view when they are presented with positive information when they predominantly anticipate negative events.
Directed-Forgetting Task: A task used to index cognitive inhibition or forgetting. Participants engage in an intentional or incidental learning task. Half-way through this task participants are informed that the trials presented so far are ‘to-be-forgotten’ (TBF) because they were merely practice. However, at the end of the subsequent list, participants are then asked to recall all of the previous items including those that they were instructed to forget. Efficient inhibitory control or forgetting is indexed by significantly more ‘to-be-remembered’ material recalled than TBF material (Paz-Caballero et al., 2004).

Elaborative Attention: Also known as thoughtful attention. It involves the strategic, top-down processes which form or reinforce association between triggered schemata (Williams et al., 1988; 1997).

Electroencephalograph: A test that measures and records electrical activity of postsynaptic dendrites of millions of pyramidal neurons of the brain, in most cases via electrodes that are applied to the scalp.

Emotional Reappraisal: An antecedent-focused strategy that works to alter the experience of emotion by changing the focus of a person’s thoughts (usually towards positive events or problem solving) during stress (Gross & John, 2003).

Emotional Regulation: Emotional regulation is a set of processes that allow for the altering of emotional experience or expression (Gross & John, 2003).

ERP Amplitude: An index of the magnitude of neural activation, possibly reflecting the raw number of co-activated neurons (Luck, 2005).

ERP Arousal Effect: The enhanced ERP positivity occurring around 300-400 ms following presentation of high-arousing compared to low-arousing stimuli (Chiu et al., 2008; Codispoti, et al., 2006; Cuthbert et al., 2000; Delplanque et al., 2006; Dolcos & Cabeza, 2002).

ERP Latency: An index of temporal processing of stimuli (Luck, 2005).

ERP Valence Effect: The observation that emotional stimuli reliably modulate ERP components, appearing as early as 100 ms post stimulus presentation (Esslen et al., 2004; Ortigue et al., 2004; Pizzagalli et al., 2002; Pourtois et al., 2004).

Event-Related Potential (ERP): Positive or negative voltage fluctuations that result from sensory, motor, or cognitive events that are repeated and averaged to resolve them from the flux of background electroencephalograph (EEG). Typically named by their voltage—P for positive and N for negative—and by their order of appearance (i.e., P1, N1, P2, N2, and P3) or time of manifestation (i.e., P100, N100, P200, N200, and P300).

Executive Functions: Executive functions (also known as cognitive control/executive control/supervisory attentional system) are defined as basic cognitive control mechanisms that contribute to more complete cognitive functioning. They are considered to be volitional and effortful. They are centrally located in the frontal lobes and maintain tight connection throughout the brain (Miyake et al., 2000; Williams et al., 2009).
Explicit Memory: Also called declarative memory. It involves the conscious, intentional memory of experiences and information.

Familiarity: A feeling or sensation that a stimulus has been previously observed, but this is not accompanied by any contextual information about that experience (Jacoby, 1991; Mandler, 1980; Rugg & Curran, 2007; Yonelinas et al., 2002).

Go/Nogo Task: An experimental task typically used to assess behavioural and/or cognitive inhibitory control. It involves the presentation of a series of Go and Nogo cues. Participants are required to respond as quickly as possible to Go cues and to inhibit responses to Nogo cues (Aron, 2007; Chiu et al., 2008; Dillon & Pizzagalli, 2007; Smith et al., 2008).

Hypervigilance: An enhanced state of sensory sensitivity. It involves perpetual scanning of the environment for threat (e.g., sights, sounds, people, and smells).

Implicit Memory: The unconscious, automatic memory of knowledge and skills which aid in task performance.

Internal Shifting/Switching: Internal shifting is based on the ability to shift focus back and forth between mental sets or operations. The ability to shift requires the disengagement of a previous set which is no longer relevant, and an activate engagement to a new, relevant set. An example of a task that requires shifting is the neuropsychological test, Trails Making B (Arbuthnott & Frank, 2000). In this task, participants are asked to connect circles that contain either a number or a letter in alternative ascending order (e.g., 1-A-2-B-3-C) until they reach the number 13. This requires that they shift back and forth from counting by number to moving to the next letter of the alphabet (Miyake et al., 2000).

Memory Search: Involves the effortful and controlled attempted recall of a memory trace from long term storage.

Mood-Congruent Memory: A memory process that selective retrieves memories that match (are congruent with) one’s mood.

Mood-Congruent Processing: The tendency to favour processing of information in the environment that is consistent with a person’s current mood or mental status.

Negative Affective Priming Task: An experimental task typically used to assess emotional information processing. In the NAP task, two consecutive trials are presented to participants; each contains a target and a distracter stimulus (e.g., positive, negative, or neutral facial expressions). For each display, participants are required to indicate the emotion of the target and ignore the distracter. A negative bias is indicated by faster response latencies to negative targets that follow negative distracters on a previous trial.

Oddball Paradigm: An experimental task used in event-related potential (ERP) research which involves a Bernoulli (randomised) presentation of two classes of stimuli. One is a frequently occurring ‘standard’ stimulus, and the other is an infrequently occurring novel
or ‘oddball’ stimulus. Within this paradigm, all stimuli evoke ERP components which are said to represent the pre-attentive activation of the sensory input to subcortical relay nuclei and cortical sensory areas (e.g., P1, N1, and P2). For the oddball stimulus, however, these early processing waves are followed by a P300 component, which is said to represent stimulus evaluation and/or categorization.

**Oddball Stimulus**: A stimulus that occurs within the Oddball Paradigm. It is presented infrequently relative to all other stimuli and has distinct characteristics (e.g., a different tone among auditory stimuli).

**Old/New Paradigm**: An experimental task typically used to measure explicit recognition memory in which participants’ are required to learn a list of stimuli (study phase). They then perform a recognition test, in which they are presented the studied stimuli intermixed with distractor stimuli (test phase). The task requires the participants to identify if they recognise the stimuli as part of the studied list (old stimulus) or not (new stimulus).

**Orienting Attention**: Automatic attention directed towards a novel or potentially threatening stimulus (Posner, 1980).

**Priming**: Automatic, bottom-up processes involved in strengthening information schemata, making them more available (Williams et al., 1988, 1997).

**Resistance to Distractor Interference** : Denotes a form of cognitive inhibitory control that resists or resolves interference from information in the external environment that is irrelevant to the current task (Friedman & Miyake, 2004).

**Resistance to Proactive Interference**: Denotes a form of cognitive inhibition, which involves the ability to resist or remove memory intrusions that were once relevant, but have since become irrelevant to the current task (Friedman & Miyake, 2004).

**Resistance to Prepotent Responses**: Involves the purposeful inhibition of a prepotent or automatic response when necessary (Friedman & Miyake, 2004).

**Recollection**: When explicit memory for contextual information can be reconstructed about the stimulus.

**Rumination**: Focused attention and thoughts about the causes and consequences of one’s distress (Johnson et al., 2009; Nolen-Hoeksema, 1991).

**Selective Attention**: Goal-directed focus on one aspect of the environment, while ignoring irrelevant aspects.

**Semantic Incongruence**: When the content of presented semantic information is unexpected. For instance, “I take coffee with milk and dog” (Kutas & Hillyard, 1980). It evokes the N400 ERP.

**Sternberg Working Memory Task**: An experimental task used to index working memory storage and updating processes. Participants encode a number of items into short term
memory. A probe item is subsequently presented to which participants are to indicate if it was or was not presented in the preceding memory set.

**Stroop Task:** An experimental task typically used to measure attention control and flexibility. It involves the presentation of a series of colour names in different coloured ink. Participants are required report ink colour of each word; that is, they are required to manage their attention to inhibit or override one response (reading) in order to perform the other (report ink colour). The task takes advantage of the observation that word reading is an automatic ability that interferes with colour naming when a colour word is printed in a different colour (i.e., word red printed in green).

**Suppression:** A response-focused strategy that consists of attempts to prevent or reduce ongoing emotion-expressive behaviour or thoughts (Gross & John, 2003).

**Sustained Attention:** Also known as sustained processing, vigilance, and concentration. It involves the ability to maintain consistent attentional focus over a period of time.

**Top-down Processing:** The slow, deliberate, explicit, and conscious form of information processing based on task rules/knowledge (Clark & Beck, 2010; Ochsner & Gross, 2005).

**Working Memory:** A system responsible for the temporary holding and processing of information. This can include new and already stored data. It includes three main components, (1) the central executive, which acts as a supervisory system and controls the flow of information to and from its slave systems the (2) phonological loop and (3) visuospatial sketchpad. These slave systems are said to be short-term storage systems dedicated verbal and visuospatial domains, respectively. The central executive is similar to the construct of a supervisory attentional system regulating thought and goal setting and to the construct of attentional control (Baddeley & Hitch, 1974).

**Working memory Updating:** The ability to manipulate and create new representations in working memory to reflect task demands.
Appendix B

Study 1 Pilot

The aim of this pilot study was to investigate electrophysiological evidence of encoding biases in dysphoric mood states. Specifically, the results of the pilot study examined the required intensity of stimulus valence and arousal characteristics, stimulus repetition rates, the suitability of neutral stimuli as a base condition, and to assess potential impacts that gender, comorbid anxiety, handiness and medication status had on the anticipated experimental P3b results.

Participants

A power analysis was conducted to determine the number of participants needed to detect the effects using a power of 0.8 and testing at the 95% confidence interval. This was conducted using guidelines suggested by Cohen (1988). Based on a review of the literature, a medium effect was considered appropriate for the proposed pilot studies. To achieve this effect size with a power of .80, 40 total participants was deemed necessary (20 per group: Dysphoric and Control). This sample size is consistent with the sample sizes typically included in ERP investigations in this area (e.g., Deldin et al., 2010; Ilardi et al., 2007; Rushsow et al., 2008). A sample size of 50 was therefore selected to account for loss of data due to poor EEG signal-to-noise ratio (approximately 5%) and incorrect responding (approximately 5%), and high levels of anxiety or stress on day of EEG testing (approximately 10%).

The pilot study’s 50 participants were recruited from a sample of 197 first-year psychology students who completed the online screening questionnaire and met inclusion criteria (see Procedures section). Ten participants were excluded from the final analysis due to a number of reasons (see Appendix A for reasons). Of the remaining 40 participants, 20 were allocated to the Dysphoric group and 20 were
allocated to the Healthy Control group. Group placement was determined according to their DASS-42 scores:

1. **Current Dysphoric** group (Dysphoric; \( n = 20 \)): Included participants who scored above the 80\(^{th}\) percentile on the Depression subscale of the DASS-42 and denied a current or past history of other DSM-IV-TR Axis I disorders.

2. **Healthy Control** group (Control; \( n = 20 \)): Included participants who scored below the 65\(^{th}\) percentile on all DASS-42 sub-scales (within one standard deviation), and denied a current or past history of psychopathology or psychotherapy.

The resulting 40 selected participants consisted of 26 females (65% of sample) and 14 males. Their age ranged between 17 and 45 years, with a mean of 22.32 years and standard deviation of 6.38. Two participants were left-handed (5% of sample). Participant groups did not differ on their self-reported family history of mental disorders as per self-report (Mann-Whitney U Test, \( p = 1.59 \); see Table A3). As per self-report, five participants in the Dysphoric Group were currently taking an SSRI\(^8\) and one was taking a tricyclic antidepressant\(^9\) for their depressive symptoms.

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\(^{8}\) Medications and dosages were as follows: *participant a* and *participant b* - 50mg Zoloft (sertraline) /daily; *participant c* - 20mg Aropax (paroxetine)/daily; *participant d* - 20mg Lovan (fluoxetine) /daily; *participant e* – 120mg Cymbalta (duloxetine HCl)/daily.

\(^{9}\) Medications and dosages were as follows: *participant f* - 25mg Clomipramine (Anafranil) /daily.
Table A1

Participant Demographics: Ms and SD (in parenthesis) for the Pilot Studies

<table>
<thead>
<tr>
<th></th>
<th>Dysphoric</th>
<th>Control</th>
<th>Test of Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>23.40 (6.56)</td>
<td>21.25 (6.17)</td>
<td>$F(1,39) = 1.14, p = .292$</td>
</tr>
<tr>
<td>Years of Education</td>
<td>13.70(1.63)</td>
<td>13.83 (1.85)</td>
<td>$F(1,39) = 0.05, p = .822$</td>
</tr>
<tr>
<td>Female/Male ratio</td>
<td>13 ♀/7 ♂</td>
<td>13 ♀/7 ♂</td>
<td></td>
</tr>
<tr>
<td>Handiness</td>
<td>19 right/1 left</td>
<td>19 right/1 left</td>
<td></td>
</tr>
</tbody>
</table>

Materials

Experimental Stimuli. Emotional nouns were selected from the Affective Norms for English Words (ANEW) database (Bradley & Lang, 1999), which lists valence and arousal ratings for over 1,000 English words. The ANEW database was later re-normed by Stevenson, Mikels, and James (2007) to measure the emotion elicited by each word (happiness, anger, sadness, fear, disgust) on a scale of 1-5 (1 = not at all, 5 = extremely) to allow for more discrete emotional categorization. Data here reflects the norms from this latter bank. For the purpose of the pilot studies, the 18 words with the highest “happiness” ratings (> 4.5/5, e.g., joyful, friendly, nice), 18 words with the highest “sadness” ratings (>3.5/5, e.g., loser, helpless, rejected) that could be used in a self-descriptive manner were selected for the Positive and Negative stimuli, respectively. A further 62 words which were rated as not scoring high on any emotional category (< 3/5 on all scales, e.g., modest, curious, quiet) constituted the words selected for the Neutral stimuli. All words were then distributed into three lists (List A, List B, and the Distracter List). List A and B each comprised of six positive, six negative, and 28 neutral words. They were counterbalanced over participants for
use as the Target lists for the pilot of study 1 (emotional oddball task) and as the new
list for the pilot of study 2 (Old/New task). The Distractor list consisted of six
positive, six negative, and 28 neutral stimuli and was used as the distractor
interference Nogo stimuli list for the Inhibition task for the pilot of study 3. As in
previous investigations (Deldin et al., in press; Skesyuk et al., 2005; Joormann &
Gotlib, 2008) list valance ratings differed significantly within lists, with positive
stimuli having significantly greater Happiness ratings than both neutral, and negative
words, and negative words having significantly greater Sadness ratings than both
positive and neutral words (all $p < .01$). Further, the number of syllables, and word
length did not differ between positive, negative, and neutral words within both lists.
No between list differences existed for stimuli valence ratings, word length, or number
of syllables.

**Self-Report Questionnaires**

**Online Screening Questionnaire.** An online screening questionnaire was used
which asked participants to provide demographic information (i.e., age, gender, total
years of education, and contact details), and details of any personal and/or family
history of psychopathology and treatment (personal only). They were asked to record
their current substance and medication use, history of neurological disorders or
injuries (e.g., head injuries causing unconsciousness, epilepsy, history of
electroconvulsive therapy, stroke, tumour), and presence of auditory, visual, language,
or reading issues.

**The Depression Anxiety and Stress Scale** (DASS-42; Lovibond & Lovibond,
1995). The DASS-42 is a self-report measure, specifically designed to distinguish
between, and provide measures of, the three related negative emotional states of
depression, anxiety, and stress, as described by the tripartite model of affect (Watsonet
al. 1995). It provides a quantiative (dimensional) measure of the severity of each syndrome. The DASS-42 has been shown to have impressive psychometric properties in large samples drawn from both the general population and clinical samples. For instance, in a large student sample ($N = 717$; Lovibond & Lovibond, 1995) it was found that the BAI (Beck et al., 1988) and the DASS anxiety scale were highly correlated ($r = .81$), as were the BDI and DASS-42 depression scale ($r = .74$), between construct correlations were substantially lower ($r = .54$ for DASS depression and BAI; $r = .58$ for DASS anxiety and BDI). Similar patterns of correlations have been observed in clinical samples (Antony, Beiling, Cox, Enns, & Swinson, 1998). Recently, Crawford and Henry (2003) found Cronbach’s alpha of .897 (95% CI = .890–.904) for the anxiety scale, .947 (95% CI = .943–.951) for the depression scale, .933 (95% CI = .928–.937) for the stress scale, and .966 (95% CI = .964–.968) for the total score.

**Electrophysiology Recording & ERP Identification**

Similar protocols utilised in the main experiments (see Chapter 3, Section 3.2.3.1. and 3.3.3.1.) were employed.

**Procedures**

**Participant Recruitment**

Participants were recruited from a pool of male and female students aged between 17 – 45 years, who completed research participation for introduction psychology course credit ($N = 234$). Potential participants had already completed the online screening questionnaire, which assessed current depression, anxiety, and stress levels via the DASS-42, and asked them to provide demographic information (i.e., age, total years of education, and contact details), and details of any personal and/or
family history of psychopathology and treatment (personal only). Participants were placed into one of two participants groups according to their DASS-42 scores. The time interval between initial assessment and the electrophysiological testing ranged between one to three weeks, with a mean time lapse of 12.25 days ($SD = 3.34$ days).

**Emotional Oddball Task**

To assess ERPs for attention biases, participants completed an emotional oddball task, which required them to view a Bernoulli presentation of six positive, six negative, and 28 neutral words. The positive and negative words acted as the rare occurring oddballs (15% each), while the neutral words acted as the frequent stimuli (70%). Each word was presented five times to ensure participants became familiar with the Target list for the subsequent tasks and in an attempt to replicate ruminative processes. This allowed for 30 trials of the positive and negative stimuli, and 140 trials of the neutral stimuli (thus, 200 trials in total). Prior to each word, a small white dot appeared on the computer monitor for 1500 ms, which would cue participants that a new stimulus trial was about to begin. A word was then presented for 300 ms, after which the monitor went black for 2200 ms (see Figure A1). As soon as the word was presented, participants were required to indicate as fast as they could whether or not they thought the adjective was self-descriptive by pressing the ‘Yes’ or ‘No’ key. The white cue screen then reappeared, beginning a new stimulus trial. Thus, an inter-stimulus interval (ISI) of 4000 ms was employed, of which 2500 ms was used to record the event-related potentials (ERPs; Figure A1). Dependent variables were frequencies of self-attributed positive or negative adjectives, reaction times, and P2, P3a, P3b, and LPP ERPs.
Figure A1. Schematic depiction of the emotional oddball task.

**Overview of pilot Study 1 Results.**

In summary, the behavioural data found:

- Control participants endorsed more positive words as self-descriptive than the dysphoric participants (Pη² = .152, power = .719).
- Dysphoric participants endorsed more negative words as self-descriptive than the control participants (Pη² = .107, power = .548).
- Dysphoric participants had significantly faster RTs than Control participants when endorsing negative stimuli as self-descriptive (Pη² = .302, power = .855).
The electrophysiological data found:

- A PCA analysis of the pilot EEG data for study 1 indicated that six components should be retained and these accounted for 78.51% of the variance for the Promax rotation. Specifically, it found that the latency of components 5 (4.31% explained variance), 6 (3.86% explained variance), and the combined window encompassing components 3 (9.83% explained variance) and 4 (6.37% explained variance) appeared to be consistent with a priori epochs and grand average peaks for the P2, P3a, and P3b, respectively (Shestyuk & Deldin, 2010; Polich, 2003, 2007). These results indicated that the 150-250 ms temporal window was deemed a suitable epoch to measure the P2, 251-400 ms to measure the P3a, and 401-800 ms to measure the P3b.

- As predicted, no group-by-valence effects were found for the P2 ERP data ($p = .937$, $\eta^2 = .002$, power = .059).

- As predicted, no group-by-valence effects were found for the P3a ERP data ($p = .708$, $\eta^2 = .008$, power = .095).

- Unexpectedly, no group-by-valence-by-laterality effects were found for the P3b ERP data ($p = .216$, $\eta^2 = .038$, power = .381).

**Discussion for Study 1 Pilot Findings**

The results of the pilot study was able to show support for Bower’s (1980) model that dysphoric mood negatively skew data processing towards mood-congruent material. The results showed that 20 participants per group allowed sufficient power to show these robust effects.

The P3b data did not show statistically divergent results for the impact of valence or group. It might be that the less emotional internal set evident in the dysphoric sample may explain the finding of statistically similar P3b for positive and
negative stimuli in this group. It is possible that ERPs amplitudes are only sensitive to more extreme differences in incongruence between cognitive self-schemas and environmental expectations (as would be expected in clinically depressed individuals).

Alternatively, the null effects may be the result of design issues. First, the small sample resulted in a relatively low powered analysis (= .381), suggesting a greater number of participants may be required in the main analysis. Alternatively, the high repetition of stimuli may have led to habituation effects, thereby reducing the magnitude of the P3b and identification of any attention bias effects. Specifically, stimulus familiarly may have increased participant’s expectation for this information and thus, reduced the need for context updating, flattening the P3b effect (Polich & Kock, 1995; Ranganatha & Rainer, 2003). Last, the small pool of negative and positive word stimuli used in the pilot study could have resulted in reduced chances for participant self-identification with the stimuli. This is a key limitation, as attention and memory biases in depression tend to be more reliably observed under conditions of self-descriptive encoding (see section 2.3). That is, depressed individuals specifically experience an inability to disengage from personally salient negative information, rather than to negative information in general (Gotlib & Joormann, 2008; Joormann et al., 2007; Wisco, 2009). For these reasonss a larger pool of stimuli should be selected for Study 1, with each stimulus only presented once during the self-descriptive encoding task.
Appendix C

Study 2 Pilot

The aim of this pilot study was to investigate electrophysiological evidence of mood-congruent memory biases in dysphoria as evidenced by behavioural recognition and recall rates and aberrant FN400 and LPC Old/New effects. The pilot study was further used to help determine experimental power and inform the selection of experimental stimulus characteristics, presentation rate, and repetition for Study 2 of the dissertation.

Method

Participants

Participants of the pilot for study 2 consisted of the same sample of 40 university students who were recruited for study 1 pilot (Appendix A). As in the pilot for study 1, participants were placed into one of two groups according to their DASS-42 and BDI-SF results:

1. Dysphoric Group (n = 20).
2. Healthy control/never depressed group (control; n = 20).

Participants characteristics are outlined in the Results section for the pilot of Study 1 (see Appendix B).

Materials

Material for the pilot of Study 2 are outlined above in the Materials section for the pilot of Study 1 (Appendix B).

Procedures

The same EEG recording and data reduction procedures utilised in pilot of study 1 and outlined in its Method section was employed (see Appendix B).
Cognitive Task: Emotional Old/New Task

Each participant was presented with the previously presented 40 words (Target list) from the emotional oddball task (study 1 pilot task) which was randomly mixed with 40 never-seen others (New list, six positive, six negative, and 28 neutral). Following each word presentation, participants were required to answer the question “Do you recognize these words from the first task?” by pressing either the ‘Yes’ or ‘No’ key (Benedetti et al., 2005). Presentation of word stimuli will be random with the restriction that each will be presented five times, ensuring at least 30 trials for each condition (Friedman et al., 2001). Thus, there will be 400 trials in total. The same stimulus presentation timing as the emotional oddball task (study 1 pilot study) was employed (see Figure A2. It took approximately 30 minutes to complete. Dependent variables are frequency of recognition of positive or negative adjectives, error rates, reaction times, and Old/New ERP effects.
Figure A2. Schematic depiction the Old/New task for the pilot of study 2

Overview of Study 2 Pilot Results

In summary, the behavioural data found:

- As expected, control participants were found to recall more positive words than the dysphoric participants ($p > .001$, $\eta^2 = .292$, power = .967), who in turn recalled more negative words than the control participants ($p = .008$, $\eta^2 = .178$, power = .786).

- Contrary to expectations of hypothesis 1 (and to previous work: see Section 2.3.2.), no differences were found for recognition memory of positive or negative stimuli between the two participant groups ($p = .197$, $\eta^2 = .044$, power = .244).
The electrophysiological data found:

- A PCA analysis of the pilot EEG data for study 2 indicated that six components should be retained and these accounted for 74.32% of the variance for the Promax rotation. Between about 150 and 550 ms the PCA solution identified three components10 with small negative peaks (components 3 [10.11% explained variance], 4 [5.76% explained variance], and 6 [3.27% explained variance]). Inspection of the grand average ERP waveforms elicited for each group for the experimental conditions between this time window suggests that these components might represent variants of the early frontal old/new effect—an ERP often associated with familiarity (Curran, 2000; Nessler et al., 2001; Woodruff et al., 2006). The peak latency of the positive peak in component 3 and 4 appeared to be similar to the grand average ERP waveforms between 400-900 ms post-stimulus at the parietal and central electrode sites. Thus, this component appeared consistent with the LPC old/new effect—an ERP often associated with recollection (Paller & Kutas, 1992; Trott et al., 1999; Wilsing, 2000; Woodruff et al., 2006). Thus, the 150 – 500 ms temporal window was deemed a suitable epoch to the negative peak of the FN400 old/new effect; the 301 – 900 ms window was selected to measure the LPC old/new effect.

- A significant valence-by-group interaction was observed for the N400 data (, p = .013, $\eta^2 = .127$, power = .744). Control participants were found to exhibit significantly larger N400 to negative words to the positive and neutral words ($p < .001$, $\eta^2 = .480$, power > .999). This effect was averaged over novelty

10 Again, this appeared to represent the individual variance in the data set, possibly as a result of differences in clinical status and stimuli novelty and valence.
(old/new) conditions. Unexpectedly, no N400 effects were found for the
dysphoric participants (\(p = .343, \eta^2 = .055, \text{power} = .229\)).

- The LPC data observed a significant Novelty-by-Caudality-by-Valence-by-
  Group interaction (\(p = .002, \eta^2 = .185, \text{power} = .925\)). Analysis of this
  interaction found dysphoric participants to exhibit significantly larger LPC to
  positive old than new stimuli at central electrode sites (\(p = .010, \eta^2 = .298,
  \text{power} = .796\)).

**Discussion for Study 2 Pilot Findings**

The behavioural results of the pilot study generally support the idea of mood-
congruent memory in free recall (Bower, 1981). Specifically, recall data showed
individuals have enhanced free-recall for emotional information that is concordant
with their current mood state.

The observation of no statistically divergent valence recognition memory rates
for the dysphoric group supports previous findings that emotional memory shows a
linear relationship with depressive severity. Recognition memory biases for positive
materials are more typically observed in non-depressed samples (Deldin et al., 2001;
2008), while dysphoric or remitted samples show no memory biases (Jermann et al.,
2008; Ridout et al., 2009), and depressed samples more typically show memory biases
for negative materials (Gilboa-Schnechtman et al., 2002; Ridout et al., 2003).

The failure to replicate positive recognition memory bias for the control
participants questions the internal validity of the study design, specifically the high
repetition rate of the words (each word was shown five times in the study phase and
another five times during the test phase). The high stimulus repetition was selected to
ensure that participants encoded all items into memory to be able to test the cognitive
inhibition (predictions of study 3). However, it is likely that this procedure reduced
opportunity for biased encoding, which in turn improved source memory accuracy in
the dysphoric group (Dulas, Newsome, & Duarte, 2011). Further, the reduced number
of experimental words in the pilot study compared to previous Old/New ERP studies
may have rendered it too simplistic, thereby not putting enough demand on the central
executive (the suspected underpinning of the predicted biased memory processes:
Deldin et al., 2000).

The general Old/New effect components—FN400 and LPC—evoked in the
pilot study were comparable with other studies using similar paradigms (e.g., Deldin
et al., 2001; Dietrich et al., 2000; Rugg et al., 1996). This confirms the internal
validity of the selected paradigm.

The FN400 did not appear modulated by dysphoric status or stimulus valence.
These results failed to replicate the work by Dietrich et al. (2000) who observed an
increased positivity beginning approximately 250 ms post stimulus for old relative to
new items in their control participants but not in their MDD participants. Similar to
the recognition memory data, the absence of the predicted reduced Old/New ERP
effect in the dysphoric group might be due to their low depressive severity (Jermann et
al., 2008; Ridout et al., 2009). Alternatively, the null FN400 results might be due to
the influence of medication in our participant sample, with data showing improvement
of memory functioning with SSRIs (Levkovitz, Caftori, Avital, & Richter-Levin,
2002). Finally, as the dysphoric group was determined based on self-report profiles it
is difficult to determine if these results are confounded by comorbid (unmeasured)
constructs, such as the presence of other mental health conditions.

It is possible that the lack of an association between dysphoric status and the
FN400 effects might be due to high repetition of the word stimuli, thus flattening the
magnitude of the memory bias effects due to habituation (Dulas, Newsome, & Duarte,
Further, the reduced number of experimental words in the pilot study compared to previous Old/New ERP studies may have rendered it too simplistic thereby not putting enough demand on the central executive.
Appendix D

Study 3 Pilot

The aim of the pilot for Study 3 was to validate the development of the modified Sternberg Working Memory Task Go/Nogo task. Specifically, it was interested in ensuring that it evoked the Nogo-N2 and Nogo-P3 components. Stimulus repetition rate, the usefulness of neutral stimuli as a baseline condition, and stimulus presentation timings were also investigated. It also provided a power analysis of the suspected inhibitory control effects for Study 3.

**Method: Study 3 Pilot**

**Participants**

Participants of the pilot for study 3 consisted of the same sample of 40 university students who were recruited for study 1 pilot (Appendix B). As in the pilot for study 1, participants were placed into one of two groups according to their DASS-42 results:

1. Dysphoric Group \( (n = 20) \).
2. Healthy control/never depressed group (control; \( n = 20 \)).

**Materials**

Materials for the pilot of Study 3 are outlined above in the Materials section for the pilot of Study 1 (Appendix B).

**Procedures**

The same EEG recording and data reduction procedures utilised in the pilot of study 1 and outlined in its Method section was employed (see Appendix B).

**Modified Sternberg Working Memory Go/Nogo Task**

The task used the original 18 of the original 40 stimuli presented in the emotional oddball task of Study 1 (Target list: six positive, six negative, and random
selection of six/28 neutral words; see Appendix B). It also included six positive,
negative, and neutral distractor words, which were presented for the first time
(Distractor list). The experiment used a similar task as that employed by Joormann
and Gotlib (2008), modified so that it could be conducted with ERP methods. Like
the Joormann and Gotlib (2008) investigation, participants were presented with a
‘learning display’ of two lists of three words each (each list presented vertically down
the screen). One list was printed in red ink and the other list in blue ink. Participants
had 7.8 seconds to memorise each of the word stimuli (1.3 seconds per word;
Joormann & Gotlib, 2008). The lists then disappeared, and the screen went black for
800 ms. A coloured frame then appeared around the screen. The frame was either
red or blue in colour (randomised). The frame was used to cue participants to which
list to remember and respond to in the following task. If it is blue, the participant was
required to remember and respond by pressing the ‘Yes’ key to the words presented in
the blue list in the learning display. If it is red, they were required to remember and
respond to the words presented in the red list in the learning display. ERP
investigations require a number of stimulus trials (15 – 30) to derive averages with
good signal-to-noise ratios (Friedman et al., 2001). Thus, the number of stimulus
presentations used in Joormann and Gotlib (2008) after the learning display has been
altered from one to nine. This included the Bernoulli presentation of the three Go
stimuli, the three proactive interference Nogo stimuli, and three distractor interference
Nogo stimuli. These nine stimulus presentations used the same presentation timing as
ERP tasks used in the pilot for Study 1 and 2 (outlined above). Like in other ERP
Go/Nogo investigations, participants were required to press the ‘Yes’ key for the cued
words, these will constitute the Go trials. They are required to inhibit their responses
to the Nogo words (i.e., Chiu et al., 2008; Ruchsow et al., in press). To reduce any
cortical activation due to the motor responses, participants completed the first half of
the investigation with one hand and the second with the other. A visual depiction of
a run is provided in Figure A3.

The emotional Go/Nogo task will include 30 runs, giving a total of 2700
trials. This constituted 30 trials per stimulus condition. Each word in the condensed
Target list was randomly presented five times each for the Go and Nogo proactive
interference conditions; each word in the distracter list was randomly presented five
times each for the Nogo distractor interference condition.
Figure A3. Schematic depiction of presentation timings for the pilot task for study 3.
Results for pilot of Study 3

In summary, the behavioural data found:

- No group differences were observed for distractor interference error rates for positive and negative stimuli ($p = .348$, $\eta^2 = .026$, power = .200).

- For the proactive interference error rates, control participants were observed to exhibit greater errors of commission for positive stimuli compared to the dysphoric group ($p = .032$, $\eta^2 = .115$, power = .582). Unexpectedly, no group differences were observed for the negative proactive interference stimuli ($p = .597$, $\eta^2 = .007$, power = .082).

The electrophysiological data found:

- The PCA for the Nogo ERPs indicated that five components should be retained, which accounted for 67.02% of the variance for the Promax rotation. The negative peaks in component 2 (13.13% explained variance), 4 (5.44% explained variance), and the negative peak in component 5 appeared consistent with the Nogo-N2 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010), evident within the 200-500 ms a priori epoch for frontal electrodes for the grand average data. The latency of the second positive peak in component 5 and the sustained positivity (temporal window: 300-800 ms) and of component 1 (33.12% explained variance) appeared consistent with the Nogo-P3 (Beste et al., 2010; Chiu et al., 2008; Smith et al., 2010), which also seemed evident at fronto-central midline sites in the grand average data. Thus, the 200-500 ms epoch was deemed appropriate to measure the Nogo-N2, and 400-800 ms epoch to measure the Nogo-P3.
Distractor interference results.

- At electrode site Fz, dysphoric participants were found to exhibit significantly larger Nogo-N2s than control participants for both negative ($p = .037, \eta^2 = .082, \text{power} = .437$) and positive distractor interference stimuli ($p = .002, \eta^2 = .224, \text{power} = .898$).

- At electrode site Fz, dysphoric participants were found to exhibit significantly smaller Nogo-P3s than control participants for both negative ($p = .009, \eta^2 = .168, \text{power} = .770$) and positive distractor interference stimuli ($p = .058, \eta^2 = .091, \text{power} = .478$).

Proactive interference results.

- A significant Caudality-Valence-Group interaction was observed for the Nogo-N2 data ($p = .045, \eta^2 = .068, \text{power} = .656$). Here it was found that dysphoric participants indexed significantly larger (more negative µV) Nogo-N2s to successfully inhibit neutral proactive interference stimuli compared to control participants ($p = .034, \eta^2 = .113, \text{power} = .573$). No effects involving the positive or negative stimuli were observed.

- A significant Caudality-Valence-Group interaction was observed for the Nogo-P3 data ($p = .045, \eta^2 = .068, \text{power} = .656$). Here it was found that dysphoric participants indexed significantly smaller Nogo-P3s to successfully inhibit neutral proactive interference stimuli compared to control participants ($p = .022, \eta^2 = .131, \text{power} = .645$). No effects involving the positive or negative stimuli were observed.

- As expected, Go-P3 was observed to be maximum at parietal midline sites. Dysphoric participants showed significantly larger Go-P3 amplitudes for neutral compared to negative stimuli ($p > .001, \eta^2 = .502, \text{power} = .985$).
Discussion for pilot of Study 3

The Nogo-N2, Nogo-P3, and Go-P3 components evoked in the pilot study were comparable to that evoked in previous Go/Nogo tasks (e.g., Chiu et al., 2008; Zhang et al., 2007). This confirms the internal validity of the selected paradigm.

Unexpectedly, the behavioural data and ERP did not support evidence of proactive interference deficits in the dysphoric participants. It may be possible that the high repetition of a relatively small number of experimental words in the pilot study compared to previous working memory tasks (cf. Joormann & Gotlib, 2008) rendered it too simplistic thereby not putting enough demand on the central executive. It might also be that these effects are only seen in clinical samples (e.g., Joormann & Gotlib, 2008), rather than in transient negative mood states (Chiu et al., 2008). Further the high repetition of the proactive interference stimuli throughout the experiment may have reduced the magnitude of the ERPs due to habituation (Polich & Kok, 2007). Thus, a larger pool of stimuli should be used with less need for stimulus repetition.

The dysphoric participants showed greater deficits in the inhibition of both negative and positive distractor interference stimuli compared to the control group. This was evidenced by their greater frontal Nogo-N2 and smaller Nogo-P3 ERPs. A sample of 20 participants per group provided adequate experimental power to expose these medium to large effects.

Dysphoric participants were observed to exhibit significantly larger Nogo-N2 and diminished Nogo-P3s to successful inhibition of neutral proactive interference stimuli. This ERP effect might reflect less attentional engagement and thus, less access into working memory (a prerequisite of the proactive interference stimuli) by these less arousing words. The impact of stimulus arousal was also in Chiu et al.
(2008)’s Go/Nogo study, which observed both positive and negative Go words to elicit enhanced P3 responses compared to neutral words, possibly reflecting greater attentional engagement by these more arousing stimuli. It was thus determined that use of neutral stimuli would confound the experimental results, as it is difficult to definitively draw conclusions regarding cognitive inhibition processes if the ERP effects could similarly be interpreted as an index of arousal differences’ between stimuli. The Hillyard Principle suggests that ERP researchers should apply the same physical stimuli conditions to avoid confounds (Luck, 2005). For these reasons, in the dissertation studies, only the positive and negative stimuli should be used and these should be matched on arousal dimensions so to reduce any confusion in interpretation of ERP data.
Appendix E
Mood and Fatigue Ratings for the Pilot Studies

Subjective Mood Ratings

A 2 (Group: control, dysphoric) x 3 (Task: attention, memory, inhibition) factorial ANOVA was conducted to examine if participants’ subjective ratings of mood (9-point scale, 1 = depressed, 9 = positive) remained stable over the three ERP tasks for the pilot investigation. Mauchly’s test of sphericity was not significant. No interaction was found between the variables ($F(2, 76) = 0.125, p = .882, \eta^2 = .003$, power = .069; see Figure A4).

![Figure A4. Participant subjective mood ratings across the three electrophysiological pilot tasks. Error bars refer to standard error of the mean.](image-url)
The main effects of Task was significant ($F (2, 76) = 3.947, p = .023, \eta^2 = .094$, power = .693). Post-hoc within-subjects contrasts (simple difference method) found no statistically significant differences between tasks using the bonferroni corrected alpha level of .001 (.05/3 tests). However, a trend was found for participants’ subjective mood ratings to reduce from the first to the third task ($F (1, 38) = 5.59, p = .023, \eta^2 = .128$, power = .635). This trend was not apparent between the first and second task ($F (1, 38) = 3.88, p = .056, \eta^2 = .093$, power = .484), or between the second and third task ($F (1, 38) = 1.17, p = .286, \eta^2 = .030$, power = .184). The analysis also found a significant main effect of Group ($F (1, 38) = 7.36, p = .010, \eta^2 = .162$, power = .753), with the Dysphoric group reporting more depressed ratings averaged across the three tasks.

**Subjective Fatigue Ratings**

A 2 (group) x 3 (task) factorial ANOVA was conducted to examine if participants’ subjective ratings of fatigue (9-point scale, 1 = exhausted, 9 = energetic) remained stable over the three ERP tasks. Mauchly’s test of sphericity was not significant. Similar to the subjective mood ratings, no interaction was found between the variables ($F (2, 76) = 0.064, p = .938, \eta^2 = .002$, power = .059; see Figure 2).

The main effect of Task was significant ($F (2, 76) = 19.90, p <.001, \eta^2 = .344$, power > .999). Post-hoc within-subjects contrasts (simple difference method) using the bonferroni corrected alpha level of .001 (.05/3 tests) found participants reported a significant increase in fatigue ratings from the first to third task ($F (1, 38) = 3046, p < .001, \eta^2 = .445$, power > .999), and from the second to third task ($F (1, 38) = 15.66, p < .001, \eta^2 = .292$, power = .971). No significant changes in fatigue ratings were observed from the first and second task ($F (1, 38) = 6.87$, power = .724).
There was no main effect of Group on fatigue ratings ($F(1, 38) = 0.20, p = .654$, $\eta^2 = .005$, power = .072). These results are shown in Figure A5.

Figure A5. Participant subjective fatigue ratings across the three electrophysiological pilot tasks. Error bars refer to standard error of the mean.
## Appendix F

### Previous Remitted Depression Studies

**Table A2**

*Review of Previous Study’s’ Remitted-Depression Sample Characteristics (standard deviations are in parentheses)*

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Number of past MDE</th>
<th>Age</th>
<th>Gender</th>
<th>BDI-II and clinical range</th>
<th>Medication Status</th>
<th>ERP or Behavioural Investigation</th>
<th>Analysis Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current</td>
<td>13</td>
<td>1.69 (0.95)</td>
<td>20.64 (1.05)</td>
<td>13/0</td>
<td>10.54 (8.05) Normal range</td>
<td>Medication free</td>
<td>ERP Mixed</td>
<td>ANOVA</td>
</tr>
<tr>
<td>Merens et al., 2008 Study 1</td>
<td>19</td>
<td>4.9 (4.1)</td>
<td>44.2 (13.0)</td>
<td>17/2</td>
<td>11.70 (8.0) Normal range</td>
<td>All medicated with SSRI or SSNRI</td>
<td>Behavioural Mixed</td>
<td>ANOVA</td>
</tr>
<tr>
<td>Merens et al., 2008 Study 2</td>
<td>20</td>
<td>4.8 (4.4)</td>
<td>48.7 (7.9)</td>
<td>9/11</td>
<td>12.9 (10.1) Normal range</td>
<td>All medicated with SSRI or SSNRI</td>
<td>Behavioural Mixed</td>
<td>ANOVA</td>
</tr>
<tr>
<td>Vanderhasselt et al., 2012</td>
<td>15</td>
<td>4.13 (4.14)</td>
<td>27.87 (7.91)</td>
<td>8/7</td>
<td>4.75 (4.36) Normal range</td>
<td>Nil medicated at time of testing</td>
<td>ERP Mixed</td>
<td>ANOVA</td>
</tr>
<tr>
<td>Fritzsche et al., 2010</td>
<td>20</td>
<td>Not reported</td>
<td>39.95 (11.62)</td>
<td>10/10</td>
<td>8.75 (6.72) Normal range</td>
<td>6 participants were medicated with SSRI</td>
<td>Behavioural Mixed</td>
<td>ANOVA</td>
</tr>
<tr>
<td>Kerestes et al., 2012</td>
<td>19</td>
<td>4.42 (6.45)</td>
<td>33.6 (13.6)</td>
<td>15/4</td>
<td>Normal range on the HAMD</td>
<td>Medication free</td>
<td>fMRI MANOVA</td>
<td></td>
</tr>
</tbody>
</table>

*Note. MDE = Major depressive episode.*
Comorbid Anxiety Diagnoses for MDD Group

Table A3

*Major Depressive Disorder Participants Comorbid Anxiety Diagnoses based on the SCID-I/NP Diagnoses*

<table>
<thead>
<tr>
<th>Comorbid Anxiety Diagnosis</th>
<th>Count (percentage of MDD sample)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agoraphobia</td>
<td>4 (13.33% )</td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>2 (6.67% )</td>
</tr>
<tr>
<td>Post-Traumatic Stress Disorder (PTSD)</td>
<td>6 (20% )</td>
</tr>
<tr>
<td>Generalised Anxiety Disorder (GAD)</td>
<td>2 (6.67% )</td>
</tr>
<tr>
<td>Simple Phobia</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>Social Anxiety Disorder</td>
<td>7 (23% )</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder (OCD)</td>
<td>0</td>
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</tbody>
</table>

Thirteen, or 43.33%, of the study’s 30 participants in the MDD met criteria for a secondary anxiety diagnosis. This is consistent with comorbidity estimates in the general population between depression and anxiety (Kesslet, 2003; Sartorius et al., 1996), suggesting good ecological validity of the sample.

\[11\] : 1 animal type, 2 blood-injury type
Information Sheet
This project is a partial requirement for the assistant investigator Rachel Dati’s degree of Doctor of Philosophy (Clinical Psychology), in the School of Applied Psychology, Faculty of Health Sciences at Griffith University. It is a research project and is not a part of the curriculum or normal school activity.

The aim of this study is to use electrical recordings of brain activity to investigate the neural processes underpinning various memory and cognitive studies amongst individuals in different mood states.

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Overview of Study
We are interested in how mood affects cognitive processing of different types of information, specifically attention, memory and inhibitory abilities. This investigation can be broken into three phases: Phase 1 involved the administration of the Structured Clinical Interview for DSM-IV-TR (SCID-I). Phase 2 involved the use of an electroencephalograph (EEG) to record your brain activity during completion of three cognitive studies involving attention and memory processes. Phase 3 involved the online completion of two follow-up questionnaires.

**Phase 1:** The SCID-I were used to further assess the nature of your current mood and behaviours in further detail. This structured clinical interview were audio recorded to assess inter-rater reliability and other statistical issues. The interview will take approximately 1 hour and were conducted at Griffith University, Mount Gravatt. Either a fully registered or a provisionally registered psychologist who are completing their doctoral clinical training in psychology at Griffith University will conduct the interview.

Participation in this study involved minimal risk, this study may increase your level of awareness of the behaviours, thoughts and actions you rely on to manage your mood, and how you cope emotionally with stress. Some individuals might find this increased awareness distressing. Some assessment questions may elicit negative feelings about you, the assessor, or the intake session. If your experience any uncomfortable feelings or thoughts, we invite you to speak with your assessor. Alternatively, Rachel Dati (a registered psychologist) was available for a consultations and referral. In addition, the Griffith University Counselling Centre is a free campus resource that provides support for emotional or physical distress.

We are always concerned about issues of safety. If you disclose your intention to harm yourself or others, we are obligated to help you and those around you stay safe by alerting professionals who can help.

**Phase 2:** Within one to two weeks after completion of the SCID-I interview, you were asked to complete the EEG testing phase of the experiment at the Griffith University Cognitive Psychophysiology Laboratory, located on the Mt Gravatt campus. *Please do not participate in today’s interview if you feel that you were unable to complete the EEG testing phase of the study.*

During EEG testing, electrical activity is conducted from the scalp to electrodes in an Electrode Cap. Signals are then amplified, digitised, and saved to a computer hard drive. After completion of these studies, you were removed from the EEG and required to complete five short questionnaires regarding your mood and thoughts over the past few weeks. The experiment will take no longer than 2 hours.

**Phase 3:** Thirty days following the EEG experiment, the researchers will recontact you and ask you to complete two more questionnaires. This should take you approximately 20 minutes. *Please do not participate in today’s testing if you feel that you were unable to complete these follow-up questionnaires.*

Your estimated participation time were 1 hour for the SCID interview (worth 1 credit), 2 hours for the EEG experiment (worth 2 credits), and .5 hours for the 1-month
follow-up online questionnaires (worth .5 credits). Together, you were awarded with 3.5 credit points for your participation in all stages of the experiment.

Benefits of the Investigation, Your Rights, & Contact Information

The expected benefits of this investigation will allow us to gain a deeper understanding of how people cognitively process information in different affective states. It may be used to help further our knowledge of appropriate therapeutic and treatment interventions for people in these emotional states. The direct benefits to you in participating in this stage of the investigation are receiving experimental credits, and learning about psychological and physiological research. The indirect benefits are being part of research that could advance knowledge about important psychological issues.

Participation in the investigation is completely voluntary. You retain the right at all times, to discontinue without penalty, or without providing an explanation. If you decide you discontinue participation, and are a student of Griffith University, it can be assured that this decision will not influence your relationship with the University. If you withdraw from the study, you will receive an amount of extra course credit that is consistent with the proportion of time you have spent in the overall study. For instance, you were awarded 1.5 credit points if you decide to withdraw from the experiment after the initial interview stage and with 4 credit points if you decide to withdraw from the experiment after the experimental Study stage.

Storage of Collected Information

All information collected from you during the investigation was coded with a de-identified number to ensure your anonymity. The data were kept in this form in a locked research area for seven years, after which any paper copies were shredded, and electronically destroyed. For further information in regards to these issues you can consult the University’s Privacy Plan at www.gu.edu.au/ua/aa/vc/pp or telephone (07) 3735 5585.

At the conclusion of this research, feedback regarding the research findings was posted on the Noticeboard outside Room 2.10, M24, Griffith University, Mount Gravatt campus.

Ethical Clearance

Griffith University conducts research in accordance with the National Statement on Ethical Conduct for Research Involving Humans. This investigation has been reviewed and given ethical clearance by the Griffith University Ethics Committee (Ethics protocol number: PSY/66/10/HREC). If you do have any complaints concerning the manner in which the research is conducted, please forward them to myself or, if an independent person is preferred, to the Manager, Research Ethics on (07) 3735 5585 or research-ethics@griffith.edu.au.
Appendix H

Consent Form

Mood Effects on Attention & Memory: 
A Brainwave Study

CONSENT FORM

The aim of this study is to use electrical recordings of brain activity to investigate 
the neural processes underpinning various memory and cognitive studies amongst 
individuals in different mood states.

By signing below, I agree to participate in the above named project and in so doing acknowledge that:

1. I have read the Information Sheet outlining the nature and purpose of the project and the extent 
of my involvement, and have had these details explained to me. I have had the opportunity to 
ask further questions and am satisfied that I understand.

2. I am aware that, although the project is directed to the expansion of knowledge generally, it may 
not result in any direct benefit to me.

3. I realize that whether or not I decide to participate is my decision and will not affect my 
studies.

4. I realize that I can withdraw from the study at any time without having to give any reason and 
without any loss of experimental credit.

5. I understand that all information collected throughout this study was coded with a de- 
identified number to ensure my anonymity.

6. I am aware that I may request further information about the project as it proceeds. Further, I 
understand that if I have additional questions after leaving the experiment I can contact the 
research team.

7. I understand that a de-identified copy of this data may be used in future analysis which may be 
be beyond the scope of this particular experiment. However, my anonymity was safeguarded at 
all times. For further information in regards to these issues, I understand that I can consult the 
University’s Privacy Plan at www.gu.edu.au/ua/aa/vc/pp or telephone (07) 3735 5585.

8. I have been advised that the project has been reviewed and given ethical clearance to proceed 
by the Griffith University Human Research Ethics Committee based on the National Statement 
on Ethical Conduct in Human Research (2007). I understand that I can contact the Manager of 
this committee on 3735 5585 (or research-ethics@griffith.edu.au) if I have any concerns of 
ethical conduct of the research.

9. I agree to participate in the project.

.................................................................  ............/................./..................
Name : __________________________  Date
Participant

.................................................................  ......../.........../.................
Name : __________________________  Date
Researcher
## Appendix I

### Summary of Psychometrics for Selected Questionnaires

<table>
<thead>
<tr>
<th>Measure</th>
<th>Measure Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depression Anxiety Stress Scale-21 item version</strong> (DASS-21, Lovibond &amp; Lovibond, 1995)</td>
<td>The DASS is a self-report questionnaire that was specifically designed to distinguish between, and provide relatively pure measures of, the three related and clinically significant negative emotional states of depression, anxiety, and stress, as described by the tripartite model of affect (Watson et al. 1995). It provides a quantitative (dimensional) measure of the severity of each syndrome.</td>
</tr>
<tr>
<td><strong>Beck Depression Inventory-Second Edition</strong> (Silverman &amp; Albano, 1996) BDI-II, Beck, Steer &amp; Brown, 1996)</td>
<td>A 21-item, pen and paper self-report measure to assess the presence and severity of depression in individuals aged 13 years and older. Answers are recorded on a 4-point scale (0 to 4) designed to ascertain levels of severity, with each item representing a symptom characteristic of depression as prescribed by the DSM-IV, such as sadness, guilt, suicidal thoughts and loss of interest. The summed score for the BDI (range 0 to 66) is compared with pre-established degrees of depression: minimal (1-13), mild (14-19), moderate (20-28) and severe (29-63) (Beck, Steer &amp; Brown, 1996). Its length is acceptable: it takes approximately 5-10 minutes to complete.</td>
</tr>
<tr>
<td><strong>Emotion Regulation Questionnaire</strong> (ERQ; Gross &amp; John, 2003)</td>
<td>The ERQ consists of two subscales that measure habitual use of reappraisal or suppression using a 7-point scale. The reappraisal subscale (I control my emotions by changing the way I think about the situation I’m in) consists of 6 items and the suppression subscale (I keep my emotions to myself) consists of 4 items.</td>
</tr>
<tr>
<td><strong>Ruminative Responses Scale</strong> (RRS; Treynor et al., 2003)</td>
<td>The Ruminative Responses Scale (RRS; Treynor et al., 2003) to assess how participants tend to respond to sad feelings and symptoms of dysphoria. The RRS assesses responses to dysphoric mood that are focused on the self (think about all your shortcomings, failings, faults, mistakes), on symptoms (think about how hard it is to concentrate), or on possible consequences and causes of moods (analyse recent events to try to understand why you are depressed) using a 4-point scale (almost never to almost always).</td>
</tr>
<tr>
<td><strong>State Trait Anxiety Inventory –Form Y</strong> (STAI-Form Y; Spielberger, Gorsuch, Lushene, Vagg, &amp; Jacobs, 1983).</td>
<td>The STAI is an instrument that quantifies adult anxiety. This particular instrument is used to simplify the separation between state anxiety and trait anxiety, feelings of anxiety and depression. The STAI includes a 40 question response taking approximately 10-20 minutes for completion and the test is given in tens of different languages worldwide. This test is split into the S-Anxiety scale and the T-Anxiety scale, each having 20 items. These tests are answered on the basis of a 1-4 scale, with the focused areas including: worry, tension, apprehension, and nervousness. The current edition is Form Y.</td>
</tr>
</tbody>
</table>
**Appendix J**

*Experimental Word Stimuli*

**List A**

<table>
<thead>
<tr>
<th>Negative Adjectives</th>
<th>Positive Adjectives</th>
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<td>attacking</td>
<td>glorious</td>
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<tr>
<td>dismal</td>
<td>original</td>
</tr>
<tr>
<td>inattentive</td>
<td>sensible</td>
</tr>
<tr>
<td>terrible</td>
<td>spirited</td>
</tr>
<tr>
<td>scolding</td>
<td>progressive</td>
</tr>
<tr>
<td>arrogant</td>
<td>glowing</td>
</tr>
<tr>
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<td>intellectual</td>
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<td>admired</td>
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<td>dependable</td>
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<td>tender</td>
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<td>experienced</td>
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<td>stupid</td>
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<td>List B</td>
<td>Positive Adjectives</td>
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### List C

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<td>malicious</td>
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Appendix K

Participant ERP Task Instructions: Study 1

Please sit as still as possible and attend to the words, which will be presented individual on the computer monitor. Prior to each word you will see a small white dot. Look at this directly and get ready for a word to be shown. A word will then appear on the screen briefly, it will be replaced with a black screen for about two seconds.

As soon as the word is shown you are required to indicated whether, in general, that word may be associated with/or used to describe yourself. You will make this response by pressing the “YES” or “NO” button on the computer keyboard. Please make this decision as quickly as possible (you will only have until the end of the black screen to complete this response). Your responses here will be completely confidential; the investigators will not be able to see what they are during recording of your brain waves.

During this test, please keep relaxed and still as possible. Try you best to minimise eye blinks, movements (except answer responses) and speaking during recording of your brain waves. If you need to blink, please do so during the screen with the white dot.
Appendix L

Remitted Participant’s Normality Data

Table A4

Skewness and Kurtosis Statistics for Remitted-Depressed Participant’s Data

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<th>Variable</th>
<th>Skewness</th>
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<td><strong>Study 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% positive adjectives rated as self-descriptive\textsuperscript{12}</td>
<td>-3.78</td>
<td>5.83</td>
</tr>
<tr>
<td>% negative adjectives rated as self-descriptive</td>
<td>2.56</td>
<td>1.72</td>
</tr>
<tr>
<td>RT for positive adjectives rated as self-descriptive</td>
<td>0.35</td>
<td>-0.83</td>
</tr>
<tr>
<td>RT for negative adjectives rated as self-descriptive</td>
<td>0.64</td>
<td>-0.58</td>
</tr>
<tr>
<td>RT for positive adjectives rated as not self-descriptive</td>
<td>-0.58</td>
<td>-0.64</td>
</tr>
<tr>
<td>RT for negative adjectives rated as not self-descriptive</td>
<td>1.27</td>
<td>0.32</td>
</tr>
<tr>
<td>Frontal P2 for positive words</td>
<td>0.68</td>
<td>-0.05</td>
</tr>
<tr>
<td>Central P2 for positive words</td>
<td>-0.045</td>
<td>-1.33</td>
</tr>
<tr>
<td>Parietal P2 for positive words</td>
<td>0.15</td>
<td>-0.55</td>
</tr>
<tr>
<td>Frontal P2 for negative words</td>
<td>0.79</td>
<td>-0.88</td>
</tr>
<tr>
<td>Central P2 for negative words</td>
<td>0.41</td>
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</tr>
<tr>
<td>Parietal P2 for negative words</td>
<td>-0.63</td>
<td>-0.86</td>
</tr>
<tr>
<td>Left P3b for positive words</td>
<td>-0.13</td>
<td>0.36</td>
</tr>
<tr>
<td>Midline P3b for positive words</td>
<td>0.61</td>
<td>-0.63</td>
</tr>
<tr>
<td>Right P3b for positive words</td>
<td>0.24</td>
<td>-1.01</td>
</tr>
<tr>
<td>Left P3b for negative words</td>
<td>1.03</td>
<td>-0.07</td>
</tr>
<tr>
<td>Midline P3b for negative words</td>
<td>0.47</td>
<td>-0.69</td>
</tr>
<tr>
<td>Right P3b for negative words</td>
<td>0.49</td>
<td>-0.02</td>
</tr>
<tr>
<td>Frontal LPP for positive words</td>
<td>1.66</td>
<td>0.45</td>
</tr>
<tr>
<td>Central LPP for positive words</td>
<td>1.65</td>
<td>0.00</td>
</tr>
<tr>
<td>Parietal LPP for positive words</td>
<td>0.73</td>
<td>0.74</td>
</tr>
<tr>
<td>Frontal LPP for negative words</td>
<td>1.61</td>
<td>-0.02</td>
</tr>
</tbody>
</table>

\textsuperscript{12} Observed skewness and kurtosis of data is due to an identified outlier. However, this participant was deemed part of the sample and thus retained in analysis. Results here need to be interpreted with this in mind.
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Central LPP for negative words</td>
<td>1.58</td>
<td>-0.03</td>
</tr>
<tr>
<td>Parietal LPP for negative words</td>
<td>3.23</td>
<td>4.08</td>
</tr>
</tbody>
</table>

**Study 2**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>% free recall for positive words</td>
<td>1.87</td>
<td>0.47</td>
</tr>
<tr>
<td>% free recall for negative words</td>
<td>2.05</td>
<td>0.75</td>
</tr>
<tr>
<td>% recognition for positive words</td>
<td>-0.38</td>
<td>-0.48</td>
</tr>
<tr>
<td>% recognition for negative words</td>
<td>-0.58</td>
<td>-0.64</td>
</tr>
<tr>
<td>Frontal N400s for old positive words</td>
<td>0.73</td>
<td>0.03</td>
</tr>
<tr>
<td>Central N400s for old positive words</td>
<td>0.08</td>
<td>4.36</td>
</tr>
<tr>
<td>Parietal N400s for old positive words</td>
<td>0.43</td>
<td>-0.49</td>
</tr>
<tr>
<td>Frontal N400s for old negative words</td>
<td>-0.15</td>
<td>-1.24</td>
</tr>
<tr>
<td>Central N400s for old negative words</td>
<td>-0.11</td>
<td>0.01</td>
</tr>
<tr>
<td>Parietal N400s for old negative words</td>
<td>-0.04</td>
<td>-1.24</td>
</tr>
<tr>
<td>Frontal N400s for new positive words</td>
<td>1.14</td>
<td>-0.47</td>
</tr>
<tr>
<td>Central N400s for new positive words</td>
<td>1.04</td>
<td>0.88</td>
</tr>
<tr>
<td>Parietal N400s for new positive words</td>
<td>-0.55</td>
<td>-0.71</td>
</tr>
<tr>
<td>Frontal N400s for new negative words</td>
<td>1.20</td>
<td>1.48</td>
</tr>
<tr>
<td>Central N400s for new negative words</td>
<td>-0.65</td>
<td>-0.74</td>
</tr>
<tr>
<td>Parietal N400s for new negative words</td>
<td>0.59</td>
<td>-0.89</td>
</tr>
<tr>
<td>Frontal LPCs for old positive words</td>
<td>-0.57</td>
<td>0.28</td>
</tr>
<tr>
<td>Central LPCs for old positive words</td>
<td>0.73</td>
<td>0.12</td>
</tr>
<tr>
<td>Parietal LPCs for old positive words</td>
<td>0.70</td>
<td>0.78</td>
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<td>Frontal LPCs for old negative words</td>
<td>0.70</td>
<td>0.44</td>
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<tr>
<td>Central LPCs for old negative words</td>
<td>2.98</td>
<td>4.32</td>
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<tr>
<td>Parietal LPCs for old negative words</td>
<td>1.79</td>
<td>1.94</td>
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<tr>
<td>Frontal LPCs for new positive words</td>
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<td>-0.06</td>
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<td>Central LPCs for new positive words</td>
<td>0.12</td>
<td>0.38</td>
</tr>
<tr>
<td>Parietal LPCs for new positive words</td>
<td>1.00</td>
<td>0.35</td>
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<tr>
<td>Frontal LPCs for new negative words</td>
<td>1.22</td>
<td>0.82</td>
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<td>Central LPCs for new negative words</td>
<td>2.87</td>
<td>3.72</td>
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<td>Parietal LPCs for new negative words</td>
<td>1.76</td>
<td>1.47</td>
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<td>LPNs for old positive words</td>
<td>-0.911</td>
<td>0.685</td>
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<tr>
<td>LPNs for old negative words</td>
<td>-0.126</td>
<td>-0.176</td>
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<tr>
<td></td>
<td>Study 3</td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Distractor Interference Stimuli (Hypothesis 1-4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>% errors for positive words</td>
<td>1.82</td>
</tr>
<tr>
<td></td>
<td>% errors for negative words</td>
<td>3.08</td>
</tr>
<tr>
<td>Left Nogo-N2 for positive words</td>
<td>-0.27</td>
<td>1.16</td>
</tr>
<tr>
<td>Midline Nogo-N2 for positive words</td>
<td>-1.05</td>
<td>0.97</td>
</tr>
<tr>
<td>Right Nogo-N2 for positive words</td>
<td>-0.86</td>
<td>-0.21</td>
</tr>
<tr>
<td>Left Nogo-N2 for negative words</td>
<td>-0.22</td>
<td>-0.07</td>
</tr>
<tr>
<td>Midline Nogo-N2 for negative words</td>
<td>0.42</td>
<td>-0.56</td>
</tr>
<tr>
<td>Right Nogo-N2 for negative words</td>
<td>0.02</td>
<td>0.76</td>
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<tr>
<td>Left Nogo-P3 for positive words</td>
<td>0.59</td>
<td>-0.19</td>
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<tr>
<td>Midline Nogo-P3 for positive words</td>
<td>0.07</td>
<td>-0.88</td>
</tr>
<tr>
<td>Right Nogo-P3 for positive words</td>
<td>-0.29</td>
<td>-1.35</td>
</tr>
<tr>
<td>Left Nogo-P3 for negative words</td>
<td>0.34</td>
<td>0.08</td>
</tr>
<tr>
<td>Midline Nogo-P3 for negative words</td>
<td>-0.46</td>
<td>-0.85</td>
</tr>
<tr>
<td>Right Nogo-P3 for negative words</td>
<td>0.53</td>
<td>0.57</td>
</tr>
<tr>
<td>Nogo-LPP for positive words</td>
<td>-0.18</td>
<td>-0.68</td>
</tr>
<tr>
<td>Nogo-LPP for negative words</td>
<td>0.29</td>
<td>-0.93</td>
</tr>
<tr>
<td></td>
<td>Proactive Interference Stimuli (Hypothesis 5-9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>% errors for positive words</td>
<td>0.91</td>
</tr>
<tr>
<td></td>
<td>% errors for negative words</td>
<td>1.06</td>
</tr>
<tr>
<td>Nogo-N2 for positive words</td>
<td>-0.61</td>
<td>1.01</td>
</tr>
<tr>
<td>Nogo-N2 for negative words</td>
<td>0.04</td>
<td>0.12</td>
</tr>
<tr>
<td>Nogo-P3 for positive words</td>
<td>0.15</td>
<td>-0.93</td>
</tr>
<tr>
<td>Nogo-P3 for negative words</td>
<td>-0.32</td>
<td>-0.69</td>
</tr>
<tr>
<td>Nogo-LPP for positive words</td>
<td>1.55</td>
<td>0.50</td>
</tr>
<tr>
<td>Nogo-LPP for negative words</td>
<td>1.13</td>
<td>0.44</td>
</tr>
</tbody>
</table>
Appendix M

Scree plot for control participants for Study 1
Appendix N

Scree plot for remitted participants for Study 1
Appendix O

Scree plot for MDD participants for Study 1
Appendix P

Study 1 Supplementary Results

Results for the null predictions for P3a data. To confirm the null predictions for the P3a data, peak amplitudes were subjected to a 3 (group) × 3 (caudality) × 3 (laterality) × 2 (valence) factorial ANOVA (Table A5). No significant results were observed. No correlations were found between participants’ P3a scores and their self-reported results on the psychometric assessments of current or one-month depression, and anxiety, or use of rumination, suppression, or emotional regulation.

Table A5

P3a Factorial ANOVA Results: Study 1

<table>
<thead>
<tr>
<th>Variable(s)</th>
<th>ANOVA Results</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>$F(2, 70) = 0.44, p = .647, \eta^2 = .012$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Group</td>
<td>$F(2.50, 87.64) = 0.20, p = .865, \eta^2 = .006$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Group</td>
<td>$F(3.72, 130.22) = 0.33, p = .847, \eta^2 = .009$</td>
<td>ns</td>
</tr>
<tr>
<td>Valence × Group</td>
<td>$F(2, 70) = 2.06, p = .135, \eta^2 = .056$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Laterality × Group</td>
<td>$F(6.53, 228.38) = 0.87, p = .524, \eta^2 = .024$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Valence × Group</td>
<td>$F(2.82, 98.62) = 0.67, p = .562, \eta^2 = .019$</td>
<td>ns</td>
</tr>
<tr>
<td>Laterality × Valence × Group</td>
<td>$F(3.42, 119.75) = 2.17, p = .086, \eta^2 = .058$</td>
<td>ns</td>
</tr>
<tr>
<td>Caudality × Laterality × Valence × Group</td>
<td>$F(4.97, 173.76) = 0.73, p = .602, \eta^2 = .020$</td>
<td>ns</td>
</tr>
</tbody>
</table>

Note. ns $p > .05$, $^*$ $p < .05$. 
Appendix Q

CD of SPSS output files for Study 1-4

As per Griffith University thesis preparation guidelines a copy of this CD Rom is attached to the inside back cover of the dissertation in a secure sleeve arrangement.

The contents of this CD Rom include the following folders and SPSS Version 19 data output files:

Study 1
  Study 1 Psychometric Results
  Study 1 Behavioural Results
  Study 1 P2 Results
  Study 1 P3a Results
  Study 1 P3b Results
  Study 1 N400 Results
  Study 1 LPP Results

Study 2
  Study 2 Behavioural Results
  Study 2 N4 Results
  Study 2 LPC Results
  Study 2 LPN Results

Study 3
  Distractor Interference ERPs
  Distractor Interference Nogo-N2 Results
  Distractor Interference Nogo-P3 Results
  Distractor Interference Nogo-LPP Results
  Proactive Interference ERPs
  Proactive Interference Nogo-N2 Results
  Proactive Interference Nogo-P3 Results
  Proactive Interference Nogo-LPP Results
  Go ERPs
  Go-P3 Results
  Go-LPP Results

Study 4
  Discriminant Analysis Results
  Appendix Discriminant Analysis Results

Normality Data (Skewness and Kurtosis) for the Remitted Group
Appendix R

Participant Instructions for the ERP Task (the Old/New task): Study 2

Please sit as still as possible and attend to the words, which will be presented individual on the computer monitor. Prior to each word you will see a small white dot. Look at this directly and get ready for a word to be shown. A word will then appear on the screen briefly, it will be replaced with a black screen for about two seconds.

As soon as the word is shown you are required to indicate if you recognise the word from the previous task. You will make this response by pressing the “YES” or “NO” button on the computer keyboard. Please make this decision as quickly as possible (you will only have until the end of the black screen to complete this response).

During this test, please keep relaxed and still as possible. Try you best to minimise eye blinks, movements (except answer responses) and speaking during recording of your brain waves. If you need to blink, please do so during the screen with the white dot.
Appendix S

Scree plot for control participants for Study 2
Appendix T

Scree plot for remitted participants for Study 2
Appendix U

Scree plot for MDD participants for Study 2
Appendix V

Participant Task Instructions for the Go/Nogo task: Study 3

Please sit as still as possible and attend to the words, which will be presented individual on the computer monitor. Prior to the presentation of stimuli you will see a small white dot. Look at this directly and get ready for the stimuli to be shown. Two three-word lists will then appear on the screen. One will be printed in red ink and the others printed in blue ink. You will have approximately eight seconds in which to memorise these two word lists. The lists will then disappear, and the screen will go black for a second.

A coloured frame will then appear around the screen. This will either be red or blue in colour. It is used to cue you to which list to remember and respond to in the following task.

- If it is red, you are required to remember and only respond (by pressing the “YES” key) to those words presented in the red list.
- If it is blue, you are required to only remember and respond to words that were presented in the blue list.

A random presentation of nine words will be shown following the presentation of the coloured frame. Each of these words will appear individually and very briefly. When a word from the cued list is presented press the “YES” key as quickly as possible. Do not make any responses to any other word.

This procedure will be repeated 20 times, using different three-word lists in each trail. The start of a new trail is signalled by the presentation of the white dot.

During this test, please keep relaxed and still as possible. Try you best to minimise eye blinks, movements (except answer responses) and speaking during recording of your brain waves. If you need to blink, please do so during the screen with the white dot.
Appendix W

Scree plot for control participants Nogo data for Study 3
Appendix X

Scree plot for remitted participants Nogo data for Study 3
Appendix Y

Scree plot for MDD participants Nogo data for Study 3
Appendix Z

Scree Plot for Go data for Study 3
Appendix AA

**Discriminant Analysis - Separation of depression status based on Joormann et al.’s cognitive control model**

**Separation of Depression Status based on Joormann et al.’s (2007) Model**

A supplementary discriminant analysis to that completed in Study 4 was conducted to determine group prediction based on current depressed/non-depressed status. The behavioural markers of inhibitory control deficits included the distractor interference and proactive interference error bias scores; markers of mood-congruent memory biases scores included participants free-recall and recognition memory bias scores; and emotional regulation processes was measured by their self-reported use of rumination in the RRS and the use of reappraisal and suppression techniques on the ERQ. Wilk’s lambda was used to evaluate the significance of the discrimination function as a whole.

The analysis was also conducted on the original sample of 73 cases (30 currently depressed, 43 not depressed). The discriminant function found a strong connection between groups and predictors, $\Lambda = .394$, $\chi^2(7, N = 73) = 62.83, p < .001$. A canonical $R^2$ of .60 between predictors and groups was found (see Figure A6).
Figure A6. Discriminant functional analysis for cognitive processes implicated in Joormann’s (2011) model of depression. Group centroid scores indicated by thin grey line (non-depressed group centroid score = -1.02; depressed group centroid score = 1.46).

The structure (loadings) matrix of correlations between predictors and the discriminant function, as seen in Table A6, suggests that the best predictors for distinguishing between depressed and non-depressed status are increase use of rumination, decreased utilisation of emotional regulation, and greater mood-congruent
free-recall and recognition memory for negative than positive stimuli. Although only small, greater proactive interference deficits for negative than positive information also loaded on this function.

Table A6

 Discriminant Analysis Structure Matrix between Predictors and Discriminant Functions

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rumination (RRS-Total)</td>
<td>.819</td>
</tr>
<tr>
<td>Free-Recall Negativity Bias</td>
<td>.438</td>
</tr>
<tr>
<td>Emotional Regulation (ERQ-R)</td>
<td>-.453</td>
</tr>
<tr>
<td>Recognition Memory Negativity Bias</td>
<td>.413</td>
</tr>
<tr>
<td>Emotional Suppression (ERQ-S)</td>
<td>.320</td>
</tr>
<tr>
<td>Proactive Interference Negative Error Bias</td>
<td>.238</td>
</tr>
<tr>
<td>Distractor Interference Negative Error Bias</td>
<td>.136</td>
</tr>
</tbody>
</table>

Eigenvalue 1.54
Table A7 contains the classification means for the two participant groups.

Table A7

*Group Means (SD in parentheses) on the Discriminant Functions for the Predictor Variables*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Clinical Status</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not-Depressed</td>
<td>Depressed</td>
</tr>
<tr>
<td>Rumination (RRS-Total)</td>
<td>37.56 (12.69)</td>
<td>61.37 (10.10)</td>
</tr>
<tr>
<td>Free-Recall Negativity Bias</td>
<td>-5.58 (5.90)</td>
<td>0.67 (5.47)</td>
</tr>
<tr>
<td>Emotional Regulation (ERQ-R)</td>
<td>31.69 (5.52)</td>
<td>24.13 (8.16)</td>
</tr>
<tr>
<td>Recognition Memory Negativity Bias</td>
<td>-16.59 (12.98)</td>
<td>-4.52 (9.72)</td>
</tr>
<tr>
<td>Emotional Suppression (ERQ-S)</td>
<td>12.72 (4.41)</td>
<td>16.47 (5.10)</td>
</tr>
<tr>
<td>Proactive Interference Error Bias</td>
<td>-1.32 (9.00)</td>
<td>4.55 (11.12)</td>
</tr>
<tr>
<td>Distractor Interference Error Bias</td>
<td>-1.01 (3.05)</td>
<td>0.11 (3.66)</td>
</tr>
</tbody>
</table>

Jacknifed (one case at a time deleted) quadratic classification method found 62 of the 73 participants (84.9%) were correctly classified based on the linear combination of the predictor variables, compared with the approximate 38 (52%) who would have been properly classified by chance alone. These results are outlined in Table A8.
Table A8

*Discriminant Analysis Jacknife Classification Results (percentage classified in parentheses)*

<table>
<thead>
<tr>
<th>Predicted Group</th>
<th>Actual Group</th>
<th>Non-Depressed</th>
<th>Depressed</th>
<th>Total n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Depressed</td>
<td>36 (83.7%)</td>
<td>7 (16.3%)</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Depressed</td>
<td>4 (13.3%)</td>
<td>26 (86.7%)</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>