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The Effects of Late-Life Depression on Memory

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Abstract

Depression is a common mental health issue. It can result in a number of difficulties with cognitive functions such as memory. This is particularly pertinent for those experiencing late-life depression (those aged 65 years and older), as it can lead to a reduced quality of life. The relationship between depression, short-term memory, working memory, and prospective memory in the literature was explored, with a special emphasis on memory and late-life depression. In general, past research is somewhat mixed although tends towards depression having a negative impact on all the above types of memory.

The impact of depression on short-term, working, and prospective memories was investigated in a group of younger adults aged 20-29 years ($n = 50$) and older adults aged 70-79 years ($n = 50$). A between groups design was used, with each participant completing measures of depression, short-term memory (recall list), working memory (letter-number sequencing and reading span tasks), and prospective memory (with both time-based and event-based tasks). As moderating factors of memory, processing speed and an estimate of IQ were measured. Anxiety and perceived stress were measured as moderating factors for depression.

The results of the present study were mixed. There were small effects for Depression and Depression $\times$ Age interactions across some of the working memory and time-based prospective memory analyses. Interesting results were obtained when the moderators were investigated. Visual processing speed and IQ had some small to medium effects for Depression and Depression $\times$ Age interactions for recall, reading span, and time-based prospective memory, although these effects were not always in the anticipated direction. Similar variable patterns appeared when the impact of anxiety and stress on memory were analysed.

The implications of these mixed results are that a review of the way the constructs of depression, anxiety and stress are defined and measured is necessary for ongoing research in this area. Tests of depression are variable and have correlations with each other that are only marginally higher than their correlations with anxiety and stress measures. Difficulties with construct measurement extend to tests of working memory, with low correlations between working memory tests. This indicates that these tests may not be measuring the same things, yet current research on depression and memory often compares findings across multiple depression scales and tests of memory. This may have led to the inconsistent findings in the literature and may continue to do so until the issues of construct definition and measurement are addressed.

Preface

The decision to explore another aspect of memory and ageing came easily to me, building on my Master of Arts thesis which investigated the effects of differing berry juices on cognitive functioning in older adults. It was through my Master's degree that I learnt of prospective memory and became greatly interested in this topic. I knew that I wanted to do research with prospective memory, and became fascinated by the relative lack of existing research when compared with retrospective types of memory. I was especially puzzled about this research gap when I thought of the everyday implications of prospective memory failures. From there, I became interested in ecological validity and the types of memory relied on for a high quality of life. Through reading the literature, I became interested in the impact of depression on memory, and how depression-related memory impairment could affect a person's daily functioning. While my interest in the everyday aspects of memory remained, I became aware that the state of the research on depression-related memory impairment was not yet ready for a focus on ecological validity. There is still much research to be done on better defining the concepts and theories of why such impairments may occur, and why the study of the effects of depression on memory has yielded such inconsistent results.

With this understanding that further research into the relationship between depression and memory was needed in order to clarify inconsistencies in current research, I decided to adopt a more traditional quasi-experimental design which was matched to the literature I was reviewing. This led to my choice to focus on three types of memory; short-term, working, and prospective memory. These types of memory were chosen for two reasons. Firstly, they were chosen in acknowledgement of my original wish to have an aspect of my study with everyday relevance, and are the types of memory that we use to carry out our activities of daily living (further evidence of these origins can be seen in the free recall task used in this study, which is a shopping list task). Secondly, my primary interest was prospective memory, and this has both

short-term and working memory components. This thesis reviews the construct of, and models of, depression. It explores models of memory, and how age and depression impact memory functioning. Frameworks that have been proposed to account for depression-related changes in memory seen in some studies are discussed, leading to the rationale and hypotheses for the present study.

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Chapter One

Depression

This chapter briefly addresses depressive symptomatology, and reviews different frameworks that have been proposed over time to conceptualise depression. These frameworks have been sampled from cognitive, behavioural, and biological perspectives. Lastly, late-life depression is defined and discussed.

Depression as a clinical syndrome is more than a feeling or emotion. It is thought to last at least four months if left untreated, and while most people will make a full recovery from a depressive episode, subclinical depressive features can last from months to years after the main episode has resolved in under a third of those with depression (American Psychiatric Association [DSM-IV-TR], 2000). There are many and varied symptoms and symptom patterns seen in depression. A common framework used to explore these symptoms is to categorise them into the affective or emotional domain, cognitive domain, behavioural domain, and the physical domain.

Affective/emotional symptoms

When people talk about depression, they are most commonly referring to a feeling or mood. The feeling of depression is often characterised by feelings of low mood and dysphoria (Carr & McNulty, 2006). As with all emotions, the feeling of depression can range in severity. In its mild form, it can be a sense of vague unhappiness that fluctuates throughout the day. In more severe cases it can be experienced as a smothering and constant sense of desolation and despair. However, these features are not essential, and the affective component of depression can be experienced as irritability, anhedonia, or a sense of

emptiness by some (American Psychiatric Association [DSM-IV-TR], 2000). A combination of such feelings is common.

Behavioural Symptoms

A significant symptom of depression is withdrawing from social contact and from previously enjoyable activities. This is thought to be partially from another behavioural symptom, the reduction in motivation, and also from the reduced sense of enjoyment often experienced (Hammen & Watkins, 2008). Reduced motivation can extend to a lack of impetus to carry out activities of daily living. In mild cases, a person experiencing depression may still be able to carry out such activities but receives little sense of reward from them.

Physical Symptoms

There are a number of physical symptoms that can be experienced including changes in appetite and weight, fatigue, loss of interest in sex, and disrupted sleep. Some people experiencing depression report a chronic sense of tiredness and are easily exhausted. There is often a disruption to sleeping patterns which varies between people. Some people experience early morning waking and have great difficulty going back to sleep again, despite feeling tired, while others may have sleep that is disturbed throughout the night (Sadock & Sadock, 2007). Others can experience hypersomnia, sleeping considerably more than they used to. There can also be diurnal variation in mood. People with depression can feel worse in the morning, and notice improvement in their mood across the day (Carr & McNulty, 2006).

People affected by depression may experience psychomotor retardation – a sense of being slowed or weighed down in action or activities. Or they may experience psychomotor agitation, which is a sense of feeling restless, 'keyed up', and twitchy (Sadock & Sadock, 2007).

Cognitive Symptoms

Self-esteem is generally low, with depressed people placing little value on their own self-worth (Carr & McNulty, 2006). Accompanying this can be a sense of guilt and internal blame as the person with depression assumes disproportional responsibility for negative events (Beck & Alford, 2009). Another cognitive symptom is a pervasive sense of pessimism and cynicism, in which expectancies for the future are low and situations seem hopeless (Young, Rygh, Weinberger, & Beck, 2008). The cognitive theory of depression will be discussed further in a section below. This is because there is some evidence that the cognitive symptoms of depression (such as rumination on negative thoughts) may have an impact on memory functioning.

As well as these characteristic negative thoughts, there are other cognitive features of depression such as reduced attention, concentration, memory functioning, and executive functioning. In older adults, this deterioration in memory can lead to difficulties in differential diagnosis as health professionals debate depression versus dementia for a patient presenting with memory difficulties (Davidoff & Ujkaj, 2009; Steffens, 2008; Wright & Persad, 2007).

Cognitive and Behavioural Models of Depression

Beck's model

Beck's cognitive model of depression was initially generated in the 1960's in an attempt to provide a conceptualisation of depression that could then be used to inform treatment (Beck, 2005). He expressed dissatisfaction with the ideas of the time in which conceptualisations of depression tried to assign functions to depressive symptoms, and in which such conceptualisations could be ascribed to psychopathology as a whole without differentiating depression specifically (Beck & Alford, 2009). In response to this, he built on the work of cognitive theorists of the day, including Albert Ellis, to create his own conceptualisation of depression. He wanted to create a model that had empirical support for its

construction of psychopathology as well as for efficacy in its therapeutic strategies (Beck, 2005).

There are two central components to this cognitive model of depression; the cognitive triad and the negative information processing bias. The first of these components, the cognitive triad, refers to how people with depression perceive their world, their future, and themselves. Beck proposed that people with depression see their world in a negative shade. They can feel their world consists of obstacles, that they are surrounded by ongoing losses, and often believe neutral events to be unfavourable or disparaging (Beck, 1991). The future is seen as hopeless and bleak –with no end to the negative emotions in sight. The same negative filter is applied to the self which can be seen as inadequate, unlikeable, and a failure. All of these features are accompanied by the belief that their negative experiences are due to innate flaws within themselves (Young et al., 2008).

One of the strengths of this theory is that it aims to tie together the groups of symptoms outlined above (affective, cognitive, behavioural, and physical symptoms). Depressive thoughts contribute to the affective as well as the behavioural symptoms of depression. If a person has negative expectations about their world, their future, and their capacity to cope, then becoming withdrawn and disinterested could be thought of as protecting them from further negative experience and failure (Beck & Alford, 2009). These distorted cognitions are the basis of cognitive therapy for depression – if the underlying thoughts can be challenged and altered, the affective, behavioural, and physical symptoms should cease.

The second central component of this cognitive model is that people with depression have a negative bias when processing information. This is a spontaneous bias in which negative information is attended to more readily than positive or neutral information (Hammen & Watkins, 2008). It is this information processing bias that lies behind some of the common cognitive

distortions seen in depression. These distortions include ignoring positive information/experiences or attributing this to luck, seeing things in a black and white manner, and jumping to unconstructive and cynical conclusions.

Over time, Beck developed the personality concepts of autonomous style and sociotropic style, in the belief that people would be affected by different precipitating factors, different symptoms, and different views of negative events, as well as different core beliefs. The autonomous personality profile is characterised by independence and personal success, while the sociotropic personality profile is characterised by social networks and valuing the approval of others (Beck, 1991). While Beck's theory of depression is one of the better known theories, other researchers such as Seligman and Abraham have proposed other cognitive and behavioural theories of depression.

Seligman and Learned helplessness

Seligman (1972) developed the original learned helplessness model of depression. He studied dogs' responses to electric shocks across several conditions. Initially he found dogs would attempt to escape the unavoidable shocks. After a while, the dogs learnt that escape was not possible and became resigned to the shocks without making any attempts to escape. Seligman then applied these findings to depression in people by proposing that when people go through uncontrollable stressors and punishing situations, they eventually give up trying to find solutions (Beck & Alford, 2009). As the dogs resign themselves to getting shocked, people with depression can resign themselves to ongoing negative experiences.

Seligman and his colleagues further developed the learned helplessness model by proposing the attributional styles model. The main tenet of this model is that people who believe their uncontrollable negative experiences are due to chronic and insidious internal characteristics are more likely to develop depression (Seligman, Abramson, Semmel, & von Baeyer, 1979). People are thought to make attributions about the causes of occurrences (both good and

bad) in their lives across three different continuums – global vs. specific, stable vs. unstable, and internal vs. external attributions (Abramson, Seligman, & Teasdale, 1978). When people attribute their negative experiences to global, stable, and internal characteristics, they are described as having a negative explanatory style. People with a negative explanatory style have an increased risk of developing depression (Sanjuán & Magallares, 2009). Beck and Seligman developed psychological models of depression, but there are also biologically-based models of depression.

Biological Models of Depression

Neurotransmitters/Monoamine Hypothesis

The three main neurotransmitters associated with depression are serotonin, dopamine, and norepinephrine, which are all monoamines. Monoamine neurotransmitters are vital to efficient running of the limbic system which is associated with drives, emotions, and some aspects of memory (Hammen & Watkins, 2008). The monoamine hypothesis of depression was developed in reaction to drug trials taking place in the 1950's. Once it was found that inhibiting the reuptake of monoamines was useful in the treatment of depression, monoamines became an area of greater interest in the biological causes of depression (Dunlop & Nihalani, 2006; S. Lee, Jeong, Kwak, & Park, 2010).

The monoamine hypothesis proposed that a lack of monoamines (predominantly norepinephrine and serotonin) was the cause of depression. However, further research and medical developments began to identify flaws in this hypothesis. Firstly, there are a number of people whose depression does not respond to antidepressant medication (aan het Rot, Mathew, & Charney, 2009; S. Lee et al., 2010). Secondly, taking monoamine precursor chemicals does not resolve depression, and reductions in monoamines often do not cause depression (Krishnan & Nestler, 2008). Thirdly, some antidepressant medications do not involve monoamine systems. Finally, while antidepressant

medications affect the availability of neurotransmitters within hours of their administration, the alleviation of depressive symptoms does not occur for several weeks (Duman & Monteggia, 2006; Krishnan & Nestler, 2008). This fact is used to argue that monoamines do not have a direct effect. Monoamines may have an indirect effect on symptoms of depression with antidepressants working by increasing neurogenesis in parts of the brain, such as the hippocampus (Warner Schmidt & Duman, 2006). This increase in neurogenesis may be behind the time-delay between administration of antidepressants and their effects on symptoms of depression. While the monoamine systems may not be a standalone cause of depression, it is clear the monoamine systems are involved in depression.

Neuroendocrine System

In the face of danger, our physiology supports survival by instigating a number of systems that focus our attention, and increase physiological activation and responsiveness. Long-term activation of these systems by the form of ongoing stressors can have a harmful impact on our physiology that is linked to the development of depression. The hypothalamic-pituitary-adrenal (HPA) axis has played a central role in understanding how depression may be maintained. In times of stress the hypothalamus releases corticotrophin releasing hormone (CRH) which then leads to the release of adrenocorticotrophic hormone (ACTH) from the pituitary (Pariante & Lightman, 2008). This prompts the release of cortisol and other glucocorticoid hormones from the adrenal glands to stimulate the biological systems that help us to respond adaptively to external stressors (Bao, Meynen, & Swaab, 2008). There is a negative feedback loop that normally controls the levels of ACTH and cortisol in the blood, maintaining the HPA stress response at a normal level. This feedback loop in the HPA axis fails in depression, and the nervous system continues to respond to a stressor in a chronic manner (Bao et al., 2008).

Depression is associated with increased cortisol levels, and elevated cortisol levels after treatment for depression have been shown to be an indicator of poor prognosis and likelihood of relapse (Hammen & Watkins, 2008). These increased levels of cortisol damage the hippocampus and emotion regulation systems when experienced over a long time (A. L. Lee, Ogle, & Sapolsky, 2002; McEwen & Sapolsky, 1995; Sapolsky, 1996). When this stress response is sustained over time in animals, it begins to disrupt the serotonin system by interfering with the release of serotonin and also reducing the sensitivity of serotonin receptors (Van Praag, 2004).

Stress-Diathesis Models

Not all individuals who experience a stressor become depressed, and not all people with depression have experienced a stressor. The stress-diathesis model seeks to tie the cognitive and biological models of depression aetiology together, along with negative life events. Under this model, some people have a pathophysiological vulnerability that predisposes them to depression, such as a genetic or neurobiological predisposition. This vulnerability is then triggered by the occurrence of a stressful event, and depression results. This predisposing vulnerability differs from person to person, and it is thought that those with a greater vulnerability are more prone to depression from smaller stressors (Ingram & Luxton, 2005).

The vulnerability, or diathesis, can also be cognitive in nature. As noted above, both Beck (1991) and Abramson et al. (1978) proposed that people tend to have a consistent cognitive or attributional style. While the research is still unclear, there is some support for an interaction between cognitive style and types of negative life events (Mazure & Maciejewski, 2003). Congruency between a person's cognitive style and a negative event is thought to be a trigger for depression, such as a person with an autonomous cognitive style experiencing some form of personal failure, or a sociotropic person experiencing the loss of a valued social or community connection.

The Stress-Diathesis model also proposes that stressors in early life may alter the development of brain structure and function, and may go some way towards explaining why the HPA axis feedback loop is disrupted in some people. People with depression and a history of childhood trauma have been found to have higher levels of cortisol for longer periods than healthy controls (Dunlop & Nihalani, 2006; Gunnar & Quevedo, 2007). A history of childhood trauma increases the likelihood of developing depression in response to stressors as an adult (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). In such cases, the relationship is additive with environmental stressors in childhood raising the vulnerability level and likelihood of developing depression in response to further stressors as an adult. The models of depression described above are thought to apply to adults of all ages; however, there are also models of depression specific to depression in later life.

Late-Life Depression

Ageing research is increasing rapidly as our population of older adults increases. Depression is one of the most prevalent mental health disorders in older adults (Beekman, Copeland, & Prince, 1999). In New Zealand, the prevalence rates for Major Depressive Disorder (MDD) in people aged 65 years and over is 9.8% (Oakley Browne, Wells, & Scott, 2006), although the prevalence of sub-clinical symptoms would be much higher. In the literature, depression in later life is sometimes divided into early onset (onset before 60 years of age) and late onset depression. There is a large amount of heterogeneity in adults with late-life depression, and studies have revealed that although there may be differences in the aetiologies of early and late onset late-life depression, the two tend to have similar presentations (Brodaty et al., 2001). For the purposes of the present research, late-life depression will refer to both early and late onset in older adults.

Late-life depression can have a considerable impact on life and daily activities. The most significant consequence is that late-life depression is associated with

an increased mortality rate (Gottfries, 2001). There are also multidirectional relationships between depression and disability, medical illness, psychosocial difficulty, as well as cognitive impairment (Alexopoulos et al., 2002). All of these factors can influence achievement of goals and daily activities, and contribute to a reduced quality of life.

The pattern of cognitive deficit seen in late-life depression is relatively global. People with late-life depression achieve at consistently lower levels across most cognitive domains (Twamley & Bondi, 2004). The cognitive domains most affected are executive function, retrospective memory, information processing speed, and visuospatial skills (Ganguli, Du, Dodge, Ratcliff, & Chang, 2006). Focussing on memory performance specifically, late-life depression can have a negative impact on working memory, and immediate and delayed recall for verbal and visual information (Butters et al., 2004; Twamley & Bondi, 2004).

The memory systems to be investigated in the present study are prospective memory, working memory, and short-term memory (as verbal recall). They have been chosen because they have a substantial impact on everyday tasks.

Summary

When defining depression, symptoms are generally clustered into the four different domains of affective, behavioural, cognitive, and physical symptoms. In a similar manner, the models and frameworks proposed to conceptualise depression are generally informed by a primary one of these domains. Beck's cognitive-behavioural model attempts to unify the four domains of symptomatology by unifying thought, behaviour, and emotion, but does not explore the biological domain to the same extent. The biological models tend to explore the physiological and environmental realms without comprehensively integrating cognitive or behavioural elements. The cognitive and biological models are of great importance to the proposed study of memory in late-life depression, as will become clearer in the next two chapters.

Chapter Two

Memory and Depression

The first goal of this chapter is to provide an overview of short-term memory, working memory, and prospective memory. The second goal is to review the literature on how these memory types are affected by ageing, depression, and late-life depression. Each of these three memory types build upon each other. Short-term memory is discussed first. This is then followed by a discussion of working memory in which there is a short-term memory component. Finally, prospective memory, which has strong short-term and working memory elements, is discussed.

Short-term Recall

In the late 1960's a popular three-part model of memory was proposed. This consisted of the sensory register, short-term store, and long-term store. The sensory register is where information from the senses is initially stored, and which decays quickly (Atkinson & Shiffrin, 1968). When information enters the sensory register, it gets processed and transferred to the short-term store (STS). The STS is where incoming information is held. The STS is limited to a small amount of information which still decays but not as rapidly as in the sensory register (Atkinson & Shiffrin, 1968). Through the use of rehearsal, coding strategies and decision/organizational strategies, information in short-term memory is transferred to long-term memory (Bower, 2000). The long-term store (LTS) is a memory store that is largely unlimited by size and where information does not decay as it does in the sensory register and the STS (Atkinson & Shiffrin, 1968).

Short-term memory (STM) capacity can be accessed through the use of free recall, cued recall, and recognition. Free recall relies entirely on self-initiated encoding and retrieval strategies.

Ageing

STM is one of the few memory systems thought to be relatively resistant to the ageing process. The Betula Study is a recent prospective cohort study that tested a large number of adults across the life-span in five-year intervals, each time adding new participants to control for cohort and practice effects (Ronnlund, Nyberg, Backman, & Nilsson, 2005). The Betula Study has shown that STM capacity remains relatively stable into later life, with those in the 75-80 year age group having a mean performance on the free recall word list task approximately 0.2 standard deviations below that of the 35-40 year olds (Nilsson, 2003).

Free recall tasks involving word lists generally start to show age-related decline once they involve having to maintain and update rehearsal at the same time with incoming information (Grady & Craik, 2000). In a meta-analysis of verbal memory span tasks, Bopp and Verhaeghen (2005) found there was a significant negative age effect for STM tasks. While significant, the actual mean differences were small and the STM span differences between older and younger adults were less than one word, letter, or number. There was greater age-related decline for backwards digit span than for STM span. This was an expected result given that STM tasks rely simply on storage while the backwards tasks require more complex processes similar to those needed for working memory.

Depression

STM has received attention in the depression and memory arena mostly in the form of recall tests (for both verbal and visual information). So far, findings as to whether or not there is a relationship between depression and STM have been mixed.

After a surge in research on the profile of memory impairment in depression, and some inconsistent and contradictory results, meta-analyses were conducted to establish the overall pattern of impairment. Burt, Zembar, and Niederehe (1995) conducted a meta-analysis of depression and memory studies to investigate memory impairment in depression, whether moderators were responsible for any impairment, and whether or not the impairment was exclusive to depression. They searched for articles between 1967 and 1991, and used 122 studies in their meta-analysis. Focussing specifically on their results for immediate recall (STM task) they found that recall of both verbal and visual stimuli were significantly affected by depression. They also found a significant difference between immediate and delayed recall for depressed participants, with immediate recall being more impaired. When looking at moderators, they found significant age effects in which the younger participants were more impaired than the older depressed participants for immediate recall. Other moderator findings were that memory impairment was more likely in inpatients as compared to outpatients, and that memory impairment was found in depression and schizophrenia but was not significant in anxiety or substance abuse disorders.

Another meta-analysis was then conducted by Veiel (1997) on memory and depression articles dating back to 1975 because they thought there were fundamental flaws in earlier such analyses (such as sample contamination from degenerative diseases in the older samples and that some studies used did not have adequate control groups for education and age). They were stricter in using studies with a diagnosis of depression, small mean age differences between target and control groups, no other mental illness (including bipolar), and equivalent education or intelligence with the control group. Thirteen studies met their stringent entry criteria. In the verbal recall and retention studies, they found that there was little variation between depressed and control groups in each study, but that there was considerable variability between studies. They proposed that this may be due to differences in

measurement and procedure as the memory tasks used are also reliant on adequate perception and attention for encoding.

In a more recent meta-analysis, McDermott and Ebmeier (2009) looked at the relationship between the severity of depressive symptoms in participants diagnosed with major depression (with severity measured by observer rating scales such as the Hamilton Depression Scale) and cognitive functioning. Focussing on their results for episodic memory, they found that increased severity of depression was related to poorer episodic memory performance. They note that depression severity accounted for approximately 10% of the variance in episodic memory scores. While a measure of STM was included in the episodic memory composite, it should be noted that this composite also contained tests of delayed recall, learning, and retention.

One of the proposed explanations for some of the inconsistent findings in research is that the memory impairment is related to specific subtypes or characteristics of depression. Austin et al. (1999) investigated the profile of cognitive impairment in depressed participants by looking at the two subtypes of DSM-III-R depression (melancholic and non-melancholic; now an out-dated distinction). As with other studies, they ran a battery of cognitive tests, which included a test of STM. They found that non-melancholic participants performed similarly to the control group, while those with melancholic depression were significantly impaired in STM and other cognitive functions. Airaksinen, Larsson, Lundberg, and Forsell (2004) initiated a longitudinal study that looked at more recent subtypes of depression. They looked at cognitive impairment in depression globally, as well as by subtype, using DSM-IV clinical and research diagnostic categories of MDD, dysthymia, mixed anxiety-depressive disorder, and minor depression. Among other tasks was a STM free recall task. Those with MDD performed significantly worse than the healthy controls on the free recall task. The mixed anxiety-depression group showed a non-significant trend towards impairment in free recall when compared with controls. Those with minor depression and dysthymia were not

significantly affected. In the longitudinal three-year follow-up, they investigated premorbid indicators of depression. They found that a deficit in free recall on its own in the initial study was not predictive of depression at follow-up, but that deficits in both free and cued recall were predictive of depression at the follow-up (Airaksinen, Wahlin, Forsell, & Larsson, 2007).

In a study focussing on the relationships of STM and attention with depression, Williams et al. (2000) found that for their participants with MDD, impairments in STM and directed attention (across a variety of tasks) were more prominent when the participants were experiencing a more severe level of depression and stress. In looking specifically at a digit-span task, they did not find any significant difference between their depressed and control participants. This study used a repeated measures design and when tested again after 10 weeks, they found the control group showed a significant improvement across the three testing sessions, indicating a practice effect. Although there was no significant difference between the control group and the depressed group with regard to digit-span performance, the fact that the control group improved over time when the depressed group did not may indicate that there is still an effect if the depressed participants are not benefiting from having done the task before.

STM and Late-life Depression

The majority of studies investigating late-life depression and memory functioning define depression using clinical criteria such as those for a Depressive Disorder as set out in the *Diagnostic and Statistical Manual for Mental Disorders* (4th edition; DSM-IV). The meta-analysis by Burt et al. (1995) described above found that age was a moderating variable in the depression and memory relationship. They defined their age groups with older adults being 60 years of age or greater. They found consistency across the studies for greater immediate recall impairment in younger adults with depression than for older adults.

In a meta-analysis focussing specifically on late-life depression, Kindermann and Brown (1997) found that while the effect size for immediate recall was medium across the studies, it was greater for delayed recall. They also found a similar pattern with recognition as compared to recall, with a greater effect size for recognition (although all effect sizes remained medium in size). In a small group of studies in their meta-analysis containing younger comparison groups, the finding from the Burt et al. (1995) meta-analysis was supported in that younger depressed adults had a higher degree of memory impairment than their older counterparts.

Although the impact on recognition tasks seems to be greater, recall tasks are still getting considerable research attention. In more recent studies, immediate verbal recall of a word list in adults aged 60 years and older with either minor or major depression were found to be significantly poorer when compared with a healthy control group (Elderkin-Thompson, Mintz, Haroon, Lavretsky, & Kumar, 2007). Also, such deficits in memory can persist after 18 months regardless of remission or ongoing depression, and regardless of antidepressant medication use (Köhler, Thomas, Barnett, & O'Brien, 2010).

Thomas et al. (2009) researched cognitive impairment in younger and older adults with major depression. They used a cut-off age of 60 years, with those below comprising the younger group, and those 60 and above comprising the older group. Using the Rey Auditory Verbal Learning Test (RAVLT) they found that those with late-life depression had greater memory difficulty than the younger depressed participants, and that this greater impairment was not solely the effect of ageing. The poorer performance seen in the group with late-life depression occurred when the total number of list learning trials from the RAVLT was analysed. Looking specifically at immediate free recall, there were no differences between the younger and older depressed adults, or between the depressed participants and the control group (using the first trial of the RAVLT word list). The older adults performed approximately 0.55 standard deviations

below the control group. Thomas et al. did not find any differences between early and late-onset depression for the older adults.

Community-based studies use epidemiological measures to identify levels of depressive symptomatology in the elderly population, rather than sampling from clinical populations. When comparing cognitive functioning and depressive symptomatology in such studies, the results are still mixed. In a longitudinal study by Ganguli et al. (2006), older adults (over 65 years of age) with depressive symptoms who remained free from dementia over the course of the follow-up were investigated. It was found that depressive symptoms were related to memory at baseline testing (the memory composite included both short-term recall and delayed recall). On the other hand, Comijs, Jonker, Beekman, and Deeg (2001) found no relationship between declining memory and depressive symptomatology. It should be noted that this was not strictly for STM as their memory score was a total score for a verbal learning task rather than just immediate free recall on its own. Another similar study found evidence of cognitive decline in late-life depression in the community. Again, their STM free recall task formed part of a composite score and the individual impact of STM could not be determined (R. S. Wilson, Mendes de Leon, Bennett, Bienias, & Evans, 2004).

It appears that while short-term memory impairment is greater for younger adults with depression, there are still reliable deficits found in those with major or minor depression in later life when compared to healthy control participants. STM deficits found in community-based studies looking at levels of depressive symptomatology rather than diagnosable depressive disorders appear to be much more questionable.

Working Memory

Dissatisfaction with the inability of the Atkinson-Shiffrin model to account for some research findings in short term memory led Baddeley and Hitch (1974) to

develop a working memory model. The working memory model is a more complex and comprehensive model of short term memory than Atkinson and Shiffrin's STS. Working memory still has the same general purpose and function as the short-term store. Working memory is seen as the limited capacity mechanism that communicates with long term memory and also manages current information (Baddeley, 1981). Information is temporarily kept in working memory while this information is in use and being processed. The information is either from a sensory source, or memories that have been retrieved from long term memory.

Working memory is comprised of four main elements; the phonological loop, visuospatial sketchpad, central executive, and the episodic buffer (Baddeley, 1981, 2003). The phonological loop consists of both a general storehouse for phonological information (lasting only a few seconds) and a rehearsal process that is based in articulation (Baddeley, 2003).

Originally it was thought that the storage process was like a loop similar to that of a tape where the information was repeated over and over again. Later research suggested that a different analogy was needed as it was found that suppressing the rehearsal process did not impact on tests of the loop as was expected when information was presented in an auditory manner (Baddeley, Lewis, & Vallar, 1984). This meant that, although the supposed rehearsal process was occupied with another task, participants were still using the phonological loop and were still able to access the information that was presented as long as it had been heard and not seen.

The visuospatial sketchpad is the visual and spatial component of working memory and also has a small, finite capacity (Baddeley, 2003). As with the phonological loop, the visuospatial sketchpad can also be broken down into components. It seems that spatial information is processed differently to visual information, although this can be difficult to test (Baddeley, 2002). There is less research into the visuospatial sketchpad than the phonological loop.

The central executive is the most loosely defined and least understood component of working memory. In the original model it was described as being the attentional and control processes that organised information held in working memory (Baddeley, 1996). The concept of the central executive soon became an opportune resource for avoiding some of the trickier questions about working memory, and properties were attributed to the central executive without being studied or understood (Baddeley, 2002). Over the years it was acknowledged that the concept of the central executive needed to be paid much closer attention.

In more recent research Baddeley (2003) proposed a fourth component of working memory called the episodic buffer. This buffer is thought to be where information from the visuospatial sketchpad and the phonological loop is combined, and also acts as a general working area for the central executive.

The central executive is thought to have four main functions. It is responsible for updating the contents of working memory in the slave systems (the phonological loop and the visuospatial sketchpad), inhibiting irrelevant information, shifting between tasks and stimuli, and co-ordinating two ongoing tasks at the same time (Collette & Van der Linden, 2002).

Neurological research supports the distinctions between the phonological loop, visuospatial sketchpad, and the central executive by locating each of these components in different areas of the brain. Research conducted by Smith, Jonides, and Koeppel (1996) established that auditory working memory tasks activate predominantly the left hemisphere of the brain, while visual working memory tasks activate mostly regions in the right hemisphere of the brain. It is thought that the central executive is associated with the frontal lobes as they are related to executive and attentional functions (Baddeley, 1996).

Ageing

Working memory has short-term memory capacity, as well having the function of being the central organisation point for the manipulation of information to be remembered and information retrieved from long-term memory. As noted above, STM already has a small but reliable age-related deficit. Because working memory has an STM component, it is already disadvantaged by the ageing process. Verbal working memory span tasks have consistently been shown to be negatively affected by age to a greater extent than basic STM tasks in meta-analysis results (Bopp & Verhaeghen, 2005).

In an attempt to start isolating specific functions of working memory that are affected by age, Gazzaley, Sheridan, Cooney, and D'Esposito (2007) investigated the relationships between age, task difficulty, and distraction in working memory. They found there were no significant interactions between age and task difficulty or distraction on the recall component of their working memory task. What they did find was an interaction for age, task difficulty and distraction on the recognition task with older adults in the high task difficulty group with an added distracter having the greatest difficulty with recognition. This led them to conclude that recognition difficulties may underlie age-related working memory impairment.

It is assumed that the location of age-related memory impairment in working memory is located somewhere other than the simple storage process, given the greater impact of the ageing process on working memory when compared with STM. When investigating central executive processes, Bopp and Verhaeghen (2007) found the aspect of verbal working memory older adults have the most trouble with is focussing attention on updating working memory contents while also inhibiting extraneous information. Focussing specifically on the inhibitory role of the central executive, Zeintl and Kliegel (2007) found that inhibition difficulties become more pronounced with advancing age. However, as with other aspects of memory research, there are studies that do not support

any greater impairment with age on executive processes such as inhibition (Borella, Carretti, & De Beni, 2008; Robert, Borella, Fagot, Lecerf, & De Ribaupierre, 2009; N. S. Rose, Myerson, Sommers, & Hale, 2009).

Depression

Research into the relationship between depression and working memory has revealed some interesting results. As with many other cognitive processes, working memory appears to be negatively impacted by depression. Initially, it was thought that the slave systems (the phonological loop and the visuospatial sketchpad) remained relatively unimpaired in depression, while the central executive was significantly impeded (Channon, Baker, & Robertson, 1993). However, further research has identified that the slave systems, too, are affected by depression.

Christopher and MacDonald (2005) proposed that the phonological loop and visuospatial sketchpad can be affected by depression, but that this is only for working memory tasks that are thought to rely on automatic processing. They found that during tasks designed to suppress the phonological loop, the difficulty of the suppression task had an effect. While the anxiety group's (having met the ICD-10 criteria for generalised anxiety disorder) and control group's performance decreased further, the depression group's performance improved significantly. They attributed this to the more difficult suppression task interfering with rumination/intrusive thoughts in depression and allowing the allocation of more resources than tasks that require automatic processing. Once the task complexity increased, those with depression may have allocated more attention to the task (away from ruminating) allowing performance to improve.

The majority of the research has continued to focus on deficits in the central executive, rather than those seen in the phonological loop and visuospatial sketch pad. It is likely the central executive has become the more widely studied system because it is the most complex and least understood of the

working memory components, and because the increased deficit (when compared with STM) is thought to lie here. As part of this complexity, the central executive is dependent on other executive functions (such as attention) which may also be affected by depression and, in turn, impact on the central executive's function.

As previously noted, the research conducted by Christopher and MacDonald (2005) looked at the phonological loop and found that performance increased with task difficulty for those with depression, and they attributed this to the suppression of rumination. Studies focussing on the central executive component of working memory have generally found that increasing task difficulty and attentional demands have a negative effect on those with depression. Impairment in the allocation of attentional resources has support from research monitoring event-related potentials (ERPs – a measurement of electrical activity in the brain) in depressed and non-depressed participants during memory tasks. Pelosi, Slade, Blumhardt, and Sharma (2000) studied working memory in a small sample of 14 participants experiencing a Major Depressive Episode, and 14 healthy controls using a modified Sternberg task. Participants were required to remember sets of numbers of 1, 3, or 5 digits in length and then to decide if a probe digit had been in the set. They found that the depressed participants exhibited a different pattern of brain activity, when compared to the healthy controls during the working memory task. The task complexity increased with the use of longer sets of numbers. As task complexity increased, the ERPs of the depressed participants showed prolonged electrical activity when compared with the healthy controls, which indicated that greater neuronal resources were being used to carry out the working memory task. The authors proposed a possible explanation - this greater activation of resources represents a shift to compensatory strategies to try to maintain performance at the increased complexity. In other studies of healthy controls, working memory tasks usually lead to increased activity in areas related to working memory, and a decrease in unrelated areas. It is possible that the high level of electrical activity shown by the depressed

participants means that they have had difficulty in focussing their attentional resources adequately (Pelosi et al., 2000). The allocation of attentional resources in depression will be addressed further in Chapter 3.

One aspect of the central executive that has generated research interest is the processes involved in updating the contents of working memory. Harvey et al. (2004) used an *n*-back task of increasing complexity to study updating differences in participants with depression (22 participants who met DSM-IV criteria for a major depressive episode) and non-depressed controls. As predicted, they found a difference between the two participant groups at each increasing level of difficulty. At increasing levels of complexity (1-back, 2-back, and 3-back) the *n*-back task required participants to update the contents of their working memory by maintaining a series of previously presented probes in a mental list to compare with future probes. As a new probe is presented, the oldest probe can be dropped from memory. For example in the 2-back task, two probes need to be maintained in memory to compare with the current probe (to see if the current probe matches the probe two items back). When the next probe in the series is presented, the contents of working memory need to be updated to remember the newest probe (while forgetting the oldest probe). The authors note that the 1-back task is likely to be the best indicator of updating processes as the more complex tasks are likely to have interference from other executive functions, such as attention and inhibition as well as updating processes. Even at the first level of complexity, the 1-back task, there was a difference between the two groups, with the depressed participants exhibiting impaired performance. This difference was not explained by short-term memory or attention difficulties as there had not been group differences on tasks designed to measure these factors. The difference seemed to be due to impairment in updating the contents of working memory (Harvey et al., 2004).

A more recent study by Joorman and Gotlib (2008) researched the updating of working memory in a memory task with emotional content. The purpose of this study was to investigate working memory ability and interference from

emotionally negative information (such as the negative, ruminative thoughts that are characteristic of depression in the cognitive models of depression described in Chapter 1). Their working memory task involved remembering two lists of three words presented side by side, with the two lists presented in different coloured fonts. The words in the lists had different emotional valences from positive through to negative. A probe word was presented surrounded by a coloured frame. The colour of the frame was indicative of the word list that participants were supposed to compare the probe word to, before deciding whether or not it was in that list. It was accurately predicted that depressed participants would exhibit greater interference from the words of negative valence than would healthy control participants. This interference was evidenced by the depressed participants being slower to decide if the probe word matched the correct list when the probes were negative words. This difference was also found for a group of control participants who had undergone sad mood induction at the time of the experiment. Their findings were specific to the negative words, with positive information of equivalent strength and length not showing any interference effects in the memory task. They also found that this interference was related to ruminative tendencies in their depressed participants. The Beck Depression Inventory scores and intrusion effects scores explained 61% of the variance in the scores of the rumination self-report scale in the depressed participants. Joorman and Gotlib's research provides support for the argument that rumination in depression impairs the updating of working memory with regard to the removal of negative information. That is, working memory may have a contributing role to the maintenance of depression as negative information and ruminative thoughts are maintained in working memory rather than replaced by other information as is thought to happen in normal mood self-regulation (Joormann & Gotlib, 2008).

Late-Life Depression and Working Memory

At this stage there still seems to be a fairly minimal amount of research into depression and working memory, and as with other areas of memory, there is comparatively less research on late-life depression and working memory. The impact of depression on working memory in later life appears to follow a similar pattern to both normal age-related changes in memory, and those seen in younger adults with depression. Nebes et al. (2000) found that both working memory and processing speed were significantly affected in their depressed older adult participants when compared with healthy age-matched controls. Interestingly, they also found the deficits in working memory and processing speed mediated the relationship between depression and other cognitive tasks such as immediate verbal and visual recall, learning, and visuospatial tasks. When this variance was accounted for, there were no longer significant effects for depression and the cognitive tasks. While processing speed is known to have a substantial impact on working memory abilities (as will be discussed further in Chapter 4), the above authors found that once processing speed was accounted for in their depressed sample, working memory still had a significant impact on the other cognitive tasks.

O'Brien, Lloyd, McKeith, Gholkar, and Ferrier (2004) conducted a six-month longitudinal study on late-life depression and cognitive function. Using a backwards digit-span task as their measure of working memory, they found that there were differences between the depressed older adults and their same age non-depressed controls. At the six-month follow-up, there was some improvement but this was across both groups (likely practice effects) and there was a continuing significant difference between the two groups. The participants who had achieved remission from depression at the six-month follow-up also maintained a significant difference on the working memory task, indicating the impairment continues after the resolution of depressive symptoms.

In summary, working memory deficits in late-life depression appear to follow the same pattern as the normal ageing process and the impairment seen in younger adults with depression. No studies were found that looked at working memory impairment and depression in younger and older adults at the same time.

Prospective Memory

Prospective memory involves remembering to act in the future. It helps us to remember and achieve our goals (Morris, 1992). It can be short or long term in nature and is generally divided into time-based prospective memory and event-based prospective memory. According to Lockhart (2000), time-based tasks involve remembering to do something at a specific time (such as leave for an appointment in 20 minutes), whereas event-based tasks require a person to perform an action in relation to another action or event (such as hanging out the washing after the washing cycle has finished).

There are two different schools of thought on the comparative difficulties of time-based and event-based tasks. One group of researchers believe there are no external cues for time-based tasks, making them more difficult, while another group believe that the time on the clock acts as an external cueing event (Graf & Grondin, 2006). A stronger distinguishing factor between the two types of prospective memory is the amount of warning that goes into retrieving a formulated plan. According to Graf and Grondin (2006), time-based tasks have a predictable progression before the event and one can remind themselves of the task at appropriate intervals. However, event-based tasks are less predictable, especially if reliant on other people, and so it is harder to know when to bring the planned event to mind.

The multiprocess theory of prospective memory was proposed by McDaniel and Einstein (2007) to provide a framework for understanding event-based prospective memory in the context of mixed research results. The three main assumptions of the multiprocess theory are that different strategies or processes

can be used for retrieval (spontaneous retrieval versus monitoring), that the strategy chosen and its efficacy are dependent on a number of variables, and that there is a general bias towards the use of spontaneous retrieval amongst individuals (McDaniel & Einstein, 2007).

The process of spontaneous retrieval is thought to occur in response to the target event or memory cue, in which this event automatically brings the intended action to mind with little or no effort (McDaniel & Einstein, 2000). One particular spontaneous retrieval mechanism that has been proposed is the reflexive-associative theory, in which an association is formed between the cue and the task to be carried out at the time of encoding (Einstein et al., 2005). If the association is strong enough, the intention should come to mind reflexively. Under the monitoring process, it is proposed that once an intention is formed, attentional resources are allocated to keep the intention in mind until the intention is to be carried out, which requires continuing cognitive effort (Einstein et al., 2005; McDaniel & Einstein, 2000).

Ageing

Prospective memory is thought to follow an inverted U-shape across the lifespan, with the greatest ability occurring between late adolescence and middle adulthood (Zimmermann & Meier, 2006). A framework to understand age-related memory changes was proposed by Craik (1986) in an attempt to unify the varying research outcomes that had been established at that stage. Craik proposed that remembering was the result of an interaction between the individual and their external environment or context. Because the environment in which the memory was created is usually not the same as the environment that retrieval is occurring in, remembering often relies on self-initiated processes (Craik, 1986). From here, he suggested that such self-initiated processes were the key to understanding changes in memory that occurred with age.

Under Craik's (1986) model it was proposed that age-related deficits in memory would be related to self-initiated process demands, with less environmental support and fewer external cues leading to a larger memory deficit, and that prospective memory is considerably reliant on self-initiated processes (Craik, 1986). Similarly, the multiprocess framework discussed earlier proposed that age-related deficits in event-based prospective memory would only occur when resource-demanding monitoring is being used to maintain the intention (McDaniel & Einstein, 2007).

Henry, MacLeod, Phillips, and Crawford (2004) conducted meta-analyses to investigate age-related changes in prospective memory due to inconsistent results reported in individual studies. They noted results had been more consistent for time-based than event-based tasks, which was ascribed to time-based tasks relying more on cognitive resources and self-initiation. Henry et al. (2004) found that while there was a trend towards greater age-related decline in time-based tasks than in event-based tasks, this did not reach statistical significance. The relationships between age and time-based prospective memory was $r = -.39$, while the relationship between age and event-based prospective memory was $r = -.34$. The level of resource demand in event-based tasks did show age-related effects in that the deficit was larger for resource demanding event-based tasks than for relatively automatic event-based tasks. They had also predicted there would be greater deficits for prospective memory tasks when compared with retrospective tasks because of the greater resource demands, yet they found that free-recall was associated with the largest age-related deficit in prospective memory.

When looking at studies conducted in naturalistic settings, age-related deficits are reduced and sometimes reversed. The above meta-analyses also investigated this phenomenon and found that older adults showed superiority over younger adults when prospective memory tasks were carried out in their natural environment (Henry et al., 2004).

Depression

The impact of depression on prospective memory has received even less attention than retrospective memory. There are a minimal number of published studies investigating prospective memory and depression. However, these point to deficits in prospective memory being related to depressed mood.

The first study of note was conducted by Rude, Hertel, Jarrold, Covich, and Hedlund (1999). They compared the performance of a sample of participants with major depression to a control group of non-depressed participants on a prospective memory task reliant on self-initiation. The task was a computer-based test requiring participants to press a computer key every five minutes during a general knowledge quiz. Participants did not have access to a clock but could momentarily check the time by pressing a different computer key which would display the time on the bottom of the screen. Rude et al. (1999) found that there was a significant difference between the two groups' performance on this prospective memory task. The mean score on the prospective memory task for the depressed participants was 3.5, as opposed to 4.7 for the non-depressed controls. They also found the two groups differed significantly on their time-monitoring behaviours, with the depressed group monitoring the time less frequently than the control group. Although the sample size was fairly small (20 participants in each group), these results suggest that like retrospective memory, depression is also related to impairments in prospective memory.

Research conducted by Harris and Menzies (1999) looked at the impact of depressed mood on prospective memory using a non-clinical sample of undergraduate students. The prospective memory task in this study was purposefully embedded in a recall task, which had a high working memory component. While making associations for a list of words that would need to be remembered later, participants were also required to make a note each time a target word (a piece of clothing or a body part) was presented. Remembering

to note the target words constituted the prospective memory task. Harris and Menzies conducted multiple regression analyses and did not find any significant relationship between depression and prospective memory ($r = -.01$). However, they did find significant relationships between anxiety and prospective memory ($r = -.28$), and between retrospective and prospective memory ($r = .25$). One explanation as to why there was no significant relationship between depression and prospective memory in this study may be to do with this being a community-based sample and the range of depression scores may have been restricted. No descriptive statistics for the participants' scores on the measure of depression (the Depression Anxiety and Stress Scale - DASS) were provided by Harris and Menzies. However, a normative study on the DASS conducted by Crawford and Henry (2003) found that the mean score in a large community sample was 5.55 (standard deviation of 7.48) out of a possible 42 points. If the scores in Harris and Menzies' undergraduate sample were similar, it may be that the range of scores on the depression subscale of the DASS was too restricted for any relationship between depression and prospective memory to be detected.

Late-Life Depression and Prospective Memory

In conducting the literature search for this review, only one study investigating the relationship between prospective memory and depression in late-life was found (refer to Appendix A for a review of the search strategies used). The study was conducted by Livner, Berger, Karlsson, and Bäckman (2008) and they used an epidemiological sample from Stockholm with participants aged 75-98 years. They investigated retrospective memory and also included one event-based prospective memory measure. They found only the retrospective component of the prospective memory task was related to depressive symptomatology. However, the task used was a relatively simple prospective memory task in that participants were required to remind the examiner to make a phone call at the end of the testing session. As noted above, event-based tasks are thought to be less cognitively challenging and more automatic than time-

based tasks, and this study may not have adequately tapped the complexity of prospective memory tasks.

Summary

Short-term memory is considered to be a basic storage space where incoming information is held for up to 30 seconds (depending on the sensory modality). In general, there are small but significant effects of age on verbal recall tasks. Research on depression and STM has produced more variable results. However, there appears to be a trend towards significant STM impairment when strict MDD criteria are used. In later life, the results continue to be mixed with some studies reporting greater impairment in STM tasks for depressed younger adults when compared with older adults and vice versa. When using epidemiological measures to quantify depressive symptomatology, there does not appear to be any significant relationship between late-life depression and STM.

Working memory builds on STM by involving a mental workspace as well as a brief storage system. Age-related working memory impairment is greater than that for STM tasks. It is assumed there are age-related deficits in the processing aspects of working memory, or possibly in the co-ordination of the process required to simultaneously store and process information, rather than just the storage component of STM. Older adults are found to have greater difficulty with the central executive processes of working memory that require updating, inhibition, and focussed attention. Working memory is also affected by depression. The main source of this deficit appears to be the central executive, although the storage processes are also affected to a lesser degree. As with ageing, the updating process appears to be affected, with some authors proposing that this is due to depressive rumination occupying attention. There is some support for impairment in allocating attentional resources in working memory tasks in depression from physiological studies showing greater areas of brain activation in depressed participants. The high level of activation is

thought to show attention being focussed on more than just the working memory task. Studies on working memory and late-life depression appear to mirror the ageing and depression results, with significant impairment occurring in samples with late-life depression when compared with healthy older adults. This impairment was found to outlast depression in one study (O'Brien et al., 2004), with older adults in remission still showing significant impairment.

Prospective memory is about remembering to carry out an action in the future. Generally, prospective memory is divided into time-based and event-based prospective memory. Time-based tasks rely on the passing of time or time of day as their retrieval cue, whereas event-based tasks rely on an environmental stimulus or occurrence to prompt retrieval. Age-related changes in prospective memory have produced mixed results, although there appears to be more consistency for age-related impairment in time-based prospective memory. In naturalistic settings, older adults generally out-perform their younger counterparts. Little research has been conducted on depression and prospective memory so far, but there is some initial evidence that time-based prospective memory is affected by depression. Only one study on late-life depression was found. In this study, using simple event-based task, there was no relationship between the task and depressive symptomatology.

Chapter Three

Explaining the Memory and Depression Link

This chapter reviews theories and models proposed to account for depression-related impairment in memory. As with the first chapter, consideration is given to a variety of models primarily in the cognitive and neurobiological domains. Two cognitive models, the resource allocation model and inhibitory control model, are discussed first. Both of these models highlight the importance of attention in memory. Neurobiological models centring on changes in depression are then discussed, with attention focussed on the hippocampus, frontal cortex, and changes to neurotransmitter functioning.

Cognitive Models

Resource Allocation Model

The resource allocation model (RAM) was proposed by Ellis and Ashbrook (1988). The underlying premise of the model with respect to depression is that cognitive capacity is occupied by the ruminative thoughts that are a symptom of depression. If cognitive capacity is limited, and some of this capacity is occupied with ruminative thoughts, other cognitive functions such as memory will have fewer cognitive resources to rely on for adequate task performance. This would mean people with depression would show deterioration in performance when compared with non-depressed people. Given that not all tasks tax cognitive resources to their full capacity, it would be expected that tasks requiring greater attention and cognitive resources would be affected by depression whereas more simple tasks may remain unaffected.

Many different research methodologies have been employed to investigate this model. Meinhardt and Pekrun (2003) studied the effects of emotional states on

ERPs in the brain during attentional tasks. All participants took part in four experimental conditions, while at the same time; they listened to different auditory tones and counted the number of either high or low tones presented (dependent on which tone group they had been assigned to). The four conditions were the viewing of emotionally positive images, neutral images, and negative images, as well as a condition in which they watched a focal point on the monitor while carrying out the secondary auditory task. The researchers monitored the P3 component of ERP (the P3 is a specific peak in brain activity that occurs around 300 milliseconds after a stimulus is presented) during the experimental tasks, a well documented indicator of resource allocation in dual-task paradigms. Generally, as the difficulty of the primary task increases, so does the amplitude of the P3 wave. As the difficulty of the secondary task increases, the amplitude of the wave decreases. They found that the P3 amplitude was reduced when the participants were viewing images from the negative and the positive conditions, indicating that both conditions required greater allocation of resources away from the secondary task (counting the target tones). While the participants in this study were non-depressed younger adults, it is interesting to note that the sad mood induction (and it is assumed negative cognitions consistent with the induced mood) still had an effect on diverting resources away from the task at hand.

Physiological responses indicative of attentional allocation have also been studied in depressed samples. Jones, Siegle, Muelly, Haggerty, and Ghinassi (2010) researched the relationship between depression and cognitive resource allocation to an attentional task as well as resource allocation to internal negative automatic thoughts, or rumination. Pupillary motility was used as the physiological indicator of resource allocation, as increasing pupil size is linked to changes in brain activity needed for information processing (such as changes in the pre-frontal cortex). The Paced Auditory Serial Addition Task (PASAT) was used as the attentional task because it assesses information processing as well as being known to induce frustration. Pupillary motility occurring at times other than the presentation of the auditory stimuli was assumed to reflect the

allocation of resources to internal thoughts or rumination. Consecutive errors on the PASAT were considered due to negative evaluations of performance interfering with the task. They found that depressed participants were more likely to make consecutive errors on the PASAT. They also found that off-task pupillary motility was associated with poorer PASAT performance, and the depressed participants were more likely to have off-task pupillary motility. The authors argue these results support the premise that depressed participants allocated more cognitive resources away from the task at hand, which corresponded to a decline in performance on the attentional task. This is consistent with Joormann and Gotlib's (2008) study discussed earlier (Chapter Two) in which self-reported rumination was associated with updating difficulties in working memory due to interference of negative information in depressed participants.

Inhibitory Control

One of the major models of age-related decline in working memory performance was proposed by Hasher and Zacks (1988). They postulated that the mechanism underlying the changes seen with ageing is a decline in inhibitory control. In this model, inhibition is thought to become more inefficient with age which then leads to off-task information interfering with and competing for working memory capacity. This failure of inhibition then leads to poorer performance in working memory for the desired task.

Inhibition of irrelevant information or stimuli is one of the primary functions of the central executive in working memory (Collette & Van der Linden, 2002). Research by Zeintl and Kleigel (2007) supports inhibition difficulties in ageing coming from either the access component of inhibitory control allowing irrelevant information to occupy working memory resources, or the updating component not removing the irrelevant information efficiently.

Other research supports age-related differences in verbal working memory tasks coming from deficits in central executive inhibitory functions, such as

those of supervision, but also from wider central executive functions such as the co-ordination of working memory resources (Bopp & Verhaeghen, 2007). As defined by Bopp and Verhaeghen (2007), supervision refers to the process of selectively attending to task-relevant information and excluding task-irrelevant information. The co-ordination function of the central executive is responsible for managing information in different storage systems including the phonological loop, visuospatial sketchpad, and long term memory. Both of these roles were found to be compromised for verbal working memory tasks (Bopp & Verhaeghen, 2007). Interestingly, the researchers found that not all aspects of inhibition are affected by age (with only the supervisory and co-ordination processes being affected), and also there were no age-related inhibitory control deficits for visuospatial tasks. Age-related difficulties with inhibitory control have also been reported where changes in frontostriatal and sensorimotor systems have been found to be associated with inhibitory control deficits in ageing (Sweeney, Rosano, Berman, & Luna, 2001; West & Alain, 2000).

The finding that age-related working memory deficits increase in relation to level of resource demand has implications for the role of working memory in prospective memory tasks. There is a retrospective component to prospective memory in that the content of the intention is remembered from when it was originally encoded. In addition, there is also an attentional component that monitors and recognises the prospective cues (R. E. Smith & Bayen, 2004). There is a working memory component in prospective memory, thought to be located in the central executive, that co-ordinates the recognition of the prospective memory cue and recognition that an action is required (R. E. Smith, 2003; West & Bowry, 2005). It has already been shown that advancing age has a detrimental effect on working memory, particularly the central executive component. If working memory processes are used in keeping the intended action in mind and monitoring the environment for recall cues in prospective memory, then it is little wonder that there are increasing deficits in prospective memory with increasing age for older adults. Nevertheless, while statistically

controlling for the effects of working memory reduces the age-related effects in prospective memory tasks, there is still a residual age effect not accounted for by working memory (Cherry & LeCompte, 1999; West & Craik, 2001).

It has also been established that both processing speed and inhibitory control are a part of working memory functioning. Whether or not processing speed and inhibitory control contribute to prospective memory over and above their effect on working memory is an interesting research question. West and Craik (2001) set about answering this question by investigating a number of relationships between prospective memory and the above cognitive functions of working memory, inhibitory control, and processing speed. They found the relationship between age and prospective memory was reduced but still significant after controlling for processing speed. They found the relationship between age and prospective memory was also still significant once inhibitory control was accounted for. As expected, they also found the same pattern with working memory. Age-related decline in prospective memory eased but was still significant once working memory was controlled for. When they controlled for all the above variables at the same time (using regression analysis), they found that the relationship between age and prospective memory was no longer significant, and noted the only factor making a significant unique contribution was processing speed. So while processing speed and inhibitory control did have their own effects on the relationship between age and prospective memory independent of working memory, once working memory, inhibitory control, and processing speed were accounted for at the same time, the relationship between prospective memory and age was no longer significant. There is further research that argues that working memory ability (including processing speed and inhibitory control as part of their working memory model) is responsible for the differences in prospective memory abilities for both younger and older adults (M. Martin & Schumann-Hengsteler, 2001). Interestingly, their results are similar to those of West and Craik (2001); however, they account for processing speed and inhibitory control as

components of working memory, rather than individual cognitive functions as in other research. These studies help to highlight the importance of accounting for related cognitive functions in memory research, and also the importance of looking at component memory processes that may be involved in the more complicated and convoluted models of memory, such as prospective memory and working memory.

Hasher and Zacks' (1988) inhibitory control framework has been applied to the cognitive deficits (with an emphasis on working memory) seen in depression, as well as in ageing. One of the ways in which it is thought inhibitory control impacts on cognitive functioning in depression is through reduced inhibition of ruminative thinking (Joormann, Yoon, & Zetsche, 2007). There is considerable research to support a negative processing bias in depression, in which attention to negative information is not inhibited (Goeleven, De Raedt, Baert, & Koster, 2006; Gotlib, Yue, & Joormann, 2005; Joormann, 2004). As ruminative thoughts in depression contain negative themes by definition, attention may be directed towards the thoughts and away from the task at hand, accounting for poorer performance on memory tasks. Inhibition is vital for adequately updating working memory and without sufficient inhibitory control, working memory and cognitive tasks that rely on working memory would be affected (Joormann & Gotlib, 2008; Joormann et al., 2007).

Given that inhibitory control is affected by both ageing and depression, it is hardly surprising that depressed older adults experience significant inhibitory impairment. Lockwood, Alexopoulos, and van Gorp (2002) studied the impact of depression on the executive function task of inhibitory control (as well as two types of attention) in a sample of younger adults (aged 20-60) and older adults (aged ≥ 61). They measured inhibitory control with multiple measures including Trails B (requiring participants to follow alphanumeric sequencing) commission errors on the Continuing Performance test (responding when there is no target), and by errors of perseveration on the Wisconsin Card Sorting Test and the California Verbal Learning Test. They found a significant Age x

Depression interaction on their inhibition task, with the depressed older adults in this study showing greater inhibitory impairment than both depressed younger adults and non-depressed older adults.

Studies showing inhibitory control deficits in late-life depression are supported by brain structure investigations. Elderkin Thompson, Hellemann, Pham, and Kumar (2009) found a relationship between late-life depression and smaller orbitofrontal cortex volume (a structure known to be involved in inhibition). Frontal dysfunction in late-life depression was also found in a study investigating inhibitory control and event-related potentials (Zhang, Zhao, & Xu, 2007). Given that it has already been acknowledged that the frontal and pre-frontal cortices are affected by ageing and depression, these findings linking such dysfunction with inhibition impairment come as no surprise. Such neuropsychological changes have a complex but important role in ageing, depression, and memory functioning.

Neuropsychological Changes Seen in Depression

There are changes to brain structures and processes that may underlie the changes in memory functioning that have been highlighted in ageing, depression, and late-life depression. The areas of research that have received the most attention are volume loss within the hippocampus and frontal cortex, HPA axis impairment, white matter lesions, and neurotransmitter deficits.

Hippocampus

The hippocampus is a structure that is part of the limbic system, and found bilaterally in the temporal lobes. It plays an important role in learning and the formation of memories. In particular, the hippocampus is associated with declarative, spatial, and contextual memory. A compromised hippocampus is correlated with a decline in memory performance, particularly in episodic memory (Golomb et al., 1996; Persson et al., 2006).

Loss of hippocampal volume is associated with both ageing and depression (Bell-McGinty et al., 2002; Raz et al., 2005; Saylam, Ucerler, Kitis, Ozand, & Gonul, 2006). In a study of normal ageing and Alzheimer's disease, De Leon et al. (1997) found that hippocampal atrophy was associated with age in the normal ageing group. In the group of participants aged 76 to 90 years old, 48 % had hippocampal atrophy. The relationship between hippocampal volume and MDD was studied by Colla et al. (2007). They found there was a significant loss of hippocampal volume in participants with MDD when compared with matched controls. They also found there was a negative correlation between increasing age and decreasing hippocampal volume loss, and between hippocampal volume and the duration of MDD.

The loss of hippocampal volume seen in participants with MDD also has an effect on memory function. Hickie et al. (2005) undertook research assessing memory deficits and hippocampal volume in a group of adults. They found that age, age of onset, and memory deficits were related to hippocampal atrophy in the participants with MDD. They suggest that the memory problems associated with depression are a result of the structural changes in the hippocampus rather than lack of effort and motivation.

The causes of hippocampal volume loss are still under intense investigation. Increased cortisol levels have already been shown to occur with depression and related HPA-axis dysfunction (as discussed in Chapter 1). Continued exposure to cortisol has also been linked with cognitive decline and hippocampal volume loss (O'Hara, Coman, & Butters, 2006). It is thought that elevated cortisol levels may lead to the death of neurons or a decrease in neurogenesis in the hippocampus.

Research into this area has been stimulated by Cushing's Syndrome in which there is a significant surplus of cortisol and other hormones associated with (among other symptoms) depression and impaired cognitive functioning (Forget, Lacroix, Somma, & Cohen, 2000). So far, studies investigating cortisol

levels, hippocampal volume, and memory have had mixed outcomes. In a study of patients treated with prednisone (a corticosteroid) for various rheumatic conditions, it was found that corticosteroid use was associated with smaller hippocampal volume and declarative memory deficits (E. S. Brown et al., 2004). However, a similar study found that corticosteroid treatment was associated with a decline in memory, but not in hippocampal volume as was expected (Hájek, Kopeček, Preiss, Alda, & Hóschl, 2006).

Closer examination of hippocampal atrophy has revealed that loss of volume in the posterior regions is significant, but not in the anterior regions (Neumeister et al., 2005). It is in the posterior region of the hippocampus that the structure responsible for neurogenesis, the dentate gyrus, is located (Henn & Vollmayr, 2004). It is possible that a reduction in neurogenesis in the dentate gyrus is at least partly responsible for the loss of volume sometimes seen in the hippocampus (Duman, 2004).

Getting direct evidence for a reduction in neurogenesis being associated with depression in humans is difficult. However, there is evidence that corticosteroids are one of the most potent mechanisms of decreasing neurogenesis in the hippocampus (Sapolsky, 2004). It is reasonable to believe that a reduction in hippocampal neurogenesis is occurring for people suffering from depression, particularly those with increased cortisol levels.

It has been proposed that deficits in the hippocampus could be at least partially responsible for the maintenance of depression through memory problems creating a negative bias (Becker & Wojtowicz, 2007). Because the hippocampus is associated with forming declarative memories, any reduction in this function would lead to ineffectual processing of memories. Research findings consistently show that people with depression are more likely to recall negative than positive information (Gotlib et al., 2004). This means people with depression are attending to and processing information in different ways to people without depression, and this may be lowering their mood.

Frontal and Pre-frontal Cortex

As well as hippocampal changes, changes in the prefrontal cortex are related to both ageing and depression. Smaller frontal cortex volumes have been found in depressed older adults when compared to non-depressed older adults (Bell-McGinty et al., 2002).

Changes in the frontal cortices are important because studies investigating the neurophysiological correlates of prospective memory have found increased activity in the prefrontal cortex (particularly Brodmann area 10; BA 10) and also in the hippocampus. Okuda et al. (2007) investigated the associations between brain activity, and both time-based and event-based prospective memory. In their time-based task for Study 1, participants were required to clench their hands at certain intervals (without the use of a clock), while engaged in a serial addition task. The event-based task was to clench their hands when presented with the digit "7" in the addition task. In Study 2, the time-based task was embedded in a shape recognition task and required participants to press a particular key at 1 minute intervals (with the aid of a clock). The event-based task was to press the same key when presented with an identified cue stimulus. Using Positron Emission Tomography (PET) they studied the regional cerebral blood flow in participants in the two prospective memory conditions, and found that different areas of the prefrontal cortex (Brodmann area 10) were activated dependent on whether or not the task was time-based or event-based. More specifically, they found higher levels of activation in the medial BA 10 for time-based tasks, and in the lateral BA 10 for event-based tasks.

T. Martin et al. (2007) used magnetoencephalography (MEG) to study brain activity in an event-based prospective memory task, among other tasks. They found that the hippocampus was activated in the memory tasks (both prospective and retrospective) but not for other tasks, which suggests that a recognition check of the stimulus had occurred. T. Martin et al. also found increased activation in the frontal lobes. If the hippocampus and frontal lobes

are important for prospective memory functioning, and relationships exist between smaller volumes and late-life depression, then this volume loss may be related to the memory difficulties seen in late-life depression.

White Matter Hyperintensities

White Matter Hyperintensities (WMH) is a term used to describe two types of lesion in the white matter of the brain: periventricular and deep white matter lesions. WMH are known to occur with increasing age and are common in older adults with depression, especially in the frontal lobes (Taylor et al., 2003). They have also been found in the caudate nucleus, an area of the brain known to be involved in cognitive functions and emotion regulation (Hannestad et al., 2006). It is thought these lesions interrupt neural pathways important for cognitive processes such as memory, and also for emotion regulation (Teodorczuk et al., 2007; Van Petten et al., 2004).

Researchers began to think that cerebrovascular problems may have a role in late-onset depression. A study by de Groot et al. (2000) of 1,077 participants between the ages of 60 and 90 years, compared WMH with Centre for Epidemiological Studies Depression Scale (CES-D) scores and current or historic episodes of depression. They found that there was a linear relationship between WMH and CES-D scores, with those with higher numbers of WMH more likely to have experienced depressive symptoms. They also reported a linear relationship between severity of WMH and age of onset for those participants who had experienced depression. Their results indicate that WMH severity is associated with a history of late onset depression but not early onset (prior to 60 years of age). The authors proposed disruptions to subcortical tracts involved in WMH could be responsible for changes in mood.

Some researchers propose depression-related memory impairment in older adults is linked to age-related increases in WMH. Kramer-Ginsberg et al. (1999) investigated WMH in a sample of older adults (over age 65) with a diagnosis of major depression (according to the DSM-III-R). They looked at interactions

between level of WMH severity, current experience of depression, and memory scores (as well as other cognitive functions). They rated the severity of the WMH for each participant. They found that depressed older adults in the moderate-to-severe WMH group performed significantly worse than depressed older adults without deep WMH and non-depressed older adults on composite scores from the Wechsler Memory Scales. This study provides some understanding of why memory dysfunction may occur with depression, and perhaps why there are some mixed findings in the research. If samples in other studies have had participants with differing levels of WMH, this could lead to inconsistent results. This study is somewhat limited by its use of an analysis of covariance (ANCOVA) design. Such a design is not recommended in studies where factors of interest cannot be randomly assigned (such as depression) as this can create a bias in the ANCOVA analysis. Reasons for this will be discussed further in Chapter 5.

Attempts have been made to establish whether WMH are a cause of depression and its related memory impairment. Teodorczuk et al. (2007) conducted a longitudinal study of the relationship between WMH and the development of late-life depression in a sample aged 65-84 years. They took baseline measures of WMH using Magnetic Resonance Imaging (MRI), depression using the Geriatric Depression Scale (GDS), and also took a history of any episodes of depression that required treatment. They found that white matter abnormalities evident at the baseline testing predicted depressive symptomatology (on the GDS) at a one-year follow-up. This was independent of other variables such as occurrence of stroke, quality of life factors, baseline depression, increasing disability, and education level. While the white matter changes could predict depressive symptomatology using the GDS, they did not predict clinically significant depressive episodes. The authors believe this may be due to the power of the study not being sufficient to detect such changes, or that depressive episodes were under-reported in their sample.

Neurotransmitter Deficits

The role of monoamines (serotonin, dopamine, and norepinephrine) in depression was outlined in Chapter One. While they are not the sole cause of depression, they have a clear role in the maintenance of depressive symptomatology. A change to these neurotransmitter levels and receptor sites also occurs with ageing, as well as depression. Post-mortem studies have revealed age-related decreases in some serotonin receptors in certain areas of the brain such as the frontal and occipital lobes, as well as the hippocampus (Meltzer et al., 1998). Reduced serotonin has been linked to inattention, poor executive functioning, and long-term memory difficulties (Schmitt, Wingen, Ramaekers, Evers, & Riedel, 2006). Research on the role of serotonin in working memory is more inconsistent, with researchers finding both for and against a relationship between working memory and serotonin (Barch, 2004; K. A. Ellis & Nathan, 2001; Mendelsohn, Riedel, & Sambeth, 2009).

Like serotonin, changes in the dopaminergic system have also been linked to ageing and memory. The dopamine pathways are thought to be especially susceptible to the ageing process (Stark & Pakkenberg, 2004). These age-related losses in dopamine also have an impact on cognitive functioning, as well as depressive symptomatology. In a review of current research on the dopaminergic system in ageing, Bäckman, Lindenberger, Li, and Nyberg (2010) found that studies have linked dopamine function with tasks demanding higher cognitive functions such as working memory. In younger adults, dopamine release increased during tasks tapping executive functioning when compared with the levels of dopamine released while at rest. There was no such difference in dopamine release between executive functioning tasks and rest for older adults.

Summary

The two cognitive models of depression-related memory impairment are the resource allocation model and the inhibitory control model. The resource allocation model proposes that ruminative thoughts occupy attentional resources in depression, leaving less attentional capacity for other cognitive tasks. This model has been supported by neurobiological research. The inhibitory control model was first proposed to account for age-related working memory decline. Its main premise is that the inhibitory functions of the central executive in working memory become increasingly inefficient with age, and attention can become more easily distracted away from memory. The inhibitory control model can also extend to prospective memory decline in ageing due to the large attentional component. The inhibitory control model has been applied to depression and working memory difficulties. In depression, it is thought that the inhibitory control difficulties lie predominantly in inhibiting ruminative and negative thoughts. Difficulties with inhibition have been found to be compounded by depression in older adults, and this relationship has also been supported by neurobiological studies.

Of significance to late-life depression are changes that occur in the hippocampus. People with depression have been found to have changes in hippocampal volume, and this in turn has been associated with poorer memory performance. HPA axis dysfunction and over exposure to cortisol in depression has been theorised as responsible for some of this hippocampal atrophy, possibly through a reduction in hippocampal neurogenesis. As well as the hippocampus, the frontal and pre-frontal cortices are affected by ageing and depression, with participants with late-life depression being shown to have small frontal cortex volumes. WMH may be behind some of the frontal cortex changes. WMH are associated with late-life depression and cognitive dysfunction, and often occur in the frontal lobes. Monoamine functions are reduced in ageing and depression. Reduced serotonin and dopamine

functioning has been linked with difficulties in attention, memory, and executive functioning.

Chapter Four

The Present Study

This chapter presents the rationale for the study. The influences of moderating factors on both depression and memory are considered. The aims of the study are outlined and a brief overview of the literature already reviewed is provided to support these expectations.

Rationale and Importance of Study

Depression has been shown to have a substantial impact on memory functioning across different age groups. Because some types of memory are known to deteriorate with age, older adults are at a distinct disadvantage if late-life depression also decreases memory functioning. If an older adult's memory capacity has already been reduced due to the ageing process, they will have much less to rely on. If depression then adds to that strain, problems in memory may then become apparent in their daily lives. Types of memory that are particularly important in daily activities are prospective memory and working memory.

The significance of the present research is that it will contribute to the growing literature on the cognitive profile of late-life depression. If a person is showing declines in memory functioning, this has a noticeable impact on their activities of daily living. Some specific types of memory have already been found to be affected by late-life depression and it is common for an affected person to feel that their memory has deteriorated. The present study will add more detail to the understanding of those areas of memory that have already been researched and will also add new information on prospective memory to the knowledge pool.

The present study is quasi-experimental in design. It examines short-term memory in the form of a verbal list recall task, working memory in the form of reading span and letter-number sequencing tasks, and prospective memory using a number of time-based and event-based tasks. The emphasis is on how depressive symptoms in older adults relate to memory scores.

Known Moderators for Depression and Memory

Depression and memory difficulties do not usually occur in isolation. As a result, it was deemed important that the factors known to have strong relationships with memory and depression be taken into account. The moderators of interest to this study are anxiety and stress as related to depression, and processing speed and intelligence because of their relationships with memory. The rationale for accounting for these moderators is outlined below.

Factors Associated with Depression

Anxiety

Depression does not always occur in isolation; it is often comorbid with an anxiety disorder. It has been estimated that anxiety disorders occur comorbidly in approximately 50 % of people with a mood disorder (Fava et al., 2000). Similar comorbidity has been found in older adult samples, with almost half of older adults with major depression also having an anxiety disorder, and just over a quarter of those with an anxiety disorder also experiencing a depressive disorder (Beekman et al., 2000). In New Zealand, the 12-month prevalence for an anxiety disorder in those aged 65 years and older is six percent (Oakley Browne et al., 2006), although this does not include anxious older adults who do not meet criteria for a clinical anxiety disorder. Research suggests that older adults experience anxiety differently to younger adults. In particular, worry appears to be more prevalent in younger adults than in older adults (Brenes, 2006).

Anxiety and depression often occur comorbidly, and there has been much discussion around the relationship between these two groups of symptoms. The high comorbidity has led some researchers and clinicians to investigate the concept of Mixed Anxiety-Depression (MAD). MAD was suggested as a possible diagnostic category for those with subclinical symptoms of both depression and anxiety that were impacting on quality of life but did not meet criteria for a mood or anxiety disorder diagnosis on their own (Barlow & Campbell, 2000). After reviewing a number of different frameworks to explain the high comorbidity of depression and anxiety, including the two disorder groups being on a continuum, having a shared aetiology, and being distinct categorical entities, Clark and Watson (1991) developed their tripartite model. They proposed that anxiety and depression share an element of general distress while anxiety is separated from depression by physiological arousal, and depression separated from anxiety by a sense of anhedonia.

So far, much of the research on depression and cognition has failed to acknowledge that anxiety may have a role to play by not discussing or measuring it. Given the high comorbidity between depression and anxiety, not controlling for anxiety seems like an oversight. Beuke, Fischer, and McDowell (2003) noted that research focussing on depression should always take anxiety into account (and vice versa) for several reasons. Firstly, they propose that it is much more efficient to study both depression and anxiety together under the same experimental design. This makes for easier comparison when both have been studied in the same participant pool under identical conditions. Secondly, Beuke et al. state that although there are strong associations between depression and anxiety, it is still necessary to study them as distinct concepts as they often have differing effects. The outcomes of research on depression and memory cannot simply be assumed to apply to anxiety. Thirdly, Beuke et al. note that ignoring either depression or anxiety in research on the other can lead to ambiguous results. As we already know that comorbidity between anxiety and

depression is high, any results obtained without knowledge of both depression and anxiety would be confounded.

Anxiety has been found to exert different effects on cognitive functioning. The Longitudinal Aging Study Amsterdam assessed anxiety and cognitive functioning in a sample of older adults (aged 55-85), using the Hospital Anxiety and Depression Scale (HADS), the Mini Mental State Examination (MMSE), and the Auditory Verbal Learning Test (Bierman, Comijs, Rijmen, Jonker, & Beekman, 2008). A curvilinear relationship was found between anxiety symptoms and cognitive functioning. At first, increasing anxiety symptomatology was associated with better performance on the two cognitive tests. However, the authors noted that cognitive performance started to decline once participants scored above 14 points on the HADS. Such curvilinear relationships for depression and memory have not been reported.

Further research into the relationships between anxiety and cognitive functions was conducted by Beaudreau and O'Hara (2009). In their sample of community-dwelling older adults, anxiety was significantly related to difficulties in inhibition, processing speed/shifting attention, but not to episodic memory. In the same sample, there was a trend towards a relationship between depression and episodic memory ($R^2 = .06$, $p = .059$). Those with comorbid anxiety and depressive symptoms were found to have difficulty with semantic and episodic memory, as well as processing speed and shifting attention. The impairment in inhibition, processing speed, and shifting attention is important given their known roles in memory, especially in working memory. The Beaudreau and O'Hara study is somewhat limited by its use of the Beck Anxiety Inventory which covers the physiological symptoms of anxiety well, but has less of a focus on the cognitive symptoms of anxiety.

Impairment in inhibition, shifting attention, and working memory with increasing anxiety has been noted in a number of studies (Beaudreau & O'Hara, 2009; Elliman, Green, Rogers, & Finch, 1997). Eysenck and his colleagues have

also spent considerable time researching anxiety. This research has led to the development of the Attentional Control Theory, in which anxiety diverts attention to threatening stimuli and away from the task at hand (Eysenck, Derakshan, Santos, & Calvo, 2007). The Attentional Control Theory appears similar to both the Inhibitory Control Model and the Resource Allocation Model of depression. A common element to both anxiety and depression is stress.

Stress

Because of the strong relationship often found between depression and stress, the importance of stress is effectively interwoven in any discussion of depression. For example, in the stress-diathesis model of depression, stressors interact with a person's cognitive style leading to depression. This strong relationship between stress and depression continues into later life. A meta-analysis of studies investigating stressors and late-life depression found that repetitive daily stressors in particular had a significant relationship with depression (Kraaij, Arensman, & Spinhoven, 2002).

Of particular importance to the present study is the HPA axis dysfunction. Cortisol is released during stress, and the feedback loop to suppress the release is thought to be impaired in depression. This means cortisol is released in a chronic manner, and over time it damages the brain structures involved with memory and emotion regulation.

In a review of their research in The Douglas Hospital Longitudinal Study of Normal and Pathological Aging, Lupien et al. (2005), investigating the relationships among cortisol, cognition, and ageing over a 3 to 6 year period, found that elevated cortisol levels were related to impaired declarative memory performance. They also found hippocampal volume loss of 14 percent in a subsample of those in the high cortisol group.

Summary

Given the strong relationships among anxiety, stress, and depression, it was thought necessary to take anxiety and stress levels into account in the present research. All three variables have been found to make their own unique contribution to cognitive decline in samples of older adults. The majority of research into depression and memory does not account for the effects of anxiety or stress. This seems like an oversight, given the reasonably strong relationships among these variables.

Factors Associated with Memory

Processing Speed

Salthouse has conducted considerable research into the relationship between working memory and processing speed, and has identified that processing speed mediates the relationship between increasing age and decreasing working memory ability (Salthouse, 1991, 1992, 1994). He also proposes that processing speed is related to the encoding of the task information, rather than deterioration of the memory trace (Salthouse, 1992, 1994). Salthouse argues that a slowing of processing speed is the mechanism underlying cognitive change with ageing, including that of working memory ability. He suggests that cognitive functions are limited by a person's general processing capacity, and that this processing capacity declines with age (Salthouse, 1996). While it is believed that processing speed mediates the relationship between age and working memory ability, it does not account for all of the age-related decline (Rush, Barch, & Braver, 2006; Salthouse, 2000).

Working memory also has a strong relationship with intelligence. This relationship appears to be tied to the central executive component of working memory, as short-term memory capacity explains a minimal amount of the variance in the relationship when compared with working memory (Conway, Cowan, Bunting, Therriault, & Minkoff, 2002; Engle, Tuholski, Laughlin, &

Conway, 1999). The updating process in working memory has been found to be compromised in older adults, and is related to worse performance on tests of fluid intelligence (Chen & Li, 2007). Using structural equation modelling, they found that updating working memory mediated the relationship between age and fluid intelligence. They also found once the updating component was taken into account, processing speed no longer significantly predicted performance on the fluid intelligence tests (standardised coefficients ranged between .75 to .81 for the processing speed tasks of motor speed, reaction time, and inspection time).

Intelligence

Many models have been developed in an attempt to understand intelligence. One such model is the idea of fluid and crystallised intelligence as proposed by Horn and Catell (1966). Fluid intelligence can be conceptualised as the inherent mental ability that a person poses, while crystallised intelligence represents information learned over time. The majority of research investigating memory and intelligence focuses on fluid intelligence.

Working memory in particular has been a strong focus of research investigating intelligence and memory relationships. Working memory and long-term episodic memory were found to have moderate and significant positive relationships with fluid intelligence in younger adults (Unsworth, 2010). Similar relationships between working memory and intelligence have been found in numerous other studies (see Conway, Kane, & Engle, 2003, for a review). It has even been posited that practicing working memory tasks can lead to improved fluid intelligence (Jaeggi, Buschkuhl, Jonides, & Perrig, 2008). Interestingly, the updating component of working memory is thought to mediate age-related declines in fluid intelligence (Chen & Li, 2007).

Research has also investigated the relationship between prospective memory and intelligence. In a sample of adults aged 18-89 years, Salthouse, Berish, and Siedlecki (2004) found a significant relationship between a prospective memory

composite and a fluid intelligence composite (the composites being artificial scores created from totalling the scores of several different tasks or tests). When the sample was divided into younger and older adults (older adults 50-89 years), the relationship was stronger in the older adult sample.

Summary

It is clear that processing speed, intelligence, working memory, and prospective memory are all linked. Given the high degree of shared variance of working memory and processing speed in ageing, as well as the relationship between working memory and intelligence, it is important that research into memory takes these related variables into account.

Research Aims

The aims of the current research are presented by memory type.

Short-Term Memory

STM shows a small but significant decline in capacity with age. STM findings in depression are more inconsistent than those for age, with considerable inter-study variability. While some studies have found that STM is affected by depression, others have found no effects. Looking at different types of depression, such as major depression versus dysthymia, has revealed that STM is generally more affected in major depression when compared with those with dysthymia, as well as with healthy controls. The inconsistency in results continues for older adults with depression. Some studies have found greater STM impairment with younger depressed adults, while other studies have found the reverse pattern. However, a meta-analysis indicated that STM is affected by depression in later life (Kindermann & Brown, 1997). In community-based studies (as the present study will be), the results are mixed. One of the difficulties is that in other community-based studies, STM is invariably assessed as part of a composite (such as in the Wechsler Memory Scale or in tests of learning such as the RAVLT) and information on STM only is

often not provided. Given that studies have found greater impairment for delayed rather than immediate recall (STM), the memory and depression relationship seen in some of these studies may stem from long-term memory difficulties.

The aim of investigating STM in the present study was to provide further clarification on potential STM impairment and depressive symptoms in a community-based sample of older adults compared to a younger group. It was predicted there would be a main effect for Depression on the recall task, with those in the low depression groups having superior performance over those in the high depression groups.

Working Memory

Working memory has age-related impairment over and above that of STM. These impairments seem to lie predominantly in the central executive, with inhibition and updating functions affected. In depression, the central executive is also affected. Again, this impairment seems to lie with inhibitory functions, with difficulties inhibiting ruminative thoughts distracting attention away from memory. This pattern extends to depression in later life as well.

The aim of investigating working memory in the present study was to look at the impact of depression on two different tasks of working memory. It was expected there would be a main effect for Depression in both tasks, with those in the high depression groups showing poorer working memory performance. It was also expected that there would be an Age effect, with older adults in the low and high depression groups having lower scores than their younger counterparts.

Prospective Memory

Older adults tend to outperform their younger counterparts in naturalistic studies of prospective memory (Henry et al., 2004). In lab-based studies, the reverse has been found with older adults generally showing poorer

performance. While there are some inconsistencies in the ageing research, there is a trend towards greater age-related difficulty with time-based over event-based tasks. Depression has also been shown to have an impact on time-based prospective memory, but not on event-based memory. Only one previous study investigating late-life depression and prospective memory was found. It showed that there was no relationship between event-based prospective memory and level of depressive symptomatology.

The aim of investigating prospective memory in the present study was to look at time-based and event-based tasks in the same study, as there is a lack of such research in depression and older adults. Yet there is theoretical support for time-based tasks to be affected to a greater extent. It was expected there would be a main effect for Depression in the time-based prospective memory tasks, but not the event-based task, with those in the high depression group exhibiting poorer task performance. It was expected there would be an effect for Age in the time-based task, and that the older adults' performance across the low and high depression groups would reflect the pattern of performance of the younger adults.

In summary, the hypotheses of the present study were that:

1. There would be a main effect for Depression on the recall task, with those in the low depression groups outperforming those in the high depression groups for each age group.
2. There would be main effects for Depression on both working memory tasks, again with the low depression groups outperforming the high depression groups.
3. It was hypothesised there would a main effect for Depression with the time-based prospective memory task, but not for the event-based prospective memory task. It was expected that those in the low depression groups would outperform those in the high depression groups on the time-based prospective memory task.

4. It was expected there would be a main effect for Age for each of the memory tasks.

Chapter Five

Method

The following chapter outlines the basic characteristics of the sample, the experimental design and statistical analysis plan, and the procedure for recruitment and testing, including the ethical considerations of studying a population in which participants with symptoms of depression were targeted. An overview of the measures used and their psychometric properties is also provided.

Participants

One hundred community dwelling adults participated in this study, with 50 adults in each of two age groups of 20-29 years and 70-79 years. Potential participants were recruited from Massey University and the general Manawatu community. The younger adults included a mix of students and professionals. The students in the sample were predominantly undergraduate psychology students although other colleges of study were also included. The young professionals were generally staff from either the university or from the local hospital and included a range of allied health vocations (but not Clinical Psychologists or students accepted into Clinical Psychology training programmes). The older adults generally consisted of community members involved in volunteer organisations or in groups providing social networking opportunities for retirees. Many of the older adults had been professionals prior to retirement. The exclusion criteria included familiarity with the tests being used to measure the variables of interest, a diagnosis of any form of neurodegenerative disease, a history of traumatic brain injury or stroke, the use of psychotropic medications, and a history of mental illness other than a depressive or anxiety disorder. Mild cognitive impairment was also screened through the use of the MMSE. Those who scored below the suggested MMSE

cut-off of 23 were to be excluded from the study. No one was excluded due to their MMSE result, with the lowest score being 25. However, it was deemed inappropriate to complete the MMSE with five participants from the younger age group due to their own extensive experience with administering the test themselves. The main demographic characteristics of the sample are presented in Table 1. There was no significant difference in MMSE scores between the two age groups when using an independent-samples *t*-test; $t(95) = 1.83, p = .07$ (two-tailed).

Table 1

Mean (standard deviation) age and MMSE scores for all participants

	Young			Older		
	Female n = 38	Male n = 12	Total n = 50	Female n = 36	Male n = 14	Total n = 50
Age	23.05 (2.88)	22.00 (2.99)	22.80 (2.91)	73.11 (9.47)	74.79 (2.69)	73.58 (8.16)
MMSE	29.09 (1.03)	29.00 (1.61)	29.07 (1.18)	28.61 (1.38)	28.57 (1.09)	28.60 (1.29)

The sample size of 100 participants (50 participants for each age group) provided sufficient power to detect a genuine medium effect. This was calculated using an effect size of $d = 0.58$, power of 80%, and $p < 0.05$. This effect size was the smallest effect calculated in a meta-analysis on depression and memory in older adults by Kindermann and Brown (1997).

Experimental Design and Analysis

The study used a quasi-experimental design that was cross-sectional in nature, in which level of depression was compared with age and achievement in three different types of memory test. There were two independent variables, depressive symptoms and age. The dependent variables were prospective memory, working memory, and verbal recall. A number of moderators were assessed and they were anxiety, stress, processing speed, and intelligence.

While covariates would normally be accounted for statistically through the use of ANCOVA, Lord (1967) states that ANCOVA should not be used in studies where the groups are not randomly assigned. As neither independent variable in the present study, depression and age, could be randomly assigned, the use of ANCOVA was inappropriate. This is because ANCOVA assumes that groups come from the same population, and applies a correction to factor in regression to the mean. This correction can then mask some real effects or enhance others, depending on the direction of group differences (Jamieson, 1999).

Because ANCOVA could not be used, it was decided that separate ANOVA analyses would be run, treating the moderators as factors along with Depression and Age. A series of two-way and three-way ANOVAs were run, with each factor only having two levels (high and low). The high and low groups were decided based on median splits for each of the factors except for Age, in which those in their twenties comprised the younger adults group, while those in their seventies comprised the older adults group.

Procedure

Ethical Process and Safety Plan

Given the nature of the study, it was expected that participants would have a wide range of experience with depression, including currently experiencing depressive symptoms at a level that could indicate a depressive disorder. A plan was established to deal with such eventualities prior to recruitment of participants. Participants were advised in the Information Sheet (Appendix B) that a list of support services and psychologists based in the community was available upon request. They were also advised in the Information Sheet that if there were serious concerns regarding a participant's safety, or the safety of others, that the researcher would discuss this with a registered clinical psychologist from within the School of Psychology. The plan in the first

instance was to discuss such cases with the senior clinical psychologist on the supervision team, and only approach a clinical psychologist from outside this team if the primary clinical supervisor was unavailable. After discussion with the clinical psychologist, it would be decided if a referral to the participant's general practitioner would be appropriate, and to arrange this if necessary.

The Center for Epidemiologic Studies Depression Scale (CES-D) was scored while participants were still present. If participants scored above the cut-off of 16, a discussion of depressive symptomatology took place, including screening for self-harm and suicidal ideation. The plan for participants expressing self-harm or suicidal ideation is discussed above. Participants who scored above the cut-off but who did not report any thoughts of self-harm or suicidal ideation were encouraged to contact their general practitioner, and were provided with the details of an appropriate community support service. Ten participants scored above the cut-off of 16. Of these, two of the younger participants expressed having had some suicidal ideation (but reported that they did not have any plans or intention to act on these thoughts). The above safety plan was enacted and the situations were discussed with the supervising clinical psychologist. In both cases, the participants were advised to make an appointment with their General Practitioners and were also referred to youth counselling and psychology services which they were able to access free of charge. All other participants scoring above the cut-off were advised to discuss their symptoms with their General Practitioners and were given a contact list of community services that might have been of help.

Recruitment

Potential participants were recruited from a number of different sources. To recruit older adults specifically, community organisations such as Grey Power, the Senior Citizens Association, and local Probus groups in the Palmerston North area were approached and presentations were given to members asking for volunteers.

Younger adults were recruited by attending undergraduate psychology lectures to explain the study briefly and to ask for volunteers. They were also recruited through a staff email forum at the Palmerston North Hospital, in which an email with a description of the study and an attached information sheet was available to staff members checking the forum.

To recruit for both age groups, Colour posters (Appendix C) were displayed on community notice boards in the Palmerston North City Library, General Medical Practices within Palmerston North, and at multiple sites around the Massey University Turitea campus. Age Concern distributed a black and white copy of the poster with their newsletter. Participants were also invited to encourage family and friends to volunteer.

Once potential participants made contact via telephone or email, a phone call or email conversation was had with each person to discuss the study and offer the opportunity to ask questions. A copy of the information sheet, screening questionnaire, and consent form (Appendices B, D, and E, respectively) were sent out. When these were received contact was again made to the potential participant to either arrange a time for them to take part in the testing, or to inform them that they had not met criteria for entry into the study.

Assessment

Participants were given the choice between the assessment taking place in their own home, or in an office within the School of Psychology at Massey University. The only requirement for seeing participants in their own home was that they were able to provide an environment free from distractions with a suitable writing surface, such as a dining room table.

On arrival, participants were welcomed and introduced to the researcher. A standardised overview of the schedule of activities was provided. Participants were given their compensatory voucher (Whitcoull's book voucher) and were reminded they had the right to withdraw from any task, or to withdraw from

the study completely, at any time during the activities. Participants were provided with the opportunity to ask questions about the study. They were also advised about the confidentiality of their information, and the ethical process that would need to be followed if there were concerns about their level of depressive symptomatology (as described above).

Participants were randomly assigned to one of two test orders. As the test administrator was not blind to the research hypotheses, it was deemed prudent to counter-balance the order in which the tests of depression, anxiety, and stress were administered with the administration of the cognitive tasks. Once it was established whether the participants would be presented with cognitive tests or the depression, anxiety and stress questionnaires first, the order in which the tests within those groups were administered were randomly assigned. The standardised instructions provided by test authors were used in the administration of the tasks. All tasks were completed in paper and pencil format. Testing took approximately two hours for each participant and was conducted individually. A break of 10 minutes occurred after the first hour.

Data Management

Data were kept in a filing cabinet in a locked laboratory in the School of Psychology. There were no names kept on the test forms and each participant's results were only identifiable by their participant number (known only to the researcher). The CES-D was scored with the participant present so that any concerns regarding depressive symptomatology endorsed on the test could be discussed with the participant at the time of the assessment. The DASS and MMSE were also scored on the same day so that any concerns regarding anxiety or cognitive impairment could also be addressed quickly with the participant. The CES-D, DASS and MMSE were then kept separate from the cognitive tests so that the results would not bias the scoring of the cognitive tests.

Measures

Depression, Anxiety, and Stress

The Center for Epidemiologic Studies Depression Scale (CES-D)

The CES-D (Radloff, 1977) is a 20-item self-report scale designed to measure depressive symptoms in a nonclinical population. The scale requires respondents to rate how much their past week has been affected by different symptoms of depression on a 4-point Likert scale. Some of the items are framed in a positive manner and are reverse scored. These items include such statements as “You were happy” and “You felt hopeful about the future”. The scores range from zero to 60. One caution when using the CES-D is that it measures the level of depressive symptoms and should not be used to infer severity of illness or clinical diagnosis (Radloff, 1977).

The CES-D is a valid screen for depression with high internal consistency (ranging between $\alpha = 0.82$ to 0.90 across samples) and reasonable test-retest reliabilities ($r = 0.51$ to 0.67) across periods of up to eight weeks (Lewinsohn, Seeley, Roberts, & Allen, 1997; Radloff, 1977).

Lewinsohn et al. (1997) propose that the CES-D is an appropriate screening tool for depression in older populations. Their study advocated the use of the CES-D with older adults after they found that factors such as social desirability, physical impairment or disease, and cognitive impairment did not significantly impinge on the efficacy of the CES-D.

Research on the criterion validity of the CES-D in the elderly has found that it has good sensitivity and specificity for identifying the presence or absence of depression. At the conventional cut off score of 16 and above, the CES-D had a sensitivity of 100% and a specificity of 88% for detecting respondents who had experienced MDD within the previous month (Beekman et al., 1997). Given that the specificity is the rate of true negatives, and sensitivity is the rate of true

positives, the CES-D is likely to accurately detect participants with significant symptoms of depression at the expense of incorrectly identifying a small percentage of participants as having significant depressive symptoms. Further research on the sensitivity and specificity of the CES-D with older adults also supports the validity of the scale in this population. A study by Haringsma, Engels, Beekman, and Spinhoven (2004) also found the CES-D had good sensitivity and specificity for identifying cases of MDD and clinically relevant depression, although they found a higher cut off score of 25 was needed.

The Depression Anxiety Stress Scales (DASS)

The DASS is a 42-item self-report test composed of the three scales of Depression, Anxiety, and Stress. Each of the three scales contains 14 questions that target experiences over the past week. The DASS was developed with the intention that it would be able to differentiate between depression and anxiety (S. Lovibond & Lovibond, 1993). With this aim in mind, the developers included questions pertaining to key features of clinical depression and anxiety, but did not include features that were common to both depression and anxiety (S. Lovibond & Lovibond, 1993).

While the DASS contains a 14-item depression scale, it was decided only the CES-D would be used to provide the depression ratings for the analysis. The rationale for this was that the CES-D is a more comprehensive assessment of depression that is likely to produce a greater range of scores. Also, the DASS is relatively new to research as a measure of depression and there is more support for the CES-D with older populations. Several other studies on depression and memory have used the CES-D and this allows for better comparison but those studies and the results of the present study.

The DASS has high internal consistency (Cronbach's alpha), with reports ranging from 0.89 to 0.97 for the individual scales (Antony, Bieling, Cox, Enns, & Swinson, 1998; T. A. Brown, Chorpita, Korotitsch, & Barlow, 1997; Crawford & Henry, 2003). Studies of the factor structure of the DASS also lend weight to

its reliability. Through a number of studies, a three-factor structure representing each of the three scales of Depression, Anxiety, and Stress has become clear (Antony et al., 1998; T. A. Brown et al., 1997; P. Lovibond & Lovibond, 1995). In a sample of both clinical and nonclinical populations, Antony et al. (1998) found very clear distinctions between the three scales. In their study, only items on the depression scale loaded on the depression factor. There was more cross-over with the Stress and Anxiety scales, as each of these scales had two items that also loaded onto the other factor.

The concurrent and discriminant validity of the DASS is good when compared to the Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI), and the State-Trait Anxiety Inventory – Trait Scale (STAI-T). In the previously mentioned study conducted by Antony et al. (1998), the DASS-A had a correlation of .84 with the BAI and .44 with the STAI-T. The lower correlation between the DASS-A and the STAI-T may be due to the propensity of the STAI to measure aspects of depression as well as anxiety (Bieling, Antony, & Swinson, 1998). The DASS-D correlated $r = .42$ with the BAI, but $.71$ with the STAI-T. This lends further support to the idea that the STAI-T is measuring some symptoms that are consistent with depression. The discriminant validity of the DASS is also supported in the study by Antony et al. (1998) in which it was found that the DASS-A (DASS anxiety scale) and DASS-D (DASS depression scale) had a correlation of $r = .44$. In contrast the BAI and BDI have correlations ranging from $r = .48$ to $.63$ (Beck, Epstein, Brown, & Steer, 1988; Fydrich, Dowdall, & Chambless, 1992; Hewitt & Norton, 1993). This, combined with the factor structure of the DASS, indicates that the depression and anxiety scales are assessing these two constructs with less mutual interference than is found between the BAI and the BDI.

There are no data currently available on the use of the DASS with older adults; however, recently, the psychometric properties of the DASS-21 (short form of the DASS) have been investigated in a sample of older adults by Gloster et al. (2008). Their sample consisted of adults aged 60 years and above, who had

been referred to a cognitive behavioural therapy clinic for worry and generalised anxiety disorder. Gloster et al. found that the factor structure uncovered by Lovibond and Lovibond (1995), discussed above, was still the most appropriate solution in their older sample. They also found that the DASS-21 still had high internal consistency ($\rho = .94$). Most importantly, each of the DASS-21 scales had the strongest correlations with other instruments designed to measure the corresponding construct. The DASS21-D had a correlation of $r = .76$ with the BDI-II and only $r = .51$ with the BAI, whereas the DASS21-A had a correlation of $r = .74$ with the BAI and $r = .47$ with the BDI-II (Gloster et al., 2008). The authors concluded that the DASS is a measure that is appropriate for use in an older population, although results from the DASS-A should be interpreted with caution due to the unavoidable number of somatic items needed to assess anxiety (Gloster et al., 2008).

Cognition

The Mini-Mental State Examination (MMSE)

The MMSE (Folstein, Folstein, McHugh, & Fanjiang, 2001) is a screening tool designed to detect cognitive impairment and to track deterioration over time. It consists of 11 questions which cover five basic cognitive domains – orientation, registration, attention and calculation, recall, and language. The maximum score that can be obtained is 30. It is important to note that a score below the cut off point is not diagnostic of any disorder and should only be used to indicate that some cognitive impairment is present, and that further assessment of the apparent deficits would be valuable (Folstein et al., 2001).

The MMSE has adequate internal consistency and test-retest reliability (Strauss, Sherman, & Spreen, 2006; Tombaugh, 2005). It has a moderate correlation with the Clock Drawing Task, which indicates construct validity (Adunsky, Fleissig, Levenkrohn, Arad, & Noy, 2002). The MMSE also has high sensitivity and specificity percentages. Using the traditional cut-off score of 23 or lower, the

MMSE has sensitivity of 86% and specificity of 92% in detecting either dementia or delirium in older adults (O'Connor et al., 1989).

The Wechsler Adult Intelligence Scale (3rd edition; WAIS-III)

Subtests of the Wechsler Adult Intelligence Test (The Psychological Corporation, 1997) were used to estimate intelligence, and also as a measure of working memory. Short versions of the WAIS-III can be used to provide an estimate of the WAIS-III Full Scale Intelligence Quotient (FSIQ) for research and screening purposes (Clara & Huynh, 2003; Jeyakumar, Warriner, Raval, & Ahmad, 2004; Ringe, Saine, Lacritz, Hynan, & Cullum, 2002). Because the administration of the full WAIS-III protocol can take up to 90 minutes, shorter forms of intelligence tests are recommended when working with older adults due to the impact of fatigue (Wymer, Rayls, & Wagner, 2003).

The Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999) was developed to fulfil the need for an equivalent short form to the WAIS-III and either a two subtest version or a four subtest version can be used to derive an estimate of FSIQ. The two subtest version uses Vocabulary and Matrix Reasoning subtests, while the four subtest version uses Vocabulary, Similarities, Matrix reasoning, and Block Design. In a comparison of the WASI and the matching subtests of the WAIS-III, the WAIS-III subtests obtained higher correlations with full WAIS-III scores than the WASI across Verbal Intelligence Quotient (VIQ), Performance Intelligence Quotient (PIQ) estimates, and also FSIQ estimates for the two and four subtest forms (Axelrod, 2002). However, this correlation was in part due to the fact the same scores from the subtests used in the short forms of the WAIS-III were also used to compute the FSIQ. As previously mentioned, the two subtest short form of the WASI consisted of the Vocabulary and Matrix reasoning subtests, but it has since been found that correlations between estimated and actual FSIQ scores are higher when the Vocabulary and Block Design subtests are used. Jeyakumar et al. (2004) report good psychometric properties when using the Vocabulary and

Block Design subtests, with internal consistency estimates of $r = .90$ to $.94$ for the age groups used in the present study. This particular dyad of subtests also has strong validity with corrected part-whole correlations of $.85$ to $.90$ (when compared with the WAIS-III) in the age ranges of participants in this present study (Jeyakumar et al., 2004).

Processing Speed

Kit of Factor-Referenced Cognitive Tests

The Kit of Factor-Referenced Cognitive Tests (KFRCT; (Ekstrom, French, Harman, & Derman, 1976) is, as the name suggests, a selection of tests designed to measure varying aspects of cognition. There are 72 different tests in total, with each of these tests belonging to one of 23 cognitive factors including the likes of spatial orientation, verbal comprehension, and word fluency. Processing speed is also one of these factors and the KFRCT has several tests that load onto this factor. The two KFRCT processing speed tests used in this study were the Identical Pictures Test and the Finding A's test.

The Identical Pictures Test requires the respondent to match a stimulus picture to one of five options next to it. There are many trials using different stimulus pictures and the object is to correctly mark as many of the matching pictures as possible in 1.5 minutes.

The Finding A's Test requires respondents to scan several pages containing word lists and cross out all of the words that contain an "a". There are five columns of words to a page, with each of these five columns having 41 words. Each column contains five words that contain the letter "a" and this is outlined in the instructions. As with the Identical Pictures Test, this is a timed trial and participants have 2 minutes to find as many words containing an "a" as possible.

The processing speed tests from the KFRCT are well established in the literature and have been used in extensive studies of ageing including the Seattle

Longitudinal Study and the Berlin Aging Study (Lövdén, Li, Shing, & Lindenberger, 2007; Schaie, 1989; Schaie, Willis, Hertzog, & Schulenberg, 1987). Potential factors and their tests were accepted into the KFRCT only after they had been found in three factor analyses conducted either by at least two different researchers, or in two different laboratories (Ekstrom et al., 1976).

Short-Term Memory

Recall List

A recall list was developed specifically for the purposes of this research. The development of the list focussed on the everyday aspects of this study and was created to represent a shopping list (Table 2). A small pilot study comparing the shopping list to list A from the RAVLT was conducted with 17 pilot participants. They ranged in age from 20 – 32 years with seven males and ten females. The correlation between the two tasks was moderate at $r = .48$. Some difference in scores was expected given that the two lists differed in subject matter. The shopping list task created for this study used items that could be bought from a supermarket, while items on the RAVLT come from a variety of semantic categories. A pool of words with varying written frequency scores was created, and then a random list generator was used to narrow the pool down to a list of 15 words. The higher the Kucera-Francis Written Frequency (KFWF), the more common the word is in the written English language.

Table 2

Recall list with associated score of Kucera-Francis written frequency (KFWF)

Word	KFWF	Word	KFWF	Word	KFWF
butter	27	pepper	13	wine	72
honey	25	milk	49	broccoli	1
aspirin	3	rice	33	rosemary	1
soap	22	cereal	17	cheese	9
ham	19	salmon	3	bread	49

Working Memory

The Reading Span Task

The Reading Span Task (RST) was developed by Daneman and Carpenter (1980) with the intention of measuring processing and storage in working memory. The reading span task used in the present study is a more recent version of the original, as developed by Daneman and Hannon (2001). The task involves participants reading sets of sentences and having to make a decision as to whether or not the sentence makes sense. Each time they read a sentence, the participants say “yes” or “no” depending on whether or not each sentence makes sense, and then they go on to read the next sentence in the trial. The sentences vary in length from 8-12 words and are presented in sets of 2, 3, 4, 5 and 6 sentences at a time. Once the participant had read all of the sentences in that trial, and had responded “yes” or “no” for their decision regarding the sense of the sentence, they were required to try and recall the last word of each sentence in the order that they read the sentences. Three trials in each set length were used. A copy of the sentence sets is located in Appendix F.

No psychometric data have been reported for this particular version of the RST; however, Waters and Caplan (2003) have investigated the psychometric properties of several popular versions of the RST. Both the original RST by Daneman and Carpenter (1980) and the version of the RST closest to that used in the present study (developed by Waters, Caplan, and Hildebrandt, 1987) have satisfactory reliability with internal consistency scores of .78 to .82 and .92 to .95, respectively (Waters & Caplan, 1996, 2003).

Letter-Number Sequencing Task

The letter-number sequencing task from the WAIS-III was also used as a measure of working memory. The task required participants to hold a string of disorganised letters and numbers in memory, and to sort them alpha-numerically. The strings were read aloud to participants using the

standardised WAIS-III instructions. The strings are read at the rate of one item per second. The letter-number strings start with two items per string and gradually increase by one item at a time until the strings are eight items long at the final level. Each level has three trials using different combinations, and scores range from 0 – 3 for each level. Once a participant has obtained a score of 0 for a complete level, the task is stopped.

The letter-number sequencing task was designed to be a measure of auditory working memory and attention (The Psychological Corporation, 1997). The letter-number sequencing task was standardised with 1,250 adults aged from 16-89 years. The reliability of the task was established using an odd-even split-half method. The reliability coefficients were $r = .77$, $r = .88$, and $r = .75$ for the age groups of 20-29, 70-74, and 75-79, respectively. Test-retest reliability (over a mean of 34.6 days) was $r = .70$ and $r = .80$ for the younger and older adults, respectively.

Prospective memory

The Cambridge Prospective Memory Test

The Cambridge Prospective Memory Test (CAMPROMPT; Wilson et al., 2005) is a pencil and paper test of prospective memory. It was designed to be an ecologically valid measure of prospective memory that reflected real-life tasks (Spooner & Pachana, 2006; B. A. Wilson et al., 2005). It includes three time-based prospective memory tasks, and three event-based prospective memory tasks, taking approximately 25 minutes in total to complete. During this time, the participant engages in background distracter tasks that accompany the test while awaiting the cues for the prospective memory tasks to occur. The time-based tasks consisted of having to remind the researcher of an appointment at a particular time, and engaging in two other tasks at particular points of time (such as changing pens) based on the countdown timer available. The event-based tasks required the participants to perform actions after cues within the test itself such as passing the researcher an object when coming to a particular

question in the general knowledge quiz. Each of the six subtests has a possible score of 6 points, giving a score range of 0-36 points. Only a total score is provided by the test – scores are not broken down into subscales for the time-based or event-based tasks.

The CAMPROMPT has been administered to a normative sample of 237 individuals 16-92 years of age, divided into four age groups. The oldest group begins at age 66, and also is divided into three different levels of intelligence based on estimates obtained from the National Adult Reading Test.

Reliability of the CAMPROMPT has been found to be satisfactory with an interrater reliability estimated at .998 and a test-retest reliability of .64 (B. A. Wilson et al., 2005). A small practice effect was noted. No data on internal consistency are reported in the CAMPROMPT manual. B. A. Wilson et al. (2005) reported adequate concurrent validity for the event-based tasks when compared with the Rivermead Behavioural Memory Test (RBMT). While there is no significant correlation between the time-based tasks of the CAMPROMPT and the RBMT, the test authors propose that this elevates the validity of the CAMPROMPT as the RBMT does not contain time-based prospective memory tasks.

Chapter Six

Results

This chapter presents the statistical findings of the study. Firstly, the planned comparisons are presented, in which two-way ANOVAs between Depression and Age, and their effects on the different memory tasks are presented. Secondly, an analysis is undertaken where the impact of Depression and Age on memory are explored with Visual and Verbal Processing Speeds, and Intelligence. Thirdly, the impact of Anxiety and Age on memory, along with the Processing Speeds and Intelligence are presented. Finally, the analysis of Stress, Age, Visual and Verbal Processing Speeds, and Intelligence are explored.

In order to address the hypotheses presented in Chapter 4, two-way between-groups ANOVAs were conducted to assess group differences.

A median split was used to divide each of the two age groups. Those in the low depression groups comprised half the age group that scored below the median, while the high depression groups contained those participants that scored above the median on the CES-D. The median CES-D score for the younger adults was 8, so those scoring 0-8 became the low depression group (N=26), while those scoring 9 or above became the high depression group (N=24). The median CES-D score for the older adults was 3.5, and so those scoring between 0 and 3 became the low depression group (N=25). Older adults scoring 4 or above became the high depression group (N=25). The means and standard deviations for the depression groups are presented in Table 3.

Table 3

Means (SDs) for depression scores using the CESD for age groups and depression levels*

Age Group	Low Depression	High Depression	Overall Depression
Young	3.96 (2.47)	14.29 (5.91)	8.92 (6.83)
Old	1.40 (1.26)	10.80 (6.49)	6.10 (6.63)

*Note. Unless stated otherwise, all depression scores are based on the CESD.

It was found that the younger and older adults had different mean depression scores, $t(98) = 2.09$, $p = .04$, with the younger adults having the *higher* score. For the younger adults the low depression group had a different mean depression score from the high depression group, $t(30.31) = -7.45$, $p < .001$. Scores for the older adults were also different across the depression levels, $t(25.80) = -7.11$, $p < .001$. This same process was used to assign the groups for visual processing speed, verbal processing speed, IQ, anxiety, and stress when analyses of these variables were conducted (see below).

Comparisons were carried out in order to address the main hypotheses of the study. Two-way between-groups analyses of variance (ANOVAs) were used to explore these hypotheses. Table 4 presents the means and standard deviations (SDs) for the memory tasks grouped by age and level of depression.

Table 4

Mean raw scores (SDs) for all memory tasks grouped by depression scores

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Dep	Young (N=26)	8.27 (1.95)	11.15 (1.54)	22.73 (10.74)	16.58 (3.28)	16.27 (2.74)
	Old (N=24)	7.17 (1.64)	9.68 (1.97)	13.24 (5.43)	12.92 (4.35)	13.36 (3.60)
High Dep	Young (N= 25*)	8.46 (2.02)	11.96 (2.77)	21.87 (11.57)	16.50 (1.98)	16.08 (2.00)
	Old (N=24**)	7.54 (2.13)	9.28 (2.09)	12.38 (6.52)	10.25 (5.61)	13.21 (3.20)

Note. PM = Prospective Memory.

* Recall task N=23

** Letter-Number Sequencing N=23

The results of the ANOVAs are presented in Table 5. Because multiple hypotheses were tested in each ANOVA table presented, a Bonferroni correction (Tabachnick & Fidell, 2007) was used to control for the increased Type 1 error rate in performing multiple ANOVAs. Using this correction, the significance level for the two-way ANOVAs in this study is $p = .003$, while the significance level for the three-way ANOVAs is $p = .001$. While these corrected p values were rather stringent, the size of effects (η^2_p) was given more weight than significance levels in evaluating the findings.

Table 5

Summary of two-way ANOVA for Age and Depression scores

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Depression	F	.50	.23	.23	2.88	.08
	Sig	.48	.64	.64	.09	.78
	η^2_p	.01	.00	.00	.03	.00
	power	.11	.08	.08	.39	.06
Age	F	6.47	23.77	27.70	37.50	23.76
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.20	.23	.28	.20
	power	.71	1.00	1.00	1.00	1.00
Depression	F	.05	2.00	.00	2.57	.00
Age	Sig	.82	.16	1.00	.11	.98
Interaction	η^2_p	.00	.02	.00	.03	.00
	power	.06	.29	.05	.36	.05

As can be seen in Table 5, there were no significant main effects for Depression, and there were no significant interactions between Depression and Age for any of the memory tasks. The effect of Depression on time-based prospective memory approached significance (as does the Depression x Age interaction) but the associated effect size was rather small ($\eta^2_p = .03$) for both the main effect and the interaction. There were medium to large effect sizes noted for main effects of Age across each of the memory tasks. Younger participants had a higher mean score on each of the memory tasks than the older adults.

These results conflict with the expectation there would be an effect for Depression in short term memory as measured by the recall task, although supported the prediction that there would be age effects. The assumption that Depression would have a negative impact on working memory was not confirmed by this analysis. Looking at Table 5, the within group variation was higher than the between group variation for the working memory tasks, with neither Depression nor the Depression x Age interaction having any impact whatsoever. It was predicted that Depression would have a negative impact on time-based prospective memory, but would have no effect on event-based prospective memory. As expected, there were no significant effects or interactions for the event-based prospective memory task. There was also no noticeable effect for time-based prospective memory and Depression.

Because of the failure to find any memory differences due to depression level when using a median split, another two-way between-groups ANOVA was performed, this time using a quartile split. The purpose of this was to provide greater distinction between the two groups in terms of depressive symptomatology. The low and high depression groups were comprised of those below the 25th percentile and above the 75th percentile (for each age group in this sample) on the measure of depression. The means and SDs for the depression scores for the quartile split are presented in Table 6. There is a floor effect for those in the low depression groups due to the overall low scores of the participants in these groups.

Table 6
Means (SDs) for depression scores using the CESD scale for age groups and depression levels in the quartile split analysis

Age Group	Low Depression	High Depression
Young	2.14 (1.46)	18.27 (6.83)
Old	.47 (.52)	16.18 (4.92)

The means for the high depression groups exceed the conventional cut-off score of 16. The low and high groups differ significantly on mean depression score, with $t(23) = -8.64, p < .001$ for the younger adults, and $t(10.16) = -10.56, p < .001$ for the older adults.

The means and SDs for the memory tasks for the quartile split, grouped by age and level of depression, are presented in Table 7.

Table 7

Means (SDs) for the upper and lower quartiles for depression for both the Young and Old groups

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Low Dep	Young (N=12)	8.58 (1.88)	10.75 (1.14)	24.50 (10.87)	17.67 (1.16)	17.17 (1.34)
	Old (N=12)	7.70 (1.64)	9.00 (1.48)	14.17 (4.86)	13.67 (3.70)	13.25 (3.28)
High Dep	Young (N=12)	9.00 (1.60)	13.00 (3.10)	25.67 (13.34)	16.67 (1.78)	15.67 (2.23)
	Old (N=12)	7.64 (2.16)	8.92 (2.11)	13.36 (7.79)	11.82 (5.81)	13.27 (3.64)

The results of the ANOVA are shown in Table 8 and reveal a similar pattern to those seen in Table 5. Again, there were no main effects for Depression but several results are worth considering. The difference in performance in the letter-number sequencing task between the high and low depression groups had an effect size of $\eta^2_p = .07$, which is considered to be small-to-medium; $F(1, 41) = 3.21, p = .08$. However, this effect size needs to be interpreted in the context of the interaction between Age and Depression for letter-number sequencing, with an effect size of $\eta^2_p = .08$. In looking at the group means in Table 7, the performance of the younger adults was better in the high depression group (somewhat counter-intuitively) while performance on the task for the older adults was marginally lower for those in the high depression group. The effect size for Depression produced a small but notable effect, $\eta^2_p = .04$ on the time-based prospective memory task.

Table 8

Summary results for the two-way ANOVAs comparing the upper and lower quartile depression scores

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Depression	F	.10	3.21	.00	1.90	.85
	Sig	.75	.08	.95	.18	.36
	η^2_p	.00	.07	.00	.04	.02
	power	.06	.42	.05	.27	.15
Age	F	4.21	23.25	15.67	18.37	15.47
	Sig	.05	<.001	<.001	<.001	<.001
	η^2_p	.09	.35	.27	.30	.27
	power	.52	1.00	.97	.99	.97
Depression	F	.19	3.72	.12	.17	.90
Age	Sig	.66	.06	.73	.68	.35
Interaction	η^2_p	.01	.08	.00	.00	.02
	power	.07	.47	.06	.07	.15

Table 7 also shows that time-based prospective memory performance was lower in the high depression group compared to the low depression group across both ages. There were medium to large effect sizes for Age across all memory tasks. The effect sizes for these were $\eta^2_p = .09 - .37$, with Age having the smallest impact on recall. The young participants exhibited higher mean scores on each of the memory tasks than the older participants. These results provided further support for the predictions regarding immediate recall. The results regarding the predictions for working memory and prospective memory are somewhat mixed. Unlike the analysis presented in Table 5, the results from the quartile split analysis presented in Table 8 do provide limited support for the prediction that depression will have a negative impact on working memory. There was a trend towards support for the predicted main effect for Depression and the letter-number sequencing task but not for the reading span task (Table 5). The prediction that there would be a main effect for Depression and time-based prospective memory was supported by a small effect ($\eta^2_p = .04$), with

those in the low depression group outperforming the high depression group in both ages.

Summary

The ANOVA results presented in Table 5 did not support the expectations that short-term memory, working memory, and prospective memory would be affected by depression. To investigate further, another ANOVA was conducted in which only those with the 12 highest and lowest depression scores were taken into account (presented in Table 8). There were several observable trends for Depression or Depression x Age interactions. There was a trend towards Depression having a negative impact on time-based prospective memory, but not event-based prospective memory. A small effect size for the working memory task of letter-number sequencing was also observed. This effect was qualified by the Depression x Age interaction for letter number sequencing which produced a small effect. From the means and SDs in Table 7 it appears that the older adults' performance on the letter-number sequencing task was largely unaffected by level of depression, whereas the younger adults' in the high depression group exhibited superior performance.

Moderator Analysis

Depression

There are several factors that are related to depression and memory that need be taken into account. As outlined in the introductory chapters, the present research gave priority to investigating processing speed and intelligence alongside memory, and anxiety and stress alongside depression. Given the relationships among these variables, it seemed prudent to conduct between-groups ANOVAs to investigate any moderating effects these factors may have had on depression and memory. As noted in the Method section, these factors were treated as independent variables due to the inappropriateness of using ANCOVA without random assignment to groups. Three-way ANOVAs had to

be used, considering one moderator at a time, because cell numbers were insufficient to run four-way ANOVAs. (Multiple regression analyses were also conducted separately for the young and old groups. While the results were similar to those of the ANOVA results, the analyses were discarded due to a lack of statistical power.)

It was originally thought that the visual and verbal processing speed tests would be combined to form one overall processing speed variable. However, it was found that there was only a weak relationship between the two variables within each of the age groups. For the younger adults, $r_s = .19$, n.s.; for the older adults, $r_s = .25$, n.s. For this reason, visual and verbal processing speeds were kept as individual variables.

Depression and Visual Processing Speed

Because the full data set for the present study is complex (Tables 2 and 4) data relevant to the analysis in hand have been extracted and re-presented for convenience. A median split was again used to create the visual processing speed groups. For the younger group, those scoring 80 and below on the Identical Pictures test were in the low visual processing group. For the older adults, those scoring 44 and below were in the low visual processing group. The means and SDs of the groups are presented in Table 9.

Table 9

Means (SDs) for visual processing speed scores using the Identical Pictures test for age groups and visual processing speed levels

Age Group	Low VisPS	High VisPS	Overall VisPS
Young	71.12 (5.65)	90.20 (4.64)	80.66 (10.91)
Old	37.92 (6.09)	53.68 (8.58)	45.80 (10.84)

The low visual processing speed group had a different mean score from the high visual processing speed group across both ages. For the younger adults, $t(48) = -13.05$, $p < .001$. For the older adults, $t(48) = -7.49$, $p < .001$.

The first of these data sets can be seen in Table 10, showing the means and SDs of the groups in the three-way between-groups ANOVA conducted to investigate the impact of visual processing speed on depression, age, and memory performance.

Table 10

Means (SDs) for three-way ANOVAs for five types of memory as a function of Depression, Age, and Visual processing speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Dep	Low	Young (N=12)	7.75 (1.60)	11.58 (2.02)	19.00 (8.58)	16.75 (3.75)	15.50 (3.53)
	VisPS	Old (N=10)*	7.22 (1.86)	9.10 (1.60)	12.80 (4.64)	12.60 (4.20)	12.30 (4.27)
	High	Young (N=14)	8.71 (2.16)	10.79 (0.89)	25.93 (11.64)	16.43 (2.95)	16.93 (1.69)
	VisPS	Old (N=15)**	7.14 (1.56)	10.07 (2.15)	13.53 (6.05)	13.13 (4.58)	14.07 (3.04)
High Dep	Low	Young (N=13)	8.00 (2.35)	12.62 (3.01)	24.15 (11.89)	15.54 (2.18)	16.00 (2.31)
	VisPS	Old (N=14)***	6.36 (1.60)	8.73 (1.83)	12.93 (7.12)	9.14 (6.27)	12.71 (2.76)
	High	Young (N=11)	9.00 (1.48)	11.18 (2.36)	19.18 (11.12)	17.64 (0.81)	16.18 (1.66)
	VisPS	Old (N=10)	9.20 (1.62)	10.10 (2.28)	11.60 (5.87)	11.80 (4.37)	13.90 (3.78)

Note. VisPS = Visual Processing Speed

* Recall task N=9

** Recall task N=14

*** Letter-Number Sequencing task N=15.

Table 11 summarises the results of the five ANOVAs, one for each type of memory. There were no notable effect sizes for Depression across any of the memory tasks in this analysis. There were medium to large effect sizes ranging from $\eta^2_p = .20$ to $.29$ for Age for the working memory and prospective memory tasks. With the Bonferroni correction, there was no significant main effect for Age for memory recall, although the effect size was notable at $\eta^2_p = .06$. The younger participants had higher mean scores across each group and memory task than the older adults, with the singular exception of those participants in the high depression and high visual processing speed group, where the older participants marginally outperformed their younger counterparts on the recall task. There was a near significant main effect for Visual Processing Speed for recall $F(1, 89) = 10.03, p = .002; \eta^2_p = .10$, but this was qualified by a number of interactions.

Table 11

A summary of the three-way ANOVA results for five types of memory as a function of Depression, Age, and Visual Processing Speed

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Depression	F	1.34	.42	.22	2.16	.00
	Sig	.25	.52	.64	.15	1.00
	η^2_p	.02	.01	.00	.02	.00
	power	.21	.10	.08	.31	.05
Age	F	5.63	23.29	26.86	36.36	23.69
	Sig	.02	<.001	<.001	<.001	<.001
	η^2_p	.06	.20	.23	.29	.21
	power	.65	1.00	1.00	1.00	1.00
VisPS	F	10.03	.00	.36	2.32	3.65
	Sig	.002	.95	.85	.13	.06
	η^2_p	.10	.00	.00	.03	.04
	power	.88	.05	.05	.33	.47
Depression Age Interaction	F	.19	1.08	.00	2.15	.04
	Sig	.66	.30	.98	.15	.84
	η^2_p	.00	.01	.00	.02	.00
	power	.07	.18	.05	.31	.06
Depression VisPS Interaction	F	3.93	.02	3.74	1.94	.59
	Sig	.05	.89	.06	.17	.45
	η^2_p	.04	.00	.04	.02	.01
	power	.50	.05	.48	.28	.12
Age VisPS Interaction	F	.29	7.28	.13	.19	.32
	Sig	.59	.01	.72	.67	.58
	η^2_p	.00	.07	.00	.00	.00
	power	.08	.76	.06	.07	.09
Depression Age VisPS Interaction	F	3.74	.38	1.86	.01	.08
	Sig	.06	.54	.18	.93	.78
	η^2_p	.04	.00	.02	.00	.00
	power	.48	.09	.27	.05	.06

Note. VisPS = Visual Processing Speed

There was a small effect for the interaction between Depression and Visual Processing Speed for recall, $F(1, 89) = 3.93, p = .05; \eta^2_p = .04$; and a small effect for the three-way interaction between Depression, Age, and Visual Processing Speed for recall, $F(1, 89) = 3.74, p = .06; \eta^2_p = .04$. Further investigation of these

results indicated that recall performance was marginally greater for the high depression group in both the high and low visual processing speed groups. In the older adults, there was a negligible difference in recall performance for the low depression group, regardless of visual processing speed ability, while in the high depression older adults, those with high visual processing speed performed substantially better than those with low processing speed. This interaction is represented in Figure 1 below. As can be seen from this graph, the two-way interaction between Depression and Visual Processing Speed made the major contribution to the three-way interaction.

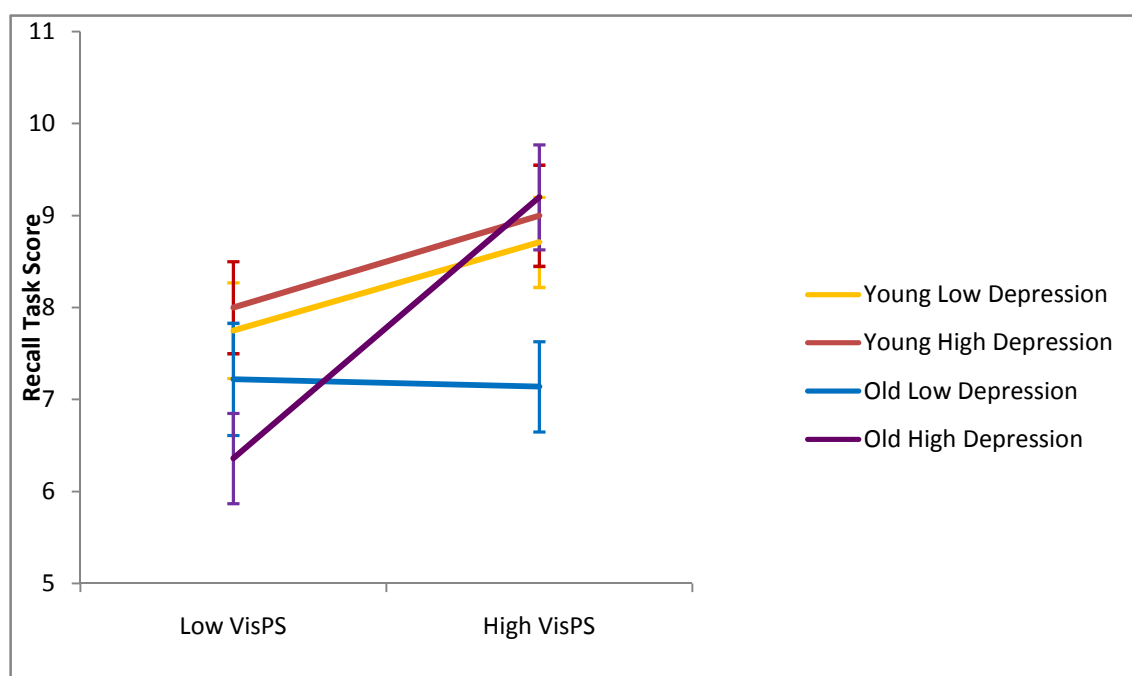


Figure 1. Graphical representations of the interactions between Depression, Age, and Visual Processing Speed on the recall task.

There was a small effect for Visual Processing Speed in the event-based prospective memory task, $F(1, 89) = 3.65$, $p = .06$; $\eta^2_p = .04$. A review of the means presented in Table 10 indicates that those with higher visual processing speed scores performed slightly better on the event-based memory tasks across both age and depression groups. The Depression $\times$ Visual Processing Speed interaction for the reading span task also revealed a small effect size, $F(1, 91) =$

3.74, $p = .06$; $\eta^2_p = .04$. The scores of those in the low depression group were a little higher on average on the reading span task in the high visual processing group across both ages, while those in the high depression group exhibited better performance on the reading span task in the low visual processing group. There was another trend towards an interaction between Age and Visual Processing Speed for letter-number sequencing, with a small to medium effect size, $F(1, 92) = 7.28$, $p = .01$; $\eta^2_p = .07$. The younger adults in the low visual processing speed group had a mean score above that of the high visual processing speed group on the letter-number sequencing task, while the opposite pattern prevailed for the older adults.

Summary

Processing speed is known to be related to memory ability, and was investigated in this study to see if it was a moderating factor in the depression and memory relationship. Due to low correlations between the two, verbal and visual processing speeds were investigated independently. Interestingly, there were two small effects for the Depression by Visual Processing Speed interaction. The first was for the recall task which was qualified by the small effect for the Depression $\times$ Age $\times$ Visual Processing Speed interaction in which the younger adults had higher recall scores in the high depression group regardless of visual processing ability. In contrast, the older adults in the low visual processing speed group showed a deterioration in performance in the high depression group, whereas the older adults in the high visual processing speed group showed superior performance in the high depression group (even out performing their younger counterparts). This interaction between Depression and Visual Processing Speed accounted for most of the three-way interaction effect.

Depression and Verbal Processing Speed

For verbal processing speed, the younger adults were in the low group with a score of 45 or lower. The older adults were in the low group with a score of 49 or lower. As can be seen in Table 12, the older adults started out with a higher mean score on the verbal processing speed task (although this difference was not significant). The mean scores for the low and high verbal processing speed groups were significantly different in each age group with $t(33.13) = -9.29$, $p < .001$ for the younger adults and $t(48) = -9.28$, $p < .001$ for the older adults.

Table 12

Means (SDs) for verbal processing speed scores using the Finding A's test for age groups and verbal processing speed levels

Age Group	Low VerPS	High VerPS	Overall VerPS
Young	36.20 (5.05)	59.28 (11.36)	47.74 (14.55)
Old	37.17 (7.67)	60.65 (9.98)	49.38 (14.79)

Table 13 presents the means and SDs of the groups for the three-way ANOVAs performed to investigate the effects of Depression, Age, and Verbal Processing Speed on memory performance. The mean performance of the younger adults exceeded that of their older counterparts in every one of the memory tasks. The mean performance on the recall task for the low depression and low verbal processing speed group of older adults was very close to that of the younger adults, with the younger adults showing the greater variability around the mean.

Table 13

Means (SDs) for three-way ANOVAs for five types of memory as a function of Depression, Age, and Verbal Processing Speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low	Low	Young (N=12)	7.33 (1.56)	11.33 (2.06)	21.83 (11.32)	16.75 (3.75)	15.67 (3.50)
Dep	VerPS	Old (N=11)*	7.30 (1.25)	9.45 (2.16)	12.55 (5.48)	12.45 (4.59)	12.91 (4.32)
	High	Young (N=14)	9.07 (1.94)	11.00 (0.96)	23.50 (10.57)	16.43 (2.95)	16.79 (1.85)
	VerPS	Old (N=14)**	7.08 (1.94)	9.86 (1.88)	13.79 (5.54)	13.29 (4.29)	13.71 (3.05)
High	Low	Young (N=13)	8.08 (2.02)	12.08 (3.07)	22.85 (12.97)	16.31 (2.14)	16.15 (2.23)
Dep	VerPS	Old (N=12)***	7.58 (2.34)	8.85 (2.41)	11.00 (5.64)	9.42 (5.45)	12.83 (3.59)
	High	Young (N=11)	8.91 (2.02)	11.82 (2.52)	20.73 (10.16)	16.73 (1.85)	16.00 (1.79)
	VerPS	Old (N=12)	7.50 (1.93)	9.75 (1.66)	13.75 (7.28)	11.08 (5.89)	13.58 (2.88)

Note. VerPS = Verbal Processing Speed

*Recall task N=10

**Recall task N=13

***Letter-Number Sequencing task N=13

Table 14 summarises the five ANOVAs looking at the impact of depression, age, and verbal processing speed on the different types of memory. There were no notable effect sizes for Depression or Verbal Processing Speed. There were medium to large effects for Age ($\eta^2_p = .20$ to $.29$), again across working memory and prospective memory. The younger participants had higher mean scores across all groups and memory tasks compared to the older participants. There were no remarkable effect sizes for the two-way interactions between Depression and Age or Depression and Verbal Processing Speed. One interaction for Age and Verbal Processing Speed produced a small effect, $F(1, 89) = 3.37, p = .07; \eta^2_p = .04$, for the recall tasks. The means provided in Table 10 show that performance on the recall task improved for the younger adults in the high verbal processing speed group while the older adults showed negligible change across the verbal processing speed groups. The three-way interactions all produced negligible effects.

Table 14

A summary of the three-way ANOVA results for five types of memory as a function of Depression, Age, and Verbal Processing Speed

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Depression	F	.68	.24	.21	2.67	.04
	Sig	.41	.63	.65	.11	.83
	η^2_p	.01	.00	.00	.03	.00
	power	.13	.08	.07	.37	.06
Age	F	6.29	23.09	26.49	36.80	23.04
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.20	.23	.29	.20
	power	.70	1.00	1.00	1.00	1.00
VerPS	F	2.09	.17	.23	.62	1.09
	Sig	.15	.68	.63	.43	.30
	η^2_p	.02	.00	.00	.01	.01
	power	.30	.07	.08	.12	.18
Depression Age Interaction	F	.01	1.73	.00	2.40	.00
	Sig	.94	.19	.98	.13	.97
	η^2_p	.00	.02	.00	.03	.00
	power	.05	.26	.05	.33	.05
Depression VerPS Interaction	F	.24	.11	.10	.23	.30
	Sig	.63	.74	.76	.63	.58
	η^2_p	.00	.00	.00	.00	.00
	power	.08	.06	.06	.08	.09
Age VerPS Interaction	F	3.37	1.20	.37	.53	.06
	Sig	.07	.28	.55	.47	.81
	η^2_p	.04	.01	.00	.01	.00
	power	.44	.19	.09	.11	.06
Depression Age VerPS Interaction	F	.45	.06	.52	.00	.26
	Sig	.51	.81	.47	.98	.62
	η^2_p	.01	.00	.01	.00	.00
	power	.10	.06	.11	.05	.08

Note. VerPS = Verbal Processing Speed

Summary

The low correlation between visual processing speed and verbal processing speed in this sample was highlighted by the differing ANOVA patterns presented. The only notable effects were for ageing in this analysis. There was an Age x Verbal Processing Speed interaction for the recall task which yielded a small effect. In this interaction, the younger adults in the high verbal processing speed group showed superior performance when compared with their low verbal processing speed counterparts, while the older adults maintained a similar level of performance across verbal processing speed groups.

Depression and IQ

The median split for the IQ groups resulted in groups with significantly different mean scores when the low and high IQ groups were compared for each age group. For the younger adults, $t(48) = -8.17, p < .001$. For the older adults, $t(48) = -7.83, p < .001$. The means and SDs for the groups can be seen below in Table 15.

Table 15

Means (SDs) for IQ scores using the short form of the WAIS-III test for age groups and IQ levels

Age Group	Low IQ	High IQ	Overall IQ
Young	107.81 (6.00)	121.63 (5.96)	114.44 (9.15)
Old	99.81 (9.20)	119.71 (8.74)	109.36 (13.42)

Table 16 presents the means and SDs for the Depression, Age, and IQ three-way ANOVAs. Again the younger adults had greater mean performances than the older adults on all memory tasks. Interestingly, the high depression and high IQ groups appeared to have the highest mean scores for the retrospective

memory tasks (Recall, Letter-Number Sequencing, and Reading Span) across both age groups.

Table 16

Means (SDs) for three-way ANOVAs for five types of memory as a function of Depression, Age, and IQ

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Dep	Low IQ	Young (N=12)	8.00 (1.95)	11.17 (1.03)	21.83 (8.97)	17.67 (1.16)	16.83 (1.80)
		Old (N=11)	7.00 (1.73)	9.45 (1.37)	11.36 (4.27)	12.82 (5.12)	13.00 (4.31)
	High IQ	Young (N=14)	8.50 (1.99)	11.14 (1.92)	23.50 (12.33)	15.64 (4.18)	15.79 (3.33)
		Old (N=14)*	7.33 (1.61)	9.86 (2.38)	14.71 (5.93)	13.00 (3.84)	13.64 (3.08)
High Dep	Low IQ	Young (N=14)	7.57 (1.99)	11.07 (2.89)	16.50 (7.36)	16.57 (2.14)	16.14 (1.99)
		Old (N=14)**	7.50 (2.50)	8.93 (2.05)	9.64 (3.97)	7.29 (4.68)	12.86 (2.98)
	High IQ	Young (N=10)	9.70 (1.34)	13.20 (2.15)	29.40 (12.48)	16.40 (1.84)	16.00 (2.11)
		Old (N=10)	7.60 (1.58)	9.80 (2.15)	16.20 (7.61)	14.40 (4.01)	13.70 (3.59)

*Recall task N=12

**Letter-Number Sequencing task N=15

The final three-way between groups ANOVAs involving IQ are displayed in Table 17. There were no main effects for Depression, while main effects for Age were significant for the working memory and prospective memory tasks, with the younger participants outperforming the older participants. The main effect for IQ in letter-number sequencing (working memory) yielded a small effect size, $F(1, 92) = 3.97, p = .05; \eta^2_p = .04$, with those in the higher IQ groups performing better on the letter-number sequencing task regardless of age or depression level. The main effect for IQ with the reading span task (working memory) achieved a medium effect $\eta^2_p = .13$, and was also significant, $F(1, 91) = 13.04, p = <.001$. Again, those with a higher IQ performed better on the reading span task than did those in the lower IQ group (although this is qualified by a Depression x IQ interaction). There was a small effect for IQ and recall, $F(1, 89) = 3.84, p = .05; \eta^2_p = .04$ in which those with higher IQ again performed better on the recall task.

Table 17

A summary of the three-way ANOVA results for five types of memory as a function of Depression, Age, and IQ

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Depression	F	.97	.67	.00	2.27	.05
	Sig	.33	.42	.96	.14	.82
	η^2_p	.01	.01	.00	.02	.00
	power	.16	.13	.05	.32	.06
Age	F	7.70	25.39	33.64	40.03	22.68
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.08	.22	.27	.31	.20
	power	.78	1.00	1.00	1.00	1.00
IQ	F	3.84	3.97	13.04	2.95	.02
	Sig	.05	.05	<.001	.09	.90
	η^2_p	.04	.04	.13	.03	.00
	power	.49	.50	.95	.40	.05
Depression Age Interaction	F	.00	2.25	.01	1.63	.03
	Sig	1.00	.14	.91	.20	.87
	η^2_p	.00	.02	.00	.02	.00
	power	.05	.32	.05	.24	.05
Depression IQ Interaction	F	.80	2.39	4.54	8.76	.21
	Sig	.37	.13	.04	.004	.65
	η^2_p	.01	.03	.05	.09	.00
	power	.14	.33	.56	.83	.07
Age IQ Interaction	F	1.97	.24	.47	10.23	1.22
	Sig	.16	.62	.49	.002	.27
	η^2_p	.02	.00	.01	.10	.01
	power	.29	.08	.10	.89	.19
Depression Age IQ Interaction	F	1.42	.99	1.40	2.93	.08
	Sig	.24	.32	.24	.09	.77
	η^2_p	.02	.01	.02	.03	.00
	power	.22	.17	.22	.40	.06

There were no significant Depression $\times$ Age, or Depression $\times$ IQ interactions for any of the memory tasks. The Depression $\times$ IQ interaction for reading span produced a small effect size, $F(1, 91) = 4.54, p = .04; \eta^2_p = .05$. Table 16 indicates that regardless of age, those in the high IQ and high depression groups

performed better on the reading span task than those with high IQ and low depression. Conversely, those in the lower IQ, low depression groups performed better than those in the low IQ, high depression groups, as can be seen in Figure 2. The Depression x IQ interaction for time-based prospective memory produced a medium effect size, $F(1, 91) = 8.76, p = <.01; \eta^2_p = .09$. As with the Depression x IQ interaction for the reading span task, the Depression x IQ interaction for time-based prospective memory indicated those with a higher IQ showed better performance in the high depression groups, while those in the lower IQ groups exhibited lower task performance with high depression. Finally, the interaction between Age and IQ for time-based prospective memory also yielded a medium effect size, $F(1, 91) = 10.23, p = <.01; \eta^2_p = .10$. There were no significant three-way interactions.

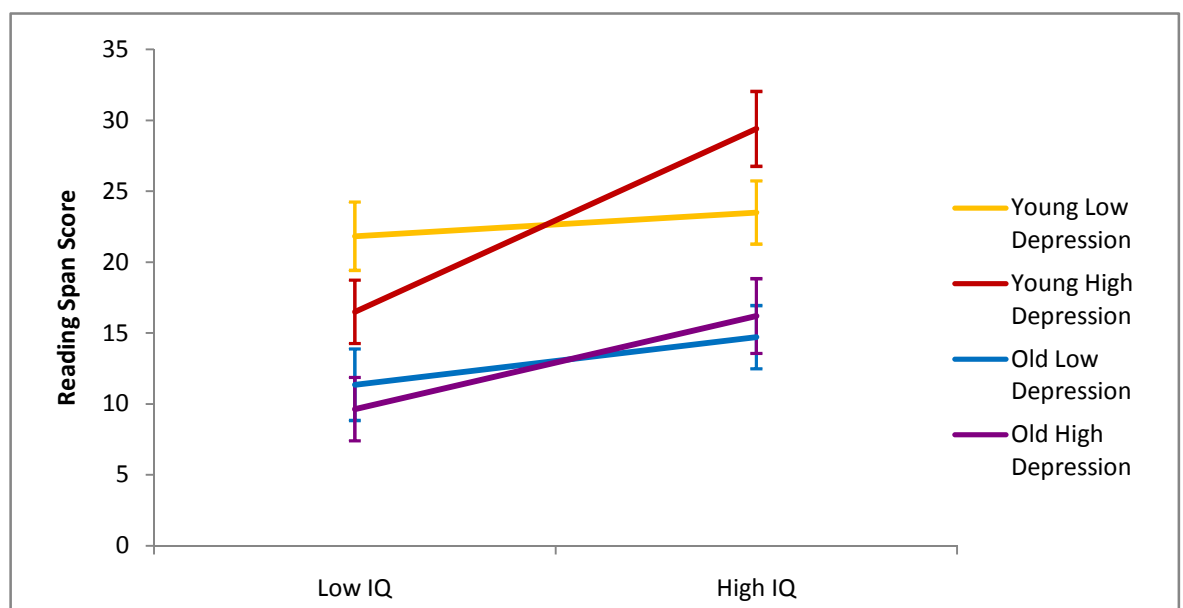


Figure 2. Graphical representation of interactions between Depression and IQ on the reading span task.

Summary

Retrospective memory was affected by IQ in this study. The main effect for IQ on the reading span task had a medium effect size. There was also a small effect for IQ for the recall and letter-number sequencing tasks. There were two notable Depression x IQ interactions. The first was for the reading span task in

which a small effect was present indicating that those with a high IQ in the high depression group tended to outperform those in the high IQ low depression group, while the reverse was true for those in the lower IQ group. The second Depression x IQ interaction was for time-based prospective memory which produced a medium effect. This followed a similar pattern to the Depression x IQ interaction for the reading span task in that those in the higher IQ and high depression groups outperformed those in the higher IQ low depression groups, with the reverse pattern seen for those in the lower IQ groups (the low depression groups outperforming the high depression groups).

Summary of the Depression and Moderator Analyses

The moderator analyses revealed a variable pattern of effects. There were small effects for Visual Processing Speed (for the recall and event-based memory tasks) and for IQ (for the recall and both working memory tasks). Yet there were no main effects across any of the memory tasks for Verbal Processing Speed. In a similar vein, only Visual Processing Speed and IQ trended towards interacting with Depression. There were small effects observed for the Depression and Visual Processing Speed interactions for the recall task and for the reading span task. There were also small effects observed for the Depression x IQ interactions for the reading span and time-based prospective memory tasks. The only three-way interaction with a notable but small effect was for the Depression x Age x Visual Processing Speed interaction for the recall task presented in Figure 1, but the latter was mainly due to the interaction between Depression and Visual Processing Speed for the older adults.

Anxiety and Stress

There are known to be high correlations between depression, anxiety, and stress, and it was deemed prudent to investigate the effects of these variables as well as depression (Beuke et al., 2003). For this reason, ANOVA was used to investigate the impact of anxiety and stress on memory. (The presentation of

these results will be limited to anxiety specific effects and interactions, and will compare them to the depression results.)

The correlations between the measures of depression and anxiety in the current sample were $r_s = .62, p < .001$ for the combined sample; $r_s = .54, p < .001$ for the younger adults, and for the older adults, $r_s = .63, p < .001$. The correlation between depression and stress in the combined sample was $r_s = .69, p < .001$. For the younger adults it was $r_s = .74, p < .001$; while for the older adults, $r_s = .55, p < .001$. The correlation between the measures of anxiety and stress were $r_s = .69, p < .001$ for the combined sample, $r_s = .65, p < .001$ for the younger adults, and $r_s = .69, p < .001$ for the older adults. All of these relationships were relatively large indicating that these three factors shared a high degree of common variance.

As with the Depression analyses, there were multiple hypotheses being tested in each of the Anxiety and Stress ANOVAs. The Bonferroni correction discussed above was again used, with a $p = .003$ significance level for the two-way ANOVAs, while the significance level for the three-way ANOVAs is $p = .001$.

Median splits were again used to define the low and high anxiety and stress groups. The means for each anxiety group are presented below in Table 18. The scores on the anxiety scale contained a high degree of variability for both age groups as can be seen by SDs that exceed the overall means. As with the depression groups, a floor effect exists for the low anxiety groups for both ages.

Table 18

Means (SDs) for Anxiety scores using the DASS-A scale for age groups and anxiety levels

Age Group	Low Anxiety	High Anxiety	Overall Anxiety
Young	1.04 (.72)	6.37 (3.79)	3.60 (3.77)
Old	.34 (.48)	5.76 (4.77)	2.62 (4.09)

The mean scores for the low and high groups were significantly different. The mean anxiety score for the high anxiety group of younger adults was significantly higher than the low anxiety group, $t(24.54) = -6.79, p < .001$. The same was true for the older adults, $t(20.30) = -5.18, p < .001$.

The means and SDs for the stress groups are presented in Table 19. When these means were considered, the low and high stress groups were significantly different. For the younger adults, $t(34.71) = -9.91, p < .001$. For the older adults, $t(25.14) = -7.16, p < .001$. Again, there is a floor effect for the low stress groups.

Table 19

Means (SDs) for Stress scores using the DASS-S scale for age groups and stress levels

Age Group	Low Stress	High Stress	Overall Stress
Young	2.92 (2.57)	14.56 (5.28)	8.74 (7.17)
Old	1.20 (.96)	10.64 (6.20)	5.92 (6.48)

Anxiety

The means and SDs for the two-way ANOVAs investigating the impact of Anxiety and Age on memory are presented in Table 20. From this table it can be seen that the younger adults had higher mean scores on all of the memory tasks when compared to the older adults. The younger adults in the low anxiety group generally outperformed their high anxiety counterparts for the memory tasks (with the exception of Letter-Number Sequencing in which the pattern was reversed). In comparison, the older adults in the low anxiety group appeared to outperform their high anxiety counterparts for the retrospective memory tasks but not for the prospective memory tasks. These differences were investigated in the ANOVA results presented below.

Table 20

Means (SDs) for all memory tasks grouped by anxiety level and age

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Anxiety	Young (N=26)	8.54 (1.90)	10.81 (1.72)	23.04 (12.45)	16.69 (2.40)	16.81 (1.60)
	Old (N=29)*	7.41 (1.95)	9.76 (2.26)	13.10 (5.31)	11.28 (5.32)	13.10 (3.54)
High Anxiety	Young (N=24)	8.17 (2.06)	12.33 (2.48)	21.54 (9.47)	16.37 (3.05)	15.50 (2.90)
	Old (N=20)**	7.30 (1.87)	9.10 (1.61)	12.40 (6.88)	12.10 (4.94)	13.55 (3.20)

*Recall task N=27

**Letter-Number Sequencing task N=21

The results for the anxiety analysis generally mirrored those of the depression analysis seen in Table 5. There were no prominent effect sizes for anxiety and memory in the analysis presented in Table 21. The interaction between Age and Anxiety for the letter-number sequencing task was small to medium, $F(1, 96) = 6.91, p = .01; \eta^2_p = .07$. This differed from the results for depression in the entire sample where there was no such interaction. However, when the upper and lower 12 depression scores were analysed, a similar interaction trend for the letter-number sequencing task was observed. The group means from Table 20 indicated that, while the younger adults' performance on the letter-number sequencing task was higher with higher anxiety, the older adults' performance on the task was lower in the high anxiety group. From these results, it appeared a higher level of anxiety may have been beneficial for younger adults completing the working memory task but detrimental for older adults.

Table 21

Summary of two-way ANOVA for Age and Anxiety scores

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Anxiety	F	.36	1.07	.37	.09	.53
	Sig	.55	.30	.55	.76	.47
	η^2_p	.00	.01	.00	.00	.01
	power	.09	.18	.09	.06	.11
Age	F	6.30	26.50	27.53	33.57	22.96
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.06	.22	.23	.26	.20
	power	.70	1.00	1.00	1.00	1.00
Anxiety	F	.11	6.91	.05	.46	2.21
Age	Sig	.74	.01	.83	.50	.14
Interaction	η^2_p	.00	.07	.00	.01	.02
	power	.06	.74	.06	.10	.31

Summary

The Anxiety results presented above were generally consistent with those observed for Depression. There were no main effects for Anxiety. The main effects for Age were similar, with working memory and prospective memory achieving medium to large effect sizes. There was a small effect for the Age x Anxiety interaction for the letter-number sequencing task, which was similar to the upper and lower quartile Depression analysis (see Table 8).

Anxiety and Visual Processing Speed

As for depression and memory, three-way between groups ANOVAs were conducted to investigate the effects of visual and verbal processing speed, and IQ on anxiety, age, and memory performance. The means and SDs for the first analysis on Age, Anxiety, and Visual Processing Speed for the 5 memory types can be seen in Table 22.

Table 22

Means (SDs) for three-way ANOVAs for five memory tasks as a function of Anxiety, Age, and Visual Processing Speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Anxiety	Low	Young (N=12)	8.42 (1.83)	11.42 (2.02)	23.83 (12.54)	16.67 (1.78)	16.83 (1.34)
	VisPS	Old (N=14)*	7.08 (1.89)	9.00 (1.80)	10.64 (3.75)	9.43 (5.64)	11.93 (3.97)
	High	Young (N=14)	8.64 (2.02)	10.29 (1.27)	22.36 (12.81)	16.71 (2.89)	16.79 (1.85)
	VisPS	Old (N=15)**	7.71 (2.02)	10.47 (2.47)	15.40 (5.63)	13.00 (4.54)	14.20 (2.78)
High Anxiety	Low	Young (N=13)	7.38 (2.06)	12.77 (2.95)	19.69 (8.34)	15.62 (3.86)	14.77 (3.61)
	VisPS	Old (N=10)***	6.20 (1.40)	8.73 (1.68)	16.00 (7.45)	12.20 (5.59)	13.40 (2.71)
	High	Young (N=11)	9.09 (1.70)	11.82 (1.78)	23.73 (10.63)	17.27 (1.35)	16.36 (1.50)
	VisPS	Old (N=10)	8.40 (1.65)	9.50 (1.51)	8.80 (3.94)	12.00 (4.50)	13.70 (4.06)

Note. VisPS = Visual Processing Speed

*Recall task N=13

**Recall task N=14

**Letter-Number Sequencing task N=11

The results of the three-way ANOVA for these factors are presented in Table 23. There were no significant main effects for Anxiety. Again, this pattern reflected that seen in the depression and visual processing speed analysis presented in Table 11. The younger adults performed better than the older adults across all tasks and conditions except for those in the low anxiety and high visual processing speed group where both age groups exhibited a similar performance on the letter-number sequencing task.

Table 23

A summary of the three-way ANOVA results for five memory tasks as a function of Anxiety, Age, and Visual Processing Speed

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Anxiety	F	.26	1.01	.31	.15	.42
	Sig	.61	.32	.58	.70	.52
	η^2_p	.00	.01	.00	.00	.01
	power	.08	.17	.09	.07	.10
Age	F	7.40	27.59	29.18	35.28	24.36
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.08	.23	.24	.28	.21
	power	.77	1.00	1.00	1.00	1.00
VisPS	F	9.80	.01	.00	2.36	3.11
	Sig	.002	.92	.99	.13	.08
	η^2_p	.10	.00	.00	.03	.03
	power	.87	.05	.05	.33	.42
Anxiety Age Interaction	F	.07	6.35	.05	.47	2.19
	Sig	.80	.01	.83	.50	.14
	η^2_p	.00	.07	.00	.01	.02
	power	.06	.70	.06	.10	.31
Anxiety VisPS Interaction	F	3.99	.10	.81	.43	.02
	Sig	.05	.75	.37	.52	.89
	η^2_p	.04	.00	.01	.01	.00
	power	.51	.06	.14	.10	.05
Age VisPS Interaction	F	.35	6.97	.49	.25	.19
	Sig	.55	.01	.49	.62	.66
	η^2_p	.00	.07	.01	.00	.00
	power	.09	.74	.11	.08	.07
Anxiety Age VisPS Interaction	F	.00	.29	5.92	2.65	2.40
	Sig	.96	.60	.02	.11	.13
	η^2_p	.00	.00	.06	.03	.03
	power	.05	.08	.67	.36	.33

Note. VisPS = Visual Processing Speed

As with the two-way ANOVAs presented above in Table 21, there was a modest effect for the Anxiety x Age interaction for the letter-number sequencing task, $F(1, 92) = 6.35$, $p = .01$; $\eta^2_p = .07$. This is comparable to the

Depression analyses in which there was a medium effect for the Depression x Age interaction for the letter-number sequencing task in the upper and lower quartile analysis. The only two-way interaction to show a trend between Anxiety and Visual Processing Speed was for the recall task, $F(1, 89) = 3.99, p = .05; \eta^2_p = .40$. In this interaction, performance for the low Visual Processing Speed group was worse for those in the high anxiety group, while the opposite occurred for the high Visual Processing Speed group, where performance in the recall task was greater for the high anxiety group. This pattern was maintained across both age groups, and was similar to the pattern observed for the depression, visual processing speed, and recall interaction.

The three-way interaction between Anxiety, Age, and Visual Processing Speed produced a small effect, and occurred for the reading span task, $F(1, 91) = 5.92, p = .02; \eta^2_p = .06$. In this interaction, performance on the reading span task was higher for the young high visual processors in the high anxiety group than the low anxiety group, whereas those young low visual processors performed better in the low anxiety group as opposed to the high anxiety group. The reverse pattern was seen in the older adults, with those exhibiting high anxiety and low visual processing speed performing better than those exhibiting low anxiety and low visual processing speed. Additionally, the low anxiety, high visual processing speed older adults performed better than the high anxiety, high visual processing speed older adults (see Figure 3). According to these results, anxiety had a detrimental effect on the reading span task for younger adults with low visual processing speed and for older adults with high processing speed. This result diverged from the depression findings in which the notable three-way interaction was for recall as opposed to reading span.

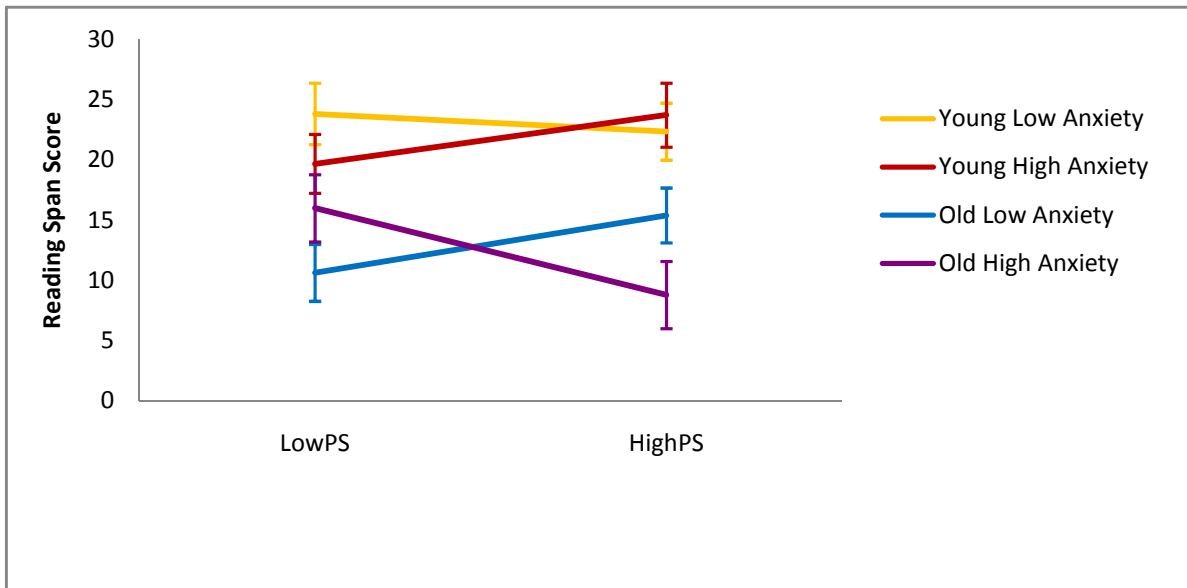


Figure 3. Graphical representation of the interactions between Anxiety, Age, and Visual Processing Speed for the reading span task.

Summary

There were some interesting similarities and differences when comparing the results of this analysis with those of the Depression and Visual Processing Speed analysis. Importantly, there was a small effect for the Anxiety x Visual Processing Speed interaction in the recall task, as there was for the Depression x Visual Processing Speed interaction in the recall task, as there was for the Depression x Visual Processing Speed interaction. In the Anxiety analysis, those in the high visual processing speed groups tended to do better in the high anxiety groups, while those in the low visual processing speed group tend to do better with low anxiety (regardless of age). The equivalent interaction with Depression x Visual Processing Speed for the recall task was qualified by age and differed at this point from the Anxiety analysis. While the younger adults showed a similar pattern of improvement with higher visual processing speed across the different levels of Depression, the older adults in the high depression and high visual processing speed group had a considerably higher mean recall score than those in the high depression and low visual processing speed group (refer to Figure 1). While there was also a small Depression x Visual Processing Speed interaction for the reading span task, there was no such trend in the Anxiety analysis. The analyses also differ in three-way interactions. As noted above,

there was a trend towards a three-way interaction between Depression, Visual Processing Speed and Age for the recall task. The only trend towards a three-way interaction for the Anxiety analysis was for the reading span task in which there was a small effect. As seen in Figure 3 above, the combination of high visual processing speed and high anxiety in the older adults results in the poorest performance. The prospective memory tasks seemed unaffected by anxiety.

Anxiety and Verbal Processing Speed

Table 24 presents the means and SDs for the Anxiety, Age, and Verbal Processing Speed ANOVAs. Generally, the younger adults outperform the older adults. There was little difference between younger and older adults in the high anxiety and low verbal processing speed groups for the Recall task and for those in the low anxiety and high verbal processing speed in the Letter-Number Sequencing task.

Table 24

Means (SDs) for three-way ANOVAs for five types of memory as a function of Anxiety, Age, and Verbal Processing Speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Anxiety	Low VerPS	Young (N=13)	8.23 (2.01)	11.08 (2.02)	25.00 (14.02)	17.38 (1.26)	17.08 (1.04)
		Old (N=16)*	7.60 (1.81)	9.44 (2.48)	12.63 (5.78)	10.94 (4.88)	13.00 (3.50)
	High VerPS	Young (N=13)	8.85 (1.82)	10.54 (1.39)	21.08 (10.87)	16.00 (3.06)	16.54 (2.03)
		Old (N=13)**	7.17 (2.17)	10.15 (1.99)	13.69 (4.84)	11.69 (6.01)	13.23 (3.72)
High Anxiety	Low VerPS	Young (N=12)	7.17 (1.47)	12.42 (3.06)	19.50 (8.98)	15.58 (3.94)	14.67 (3.65)
		Old (N=7)***	7.14 (2.27)	8.50 (1.77)	9.71 (4.50)	10.71 (6.24)	12.57 (4.89)
	High VerPS	Young (N=12)	9.17 (2.13)	12.25 (1.86)	23.58 (9.89)	17.17 (1.59)	16.33 (1.67)
		Old (N=13)	7.38 (1.71)	9.46 (1.45)	13.85 (7.64)	12.85 (4.18)	14.08 (1.85)

Note. VerPS = Verbal Processing Speed

*Recall task N=15

**Recall task N=12

***Letter-Number Sequencing task N=8

Three-way between groups ANOVAs for Anxiety, Age, and Verbal Processing Speed were conducted (see Table 25).

Table 25

A summary of the three-way ANOVA results for five types of memory as a function of Anxiety, Age, and Verbal Processing Speed

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Anxiety	F	.38	.70	.60	.01	.84
	Sig	.54	.40	.44	.93	.36
	η^2_p	.00	.01	.01	.00	.01
	power	.09	.13	.12	.05	.15
Age	F	6.73	26.47	28.14	34.08	23.81
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.22	.24	.27	.21
	power	.73	1.00	1.00	1.00	1.00
VerPS	F	2.33	.33	.52	.82	1.42
	Sig	.13	.57	.47	.37	.24
	η^2_p	.03	.00	.01	.01	.02
	power	.33	.09	.11	.15	.22
Anxiety Age Interaction	F	.10	7.61	.00	.21	1.59
	Sig	.75	.01	.98	.65	.21
	η^2_p	.00	.08	.00	.00	.02
	power	.06	.78	.05	.07	.24
Anxiety VerPS Interaction	F	1.69	.13	2.24	1.62	2.09
	Sig	.20	.72	.14	.21	.15
	η^2_p	.02	.00	.02	.02	.02
	power	.25	.07	.32	.24	.30
Age VerPS Interaction	F	3.13	1.97	.46	.62	.06
	Sig	.08	.16	.50	.43	.80
	η^2_p	.03	.02	.01	.01	.00
	power	.42	.29	.10	.12	.06
Anxiety Age VerPS Interaction	F	.20	.01	.45	.22	.15
	Sig	.66	.94	.51	.64	.70
	η^2_p	.00	.00	.01	.00	.00
	power	.07	.05	.10	.08	.07

Note. VerPS = Verbal Processing Speed

As with the two-way ANOVAs, there were no significant main effects for Anxiety. The only two-way interaction with a noteworthy effect size (medium effect) was the Anxiety x Age interaction for the letter-number sequencing task, $F(1, 92) = 7.61, p = .007; \eta^2_p = .08$, which differed from the depression results presented in Table 11 (in which there was no Depression x Age interaction for this task). As can be seen from Table 24, the younger adults' performance on the letter-number sequencing task was best in the high anxiety group, while the older adults' performance was best for the low anxiety group. There were no significant three-way interactions, and no interactions with a notable effect size – results consistent with the three-way ANOVAs conducted for depression.

Summary

Verbal Processing Speed appears to have little impact on memory in combination with anxiety. However, there was a medium effect for the Anxiety x Age interaction in the letter-number sequencing task. There were no interactions between Anxiety and Verbal Processing Speed, and there were no notable three-way interactions between Anxiety, Verbal Processing Speed, and Age, consistent with the Depression analyses.

Anxiety and IQ

The third factor to be investigated in the three-way between groups ANOVAs for Anxiety was IQ. The means and SDs for this analysis are presented in Table 26, while the results of the ANOVA are presented in Table 27. There were no significant main effects for Anxiety (Table 27). The two-way interaction for Anxiety and Age for the letter-number sequencing task produced a moderate effect size, $F(1, 92) = 7.24, p = .008; \eta^2_p = .07$. While the younger adults with high anxiety showed greater letter-number sequencing ability than their lower anxiety counterparts, the older adults with high anxiety showed a reduced letter-number sequencing capacity when compared with their low anxiety group. This result diverged from the equivalent depression result, where there was no interaction.

Table 26

Means (SDs) for three-way ANOVAs for five types of memory as a function of Anxiety, Age, and IQ

			Memory Task					
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM	
Low Anxiety	Low	Young (N=13)	8.23 (1.69)	10.31 (1.32)	19.62 (9.81)	17.54 (1.20)	17.08 (1.32)	
	IQ	Old (N=15)	7.07 (2.02)	9.27 (1.67)	10.93 (4.38)	9.53 (6.16)	12.47 (3.94)	
	High	Young (N=13)	8.85 (2.12)	11.31 (1.97)	26.46 (14.19)	15.85 (3.00)	16.54 (1.85)	
	IQ	Old (N=14)*	7.83 (1.85)	10.29 (2.73)	15.43 (5.37)	13.14 (3.59)	13.79 (3.04)	
High Anxiety	Low	Young (N=13)	7.31 (2.14)	11.92 (2.63)	18.31 (7.11)	16.62 (2.22)	15.85 (2.23)	
	IQ	Old (N=10)**	7.60 (2.46)	9.00 (2.00)	9.60 (3.75)	10.00 (4.74)	13.60 (2.91)	
	High	Young (N=11)	9.18 (1.47)	12.82 (2.32)	25.36 (10.76)	16.09 (3.91)	15.09 (3.62)	
	IQ	Old (N=10)	7.00 (1.05)	9.20 (1.14)	15.20 (8.27)	14.20 (4.39)	13.50 (3.63)	

*Recall task N=12

**Letter-Number Sequencing task N=11

There was also one interaction of medium effect for Age and IQ, for the time-based prospective memory task, $F(1, 91) = 9.82, p = .002; \eta^2_p = .10$. The older adults with high anxiety had higher scores than the low anxiety groups when compared with others in their IQ group. However, the younger adults in the low IQ group showed decreased time-based prospective memory ability with high anxiety, while the high IQ younger adults showed increasing ability with high anxiety. There were no interactions of note between Anxiety and IQ, while in the depression results there were two interactions for Depression and IQ with the reading span and time-based prospective memory tasks that had both producing small effects. There were no three-way interactions for Anxiety x Age x IQ interaction, which was in line with the depression findings.

Table 27

A summary of the three-way ANOVA results for five types of memory as a function of Anxiety, Age, and IQ

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Anxiety	F	.32	1.14	.32	.07	.59
	Sig	.57	.29	.57	.79	.45
	η^2_p	.00	.01	.00	.00	.01
	power	.09	.18	.09	.06	.12
Age	F	6.78	26.74	30.54	36.07	22.02
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.23	.25	.28	.20
	power	.73	1.00	1.00	1.00	1.00
IQ	F	2.89	3.50	11.81	3.06	.00
	Sig	.09	.06	.001	.08	.98
	η^2_p	.03	.04	.12	.03	.00
	power	.39	.46	.93	.41	.05
Anxiety Age Interaction	F	.03	7.24	.02	.47	2.18
	Sig	.85	.01	.90	.49	.14
	η^2_p	.00	.07	.00	.01	.02
	power	.05	.76	.05	.11	.31
Anxiety IQ Interaction	F	.01	.31	.04	.30	.47
	Sig	.95	.58	.85	.58	.50
	η^2_p	.00	.00	.00	.00	.01
	power	.05	.09	.05	.09	.10
Age IQ Interaction	F	2.21	.17	.30	9.82	1.11
	Sig	.14	.69	.59	.002	.30
	η^2_p	.02	.00	.00	.10	.01
	power	.31	.07	.08	.87	.18
Anxiety Age IQ Interaction	F	2.82	.18	.02	.03	.25
	Sig	.10	.67	.90	.86	.62
	η^2_p	.03	.00	.00	.00	.00
	power	.38	.07	.05	.05	.08

Summary

Medium to large main effects for age were maintained across the working memory and prospective memory tasks. There was a main effect for IQ with

the reading span task, and a small effect for IQ with the letter-number sequencing task. This is consistent with the Depression analysis, although there was also a small effect for IQ and the recall task in the Depression analysis that was not observed in the Anxiety results above. There were also small effects for the interaction between Depression and IQ for the reading span and time-based prospective memory tasks.

Summary of the Anxiety and Moderator Analyses

The overall pattern of small interactions between Anxiety and the moderators was similar to the pattern seen in the Depression and moderator analyses. There were small effects for interactions with Visual Processing Speed, and Verbal Processing Speed with Anxiety. This differed slightly from the Depression analyses in which there were no interactions between Depression and Verbal Processing Speed, but there were two for the Depression and IQ analysis (for the reading span and time-based prospective memory tasks). The location of the interactions also differed between the Anxiety and the Depression analyses. An Anxiety interaction small effect was observed for Visual Processing Speed in the recall task (compared with the recall and reading span tasks in the Depression analysis). There was a medium effect for the interaction between Anxiety and Verbal Processing Speed in the letter-number sequencing task, as opposed to no interactions in the Depression analyses. As with the Depression results, there was one three-way interaction between Anxiety, Visual Processing Speed, and Age that had a small effect, although this was for the reading span task as opposed to the recall task in the Depression analysis.

Stress

As with Depression and Anxiety, two-way ANOVAs to investigate the impact of Stress and Age on memory function were conducted (see Table 29). The means and SDs for the two-way ANOVAs investigating Age and Stress can be seen below in Table 28. The younger adults had higher mean scores than the

older adults on all of the Memory tasks. The pattern between low stress and high stress was variable, with both age groups in the high stress group outperforming their age-peers in the Recall and Event-Based PM tasks.

Table 28

Mean (SDs) for all memory tasks as a function of Stress and Age

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Low Stress	Young (N=25)	8.28 (1.90)	10.84 (1.95)	23.44 (10.50)	16.84 (2.94)	16.16 (2.76)
	Old (N=25)*	7.09 (1.81)	9.68 (2.04)	12.04 (4.93)	11.84 (5.38)	13.20 (3.48)
High Stress	Young (N=25)	8.44 (2.06)	12.24 (2.31)	21.20 (11.65)	16.24 (2.47)	16.20 (2.00)
	Old (N=24)**	7.63 (1.97)	9.28 (2.03)	13.62 (6.86)	11.38 (4.97)	13.38 (3.35)

*Recall task N=23

**Letter-Number Sequencing task N=25

There were no main effects for Stress on the memory tasks, consistent with the depression and anxiety results. The main effect for Age in letter-number sequencing was qualified by another interaction between Stress and Age, $F(1, 96) = 4.65, p = .03; \eta^2_p = .05$. Higher letter-number sequencing scores occurred for the younger adults in the high stress group, with lower scores for the low stress group. The opposite was seen in the older adults where the scores were higher for the low stress group than for the high stress group. This result differed from the Depression and Age results presented in Table 5, but was consistent with the results presented in Table 8 and the Anxiety analysis from Table 15. A small interaction for the letter-number sequencing task was seen in the upper and lower quartile two-way ANOVA for Depression, as well as the two-way ANOVA for Anxiety, but not for the two-way ANOVA investigating Depression in the entire sample. There were no other notable two-way interactions.

Table 29

Summary of two-way ANOVA for Stress and Age

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Stress	F	.78	1.43	.03	.41	.03
	Sig	.38	.23	.86	.52	.86
	η^2_p	.01	.02	.00	.00	.00
	power	.14	.22	.05	.10	.05
Age	F	6.48	24.34	27.95	35.37	23.78
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.20	.23	.27	.20
	power	.71	1.00	1.00	1.00	1.00
Stress Age Interaction	F	.23	4.65	1.14	.01	.01
	Sig	.63	.03	.29	.94	.91
	η^2_p	.00	.05	.01	.00	.00
	power	.08	.57	.18	.05	.05

Summary

The results of the two-way ANOVA investigating the impact of Stress and Age on memory were similar to those of the two-way Anxiety ANOVA and the two-way ANOVA for the upper and lower quartiles in Depression (See Tables 8 and 21). There were no main effects for Stress. The effect sizes for Age were large for the working memory and prospective memory tasks, with a small effect for the recall task. The interaction between Stress and Age for the letter-number sequencing task produced a small effect, which was also seen in the Depression and Anxiety analyses.

Stress and Visual Processing Speed.

Three three-way between-groups ANOVAs were conducted to explore the impact of Visual Processing Speed, Verbal Processing Speed, and IQ individually for the Stress and Age variables on the different tests of memory. Table 30 shows the means and SDs for the Stress, Age, and Visual Processing Speed ANOVAs. There were no easily discernible patterns in this table except

for younger adults having higher mean scores in every group across the Memory tasks. The sole exception was for the low stress and high visual processing speed group for the Letter-Number Sequencing task, in which the scores between the two age groups were even.

Table 30

Means (SDs) for three-way ANOVAs for five types of memory as a function of Stress, Age, and Visual Processing Speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Stress	Low	Young (N=12)	7.67 (1.50)	11.50 (2.39)	20.08 (7.94)	16.25 (3.96)	15.33 (3.55)
	VisPS	Old (N=13)*	6.83 (1.75)	9.15 (1.63)	10.62 (3.62)	10.92 (5.53)	12.54 (3.76)
	High	Young (N=13)	8.85 (2.11)	10.23 (1.24)	26.54 (11.89)	17.38 (1.50)	16.92 (1.55)
	VisPS	Old (N=12)**	7.36 (1.91)	10.25 (2.34)	13.58 (5.81)	12.83 (5.27)	13.92 (3.15)
High Stress	Low	Young (N=13)	8.08 (2.40)	12.69 (2.72)	23.15 (12.65)	16.00 (2.00)	16.15 (2.23)
	VisPS	Old (N=11)***	6.55 (1.75)	8.58 (1.83)	15.55 (7.41)	10.18 (6.08)	12.55 (3.08)
	High	Young (N=12)	8.83 (1.64)	11.75 (1.76)	19.08 (10.59)	16.50 (2.97)	16.25 (1.82)
	VisPS	Old (N=13)	8.54 (1.71)	9.92 (2.06)	12.00 (6.18)	12.38 (3.75)	14.08 (3.52)

Note. VisPS = Visual Processing Speed

*Recall task N=12

**Recall task N=11

***Letter-Number Sequencing task N=12

The first set of three-way ANOVAs is presented in Table 31, showing the effect of Stress, Age, and Visual Processing Speed on the various types of memory. There were no main effects for Stress, but several of the two-way interactions produced small effect sizes. The main effect for Age and letter-number sequencing was qualified by a small effect Stress $\times$ Age interaction, $F(1, 92) = 4.87, p = .03; \eta^2_p = .05$. In this interaction, the younger adults in the high stress group obtained higher scores on the letter-number sequencing task than did the younger adults in the low stress task. The older adults exhibited the reverse pattern in which performance in the high stress group was worse than for those in the low stress group. There was a similar interaction for the Anxiety results. The other two-way interaction of note was for Stress and Visual Processing Speed for the reading span task, $F(1, 91) = 5.76, p = .018; \eta^2_p = .06$. Across both age groups, those in the low visual processing speed, high stress groups

outperformed those in the low visual processing speed, low stress groups. Furthermore, those in the high visual processing speed, low stress groups outperformed those in the high visual processing speed, high stress groups. A similar interaction was seen for Depression and Visual Processing Speed for the reading span task. None of the three-way interactions between Stress, Age, and Visual Processing Speed produced any effects.

Table 31

A summary of the three-way ANOVA results for five types of memory as a function of Stress, Age, and Visual Processing Speed

		Memory Task				
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Stress	F	.71	1.23	.02	.49	.02
	Sig	.40	.27	.88	.49	.89
	η^2_p	.01	.01	.00	.01	.00
	power	.13	.20	.05	.10	.05
Age	F	7.36	25.51	27.32	35.40	23.97
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.08	.22	.23	.28	.21
	power	.77	1.00	1.00	1.00	1.00
VisPS	F	8.53	.02	.07	2.98	3.77
	Sig	.004	.89	.80	.09	.06
	η^2_p	.09	.00	.00	.03	.04
	power	.82	.05	.06	.40	.49
Stress Age Interaction	F	.10	4.87	1.19	.00	.00
	Sig	.75	.03	.28	.99	.99
	η^2_p	.00	.05	.01	.00	.00
	power	.06	.59	.19	.05	.05
Stress VisPS Interaction	F	.46	.12	5.76	.01	.32
	Sig	.50	.73	.02	.92	.57
	η^2_p	.01	.00	.06	.00	.00
	power	.10	.06	.66	.05	.09
Age VisPS Interaction	F	.15	8.07	.17	.55	.27
	Sig	.70	.01	.68	.46	.61
	η^2_p	.00	.08	.00	.01	.00
	power	.07	.80	.07	.11	.08
Stress Age VisPS Interaction	F	1.53	.00	.32	.08	.49
	Sig	.22	.96	.57	.78	.49
	η^2_p	.02	.00	.00	.00	.01
	power	.23	.05	.09	.06	.11

Note. VisPS = Visual Processing Speed

Summary

The three-way Stress, Age, and Visual Processing Speed ANOVA shared similarities and differences with both the Depression and Anxiety analyses, and appeared to lie somewhere in between the two. There was a small effect for the Stress x Age interaction for the letter-number sequencing task which was also present in the Anxiety analysis but not for the Depression analysis. There was also a small effect for the Stress x Visual Processing Speed interaction for the reading span task which was present for Depression, but not for Anxiety. Interestingly, both the Depression and Anxiety results included a small effect for their interactions with Visual Processing Speed for the recall task, but this was absent in the Stress results. There were no three-way interactions between Stress, Age, and Visual Processing Speed, although there was one such interaction for Depression in the recall task, and one for Anxiety in the reading span task.

Stress and Verbal Processing Speed

The impact of Stress and Verbal Processing Speed on memory was also investigated through the use of three-way ANOVAs. The means and SDs are presented in Table 32. The results for the Stress and Verbal Processing Speed groupings were variable with those in the high stress group outperforming the low stress group at times. The younger adults had consistently higher mean scores with the exception of those in the high stress and low verbal processing speed group for the Recall task in which both age groups had the same mean score.

Table 32

Means (SDs) for three-way ANOVAs for five types of memory as a function of Stress, Age, and Verbal Processing Speed

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Stress	Low VerPS	Young (N=13)	7.46 (1.56)	11.23 (2.31)	21.85 (10.79)	16.38 (3.82)	15.69 (3.45)
		Old (N=13)*	7.00 (1.71)	9.38 (2.06)	11.31 (4.97)	11.62 (5.58)	12.62 (3.69)
	High VerPS	Young (N=12)	9.17 (1.90)	10.42 (1.44)	25.17 (10.37)	17.33 (1.56)	16.67 (1.78)
High Stress	Low VerPS	Old (N=12)**	7.18 (1.99)	10.00 (2.04)	12.83 (4.97)	12.08 (5.40)	13.83 (3.27)
		Young (N=12)	8.00 (2.09)	12.25 (2.90)	22.92 (13.58)	16.67 (1.78)	16.17 (2.17)
	High VerPS	Old (N=10)***	8.00 (2.11)	8.82 (2.56)	12.30 (6.34)	9.90 (4.73)	13.20 (4.26)
			Young (N=13)	12.23 (1.74)	19.62 (9.83)	15.85 (3.00)	16.23 (1.92)
			Old (N=14)	9.64 (1.50)	14.57 (7.28)	12.43 (5.03)	13.50 (2.68)

Note. VerPS = Verbal Processing Speed

*Recall task N=12

**Recall task N=11

***Letter-Number Sequencing task N=11

A three-way ANOVA conducted to investigate the impact of verbal processing speed, stress, and age on the memory tasks (see Table 33) showed a similar pattern to the other analyses already presented. In this table, it can be seen that none of the main effects for Stress, Age, or Verbal Processing Speed produced any noteworthy effect sizes. There was a small effect size for the two-way interaction for Stress and Age with the letter-number sequencing task, $F(1, 92) = 4.95$, $p = .03$; $\eta^2_p = .05$. In this interaction, the performance of the younger adults was better in the high stress group when compared with the low stress group, while the older adults' performance was worse in the high stress group when compared with the low stress group. This interaction was not evident for the Depression variable. There were no two-way interactions between Stress and Verbal Processing Speed, consistent with the Depression results, and there were no three-way interactions.

Table 33

A summary of the three-way ANOVA results for five types of memory as a function of Stress, Age, and Verbal Processing Speed

			Memory Task			
Group		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Stress	F	.80	1.28	.06	.59	.01
	Sig	.37	.26	.81	.44	.91
	η^2_p	.01	.01	.00	.01	.00
	power	.14	.20	.06	.12	.05
Age	F	6.36	24.07	28.04	36.31	23.17
	Sig	.01	<.001	<.001	<.001	<.001
	η^2_p	.07	.21	.24	.29	.20
	power	.70	1.00	1.00	1.00	1.00
VerPS	F	1.79	.13	.28	.87	1.12
	Sig	.18	.72	.60	.35	.29
	η^2_p	.02	.00	.00	.01	.01
	power	.26	.07	.08	.15	.18
Stress Age Interaction	F	.38	4.95	.98	.00	.01
	Sig	.54	.03	.32	.96	.93
	η^2_p	.00	.05	.01	.00	.00
	power	.09	.60	.17	.05	.05
Stress VerPS Interaction	F	1.16	.35	.65	.01	.58
	Sig	.28	.55	.42	.93	.45
	η^2_p	.01	.00	.01	.00	.01
	power	.19	.09	.13	.05	.12
Age VerPS Interaction	F	3.73	1.81	.27	.73	.04
	Sig	.06	.18	.61	.40	.84
	η^2_p	.04	.02	.00	.01	.00
	power	.48	.27	.08	.14	.05
Stress Age VerPS Interaction	F	.00	.12	1.03	1.31	.00
	Sig	.98	.73	.31	.26	1.00
	η^2_p	.00	.00	.01	.01	.00
	power	.05	.06	.17	.20	.05

Note. VerPS = Verbal Processing Speed

Summary

The Stress ANOVA results presented above in Table 33 mirror those seen in the Depression and Anxiety analyses with Verbal Processing Speed. These results shared a small effect for an interaction between Stress and Age for the letter-

number sequencing task with the Anxiety analyses. They also shared a small effect for the Age x Verbal Processing Speed interaction with the Depression analysis. As with the Depression and Anxiety analyses, there were no trends indicating interactions between Stress and Verbal Processing Speed.

Stress and IQ

Finally, the impact of Stress in combination with IQ was investigated. The same variable pattern (as seen in the Depression and Anxiety analyses) was present in the means and SDs (see Table 34), with the younger adults generally having higher mean scores than the older adults. Interestingly, the older adults marginally outperformed the younger adults in the high stress, low IQ group for the Recall task.

Table 34

Means (SDs) for three-way ANOVAs for five types of memory as a function of Stress, Age, and IQ

			Memory Task				
Group			Recall	Letter- Number Sequencing	Reading Span	Time-Based PM	Event- Based PM
Low Stress	Low IQ	Young (N=15)	8.13 (1.85)	10.67 (1.80)	20.33 (8.85)	17.33 (1.80)	16.80 (1.82)
		Old (N=11)	6.82 (2.14)	9.27 (1.56)	9.82 (3.92)	9.55 (6.01)	12.45 (3.88)
	High IQ	Young (N=10)	8.50 (2.07)	11.10 (2.23)	28.10 (11.49)	16.10 (4.12)	15.20 (3.68)
		Old (N=14)*	7.33 (1.50)	10.00 (2.35)	13.79 (5.06)	13.64 (4.22)	13.79 (3.14)
High Stress	Low IQ	Young (N=11)	7.27 (2.05)	11.73 (2.61)	17.09 (7.80)	16.73 (1.85)	16.00 (2.00)
		Old (N=14)**	7.64 (2.21)	9.07 (1.98)	10.86 (4.35)	9.86 (5.36)	13.29 (3.36)
	High IQ	Young (N=14)	9.36 (1.60)	12.64 (2.06)	24.43 (13.35)	15.86 (2.88)	16.36 (2.06)
		Old (N=10)	7.60 (1.71)	9.60 (2.17)	17.50 (8.02)	13.50 (3.60)	13.50 (3.50)

*Recall task N=12

**Letter-Number Sequencing task N=15

As can be seen from Table 35, there were no main effects for Stress. However, there was a main effect for IQ for the reading span task, consistent with the Depression and Anxiety analyses, $F(1, 91) = 13.92, p = <.001; \eta^2_p = .13$.

Table 35

A summary of the three-way ANOVA results for five types of memory as a function of Stress, Age, and IQ

Group		Memory Task				
		Recall	Letter- Number Sequencing	Reading Span	Time- Based PM	Event- Based PM
Stress	F	.49	1.37	.10	.04	.14
	Sig	.49	.24	.76	.83	.71
	η^2_p	.01	.02	.00	.00	.00
	power	.11	.21	.06	.06	.07
Age	F	6.14	23.08	30.39	36.60	22.05
	Sig	.02	<.001	<.001	<.001	<.001
	η^2_p	.07	.20	.25	.29	.20
	power	.69	1.00	1.00	1.00	1.00
IQ	F	3.51	2.34	13.92	3.07	.02
	Sig	.06	.13	<.001	.08	.90
	η^2_p	.04	.03	.13	.03	.00
	power	.46	.33	.96	.41	.05
Stress Age Interaction	F	.49	3.54	2.87	.10	.01
	Sig	.49	.06	.09	.75	.94
	η^2_p	.01	.04	.03	.00	.00
	power	.11	.46	.39	.06	.05
Stress IQ Interaction	F	.55	.03	.11	.00	.12
	Sig	.46	.87	.75	.98	.73
	η^2_p	.01	.00	.00	.00	.00
	power	.11	.05	.06	.05	.06
Age IQ Interaction	F	1.61	.00	.43	9.35	1.34
	Sig	.21	.96	.52	.003	.25
	η^2_p	.02	.00	.01	.09	.01
	power	.24	.05	.10	.86	.21
Stress Age IQ Interaction	F	2.13	.16	.20	.07	1.62
	Sig	.15	.69	.65	.80	.21
	η^2_p	.02	.00	.00	.00	.02
	power	.30	.07	.07	.06	.24

Looking at the two-way interactions, the Stress $\times$ Age interaction for the letter-number sequencing task produced a small effect accounting for about 4% of the variance, $F(1, 92) = 3.54$, $p = .06$; $\eta^2_p = .04$. This small effect size revealed that letter-number sequencing scores tended to be greater for the younger adults in

the high stress condition when compared with the low stress condition, but was worse for the older adults in the high stress condition when compared with the low stress condition. There were no interactions for Stress and IQ for any of the memory tasks. This was consistent with the Anxiety variable but not for the Depression variable, where the interaction for Depression and IQ neared significance for the reading span and time-based prospective memory tasks. There were no three-way interactions.

Summary

There were small to medium main effects for IQ in the retrospective memory tasks of this analysis. These results shared a small main effect for IQ in the recall task with the Depression results, and a medium main effect for IQ in the reading span task with Anxiety. There was also a small effect for IQ in the letter-number sequencing task that was present for Depression and Anxiety, but not for Stress. There was a small Stress x Age interaction effect for the letter-number sequencing task, consistent with the results presented in Table 29. There were no Stress x IQ interactions. The medium effect for the interaction between Age and IQ for the time-based prospective memory task was consistent with the Depression and Anxiety analyses.

Summary of the Stress and Moderator Analyses.

The Stress analyses followed the same general pattern as the Depression and Anxiety analyses, although there were some important differences. The main effects for Age were significant for the working memory and prospective memory tasks, but not for recall. There was a small effect for the Stress by Visual Processing Speed interaction for the reading span task, similar to that seen in the Depression analyses. There was no Stress by Visual Processing Speed interaction for the recall task, as there was for Depression and Anxiety. There were no notable interactions between Stress and Verbal Processing Speed, consistent with the Depression and Anxiety analyses. There were no

Stress x IQ interactions in contrast to the two small effects for the Depression x IQ interactions for the reading span and time-based prospective memory tasks.

Chapter Seven

Discussion

This chapter presents a discussion of the results from the present study, and compares the results to the predictions made as well as with previous research. The chapter starts by reviewing the aims and results of the study, and then discusses the analyses for Depression, Anxiety and Stress. A general discussion is then presented which integrates the different analyses by memory type. The implications of the study are discussed. Finally, the limitations of the study and future research directions are explored.

Summary of aims and predictions

The aims for the present study were categorised by type of memory. There is inconsistency in past research on the impact of depression and late-life depression on short-term memory in particular. Using a free recall word list task, the current study aimed to provide further information on STM and its relation to depression. It was predicted that STM would be affected by depression, and there would be a main effect for Depression on the recall task.

Working memory is affected by depression in later life, and it is thought the central executive plays a large role in this. Two different working memory tasks were used. It was expected depression would have a marked effect on the working memory tasks. The other primary aim of investigating working memory in the present study was to compare any depression-related memory impairment in younger and older samples, with a specific focus on the impact of the ageing process.

Very little research on the relationship between depression and prospective memory has been conducted. In the one study of prospective memory and late-life depressive symptomatology, no relationship was found using an event-based task. The present study used both a time-based task and an event-based task, with the expectation the time-based task would be affected by both depression and ageing. It was expected the event-based prospective memory task would remain unaffected by depression or age.

Summary of results

The initial planned comparisons did not reveal any effects for Depression on any of the Memory tasks, or any Depression x Age interactions. When the upper and lower quartiles for depression scores were used, a similar pattern of results emerged. There was a small but notable effect size for Depression and time-based prospective memory. There was also a small to medium main effect for the Depression x Age interaction on working memory for the letter-number sequencing task. Interestingly, the younger adults in the high depression group outperformed their low depression counterparts, while the reverse pattern was seen for the older adults.

Some interesting patterns were revealed in the moderator variables analyses. Once Visual Processing Speed was added to the analysis, small effect sizes were seen for the Depression x Visual Processing Speed interactions for recall and reading span (working memory task), as well as a small effect size for the three way Depression x Age x Visual Processing Speed interaction for recall. In this sample, the younger adults in the high Visual Processing Speed group produced higher memory scores compared to those in the low Visual Processing Speed group regardless of high or low depression grouping. On the other hand, the older adults in the low depression group maintained an approximately even performance on the recall task irrespective of Visual Processing Speed ability, whereas the older adults in the high depression and high Visual Processing Speed group outperformed the high depression and low

Visual Processing Speed group. The analysis of the other potential moderator, IQ, revealed the most interesting results. There was a small effect size for the Depression x IQ interaction for the reading span task, and a medium effect size for the time-based prospective memory task.

For the anxiety analyses, there were no significant main effects for Anxiety in the two-way ANOVAs. There was an Age x Anxiety interaction with a small effect size for the letter-number sequencing task. In the three-way ANOVAs investigating the impact of Visual Processing Speed, there was a small interaction for Anxiety x Visual processing Speed for the recall task. There was also a small effect size for the three-way Anxiety x Age x Visual Processing Speed interaction for the reading span task. As can be seen in Figure 1, this effect is mostly located in the Anxiety x Visual Processing Speed interaction for the older adults. For the younger adults, high anxiety resulted in better performance by the high visual processing speed group but poorer performance for the low visual processing speed group. For the older adults, reading span performance deteriorated with higher visual processing speed for the high anxiety group, but improved with higher visual processing speed for the low anxiety group.

The two-way ANOVAs for Stress revealed no main effects. However, there was a small effect size for the interaction between Stress and Age for the letter-number sequencing task. There was also a Stress x Visual Processing Speed interaction for the reading span task, which produced a small effect size. Those in the low stress high visual processing speed groups outperformed those in the high stress high visual processing speed groups.

Depression

Short-Term Memory

The aim of investigating STM was to provide further clarification on potential STM impairment and depressive symptomatology in a community-based

sample of older adults compared to a younger group. It was predicted there would be a main effect for depression, with poorer performance on the recall task associated with higher scores on the depression measure. The results for STM did not support this prediction, as there was no effect for Depression on the recall task. These results are generally inconsistent with previous research focussing on clinically depressed samples. The meta-analysis conducted by Kindermann and Brown (1997) found an overall depression-related impairment in immediate recall for older adults. More recently, Elderkin-Thompson et al. (2007) found STM impairment in older adults with major *and* minor depression. Research from community-based samples, such as the one used in the present study, are less conclusive (Comijs et al., 2001; Ganguli et al., 2006; R. S. Wilson et al., 2004). No other studies have found any better performance associated with depression as was found in the present study.

Working Memory

As with the results obtained for the recall task, there were some surprising findings with regard to working memory. Some of these results were expected; however, other findings were quite unexpected, although they produced only small effects. The expectations that there would be main effects for Depression in both working memory tasks were not supported by the present study. There was a small effect size for the Depression x Age interaction in the letter-number sequencing task, but not for the reading span task. Interestingly, this small interaction persisted over the analyses of depression, anxiety, and stress. This Depression x Age interaction was actually due to an increase in younger adults' letter-number sequencing scores in the high depression group when compared with the low depression group. So, while the younger adults had the best performance in the high depression group, the older adults in the high depression group showed a small deterioration in performance.

The literature previously reviewed supports the finding of a slight difference in performance between the low and high depression groups for the older adults

through the double impact of the ageing process and the effects of depression on cognitive processes. Yet the literature does not explain why the younger adults in the high depression group outperformed all other participants in the current study. A previous study by Thomas, Goudemand, and Rousseaux (1999) found that performance in working memory tasks for people with depression improved, when compared with healthy controls, as task difficulty increased. Task difficulty was increased through the use of a dual task paradigm. When task difficulty was increased using a decision making task, the performance of those with depression was inferior to the controls. They attributed the superiority of those with depression completing the dual task to the more demanding task occupying all cognitive resources and interrupting the rumination that was thought to be the cause of impairment. This may explain why there was a trend for the improvement in younger adults in the high depression group in the letter-number sequencing task but not the reading span task. The reading span task is thought to require greater cognitive resources and also contains a decision making component. The meaning of this for the ageing process remains unclear.

Christopher and MacDonald (2005) also found their depressed participants' performance improved on their working memory task when task complexity increased. They attributed this to reallocation of attentional resources away from rumination. It may be that the attentional load of the working memory task in the present study allowed those in the high depression group to allocate resources away from rumination. At this stage, this is merely speculation given that neither attention nor rumination was measured. Furthermore, this prediction does not explain why those in the high depression group outperformed their low depression counterparts, rather than there being no difference between these two groups.

Because the effects of depression on working memory are small, it would be unwise to draw any conclusions from the present results. Replication is necessary, as is the use of different depression and working memory tasks.

Different scales do not always yield the same results (Beck et al., 1988; Bieling et al., 1998; Conway et al., 2002; Fydrich et al., 1992). It needs to be ascertained that the study findings are not unique to a particular test of depression or working memory.

Prospective Memory

The aim of studying prospective memory in this study was to investigate its component types (time-based and event-based) and to look at prospective memory in a sample of older adults with depressive symptoms. It was expected there would be a main effect for Depression in the time-based but not the event-based prospective memory task. This expectation was based on previous research indicating that time-based prospective memory is most at risk of depression-related impairment because of its reliance on self-initiated processes, and because of a need for allocation of high attentional resources (already shown to suffer in depression).

Depression had no effect on the event-based prospective memory tasks, as predicted. Previous studies have also found that depression does not impact event-based prospective memory. Harris and Menzies (1999) studied depressed mood and event-based prospective memory in a non-clinical sample and found no significant relationship. Livner et al. (2008) researched late-life depression and found their simple event-based task was not related to depression. It is thought event-based tasks require fewer attentional resources because the cue to remember the task is external, and does not rely on internal monitoring to the same extent as time-based tasks (McDaniel & Einstein, 2007). The lack of findings on the impact of depression on event-based prospective memory seems to indicate that such tasks do not exceed the processing resources available in depression.

There was a small effect for Depression on time-based prospective memory in the quartile split analysis, consistent with the prediction. The performance of those in the high depression groups was poorer than those in the low

depression groups, independent of age. The literature on the impact of depression on prospective memory is scant for young to middle-aged participants, and almost non-existent for studies of late-life depression. The small effect for Depression is supported by research by Rude et al. (1999), who found time-based prospective memory in their sample of participants with major depression was worse than that of the non-depressed participants. There was no Depression x Age interaction for time-based prospective memory. This outcome was inconsistent with the present study's prediction that older adults in the high depression group would be the most impaired in time-based prospective memory. The effects of depression on time-based prospective memory were small and replication is necessary to explore the impact of depression on prospective memory further.

Memory Moderator analysis

Processing speed and intelligence are known to be strongly correlated with memory (Conway et al., 2002; Conway et al., 2003; Salthouse, 1996), and so it was thought prudent to investigate these factors as possible moderators. It was predicted there would be some main effects for processing speed and IQ in this sample, but this was not a focus of the research as it has been thoroughly covered in the above studies. Despite the moderating impact of processing speed and IQ on memory (working memory in particular), being well established, very few studies investigating depression and memory have taken these moderators into account. Given the literature on the relationships between depression, memory, and these potential moderators is sparse, no predictions were made regarding their effects on memory in the current study.

Processing Speed

There was a Depression x Age x Visual Processing Speed interaction for the recall task. The majority of this interaction was located in the Depression x Visual Processing Speed interaction for the older adults. In this small

interaction, the older adults in the low depression group maintained a reasonably stable level of performance on the recall task. On the other hand, the older adults in the high depression high visual processing speed group dramatically outperformed those in the high depression and low visual processing speed group (refer to Figure 1).

Comparisons of these results with previous research is not possible, as no studies were found that investigated relationships between STM, depression, and processing speed. Ilsley et al. (1995) measured psychomotor speed along with memory in a sample of adults with major depression. Psychomotor speed and reaction time are often used as measures of processing speed (Salthouse, 2000). While Ilsley et al. found a significant difference between the depressed and non-depressed groups on the measure of psychomotor speed, they did not find any significant differences between the two groups on STM performance. A similar study also found no significant difference between depressed and control participants on measures of STM (Austin et al., 1999). They measured reaction time and psychomotor speed, and found that reaction time (but not psychomotor speed) was significantly affected by depression. Because neither of these studies found that STM was affected, they did not perform any further analyses to look at the relationships between STM, depression, and processing speed in their samples. Also, the above studies used adult (as opposed to older adult) samples. The majority of the Depression x Age x Visual Processing Speed interaction was located in the older adults. There is no literature that helps to explain why the combination of high visual processing speed and high depression had such a positive effect on the recall task in the older adults. It is fascinating that the two high depression and high visual processing speed groups have the highest level of recall. It is interesting to speculate that high visual processing speed may be protective in any recall difficulties associated with late-life depression. This is especially so, given the opposite trend of low visual processing speed being more beneficial in the reading span task of working memory. More research is needed to focus on the factors involved in

the better identified short-term memory performance, especially with regard to older adults and the influence of visual processing speed.

The reading span task was the only working memory measure that was affected by a Depression $\times$ Visual Processing Speed interaction. Regardless of age, performance on the reading span task was better in the high visual processing speed groups for both low depression and low stress, as compared to the low visual processing speed groups. However, the reverse was seen for the high depression and high stress groups, with performance in the high visual processing speed group being worse than that in the low visual processing speed group. Interestingly, the higher visual processing speed appears to be disadvantageous for those in the high depression and high stress groups. It is difficult to explain these small effects due partly to the lack of previous research on processing speed and memory in combination with depression. Nebes et al. (2000) found that processing resources (processing speed and working memory) mediated depression-related change on other cognitive tasks for older adults. They also found processing speed contributed to the considerable amount of variance in the depression and working memory relationship, but noted a significant amount continued to be explained by working memory once processing speed was controlled for. Under this relationship, processing speed and working memory both deteriorate with depression. Such a linear explanation does not support the results found in the present study in which poorer performance in working memory was found in those with a higher level of visual processing speed in the high depression and high stress groups.

In contrast to Visual Processing Speed, Verbal Processing Speed had little effect, an unexpected outcome. All of the tasks had a verbal component, and verbal working memory is supposed to be maintained in the phonological loop. Thus, it was expected that Verbal Processing Speed would at least have some effect on the memory scores. However, there were no main effects for Verbal Processing Speed, and no interactions with depression.

Intelligence

In the IQ analysis, there were two noteworthy interactions between Depression and IQ. Both of these interactions were with memory types that were anticipated to be subject to the greatest impairment in depression, working memory and time-based prospective memory. Not all of the findings lie in the predicted directions, however. Firstly, there was a small Depression $\times$ IQ interaction for the working memory task of reading span. Overall, the scores across all groups improved with higher IQ. The greatest difference in performance was in the high depression group for both ages. Those in the high depression and high IQ groups outperformed those in the low depression high IQ groups (as well as all low IQ groups) when compared with their own age groups.

As with the Visual Processing Speed results, a couple of studies have compared depressed and non-depressed samples on aspects of intelligence, but have not taken the next logical step of investigating IQ as a moderator of working memory ability in depression. Christopher and MacDonald (2005) measured verbal IQ through the use of the Vocabulary subtest of the Revised Wechsler Adult Intelligence Scale (WAIS-R) and found no significant difference in scores between their depressed and non-depressed participants. They did find differences between the groups in working memory, but they did not investigate the effect IQ may have had on the working memory differences. Rose and Ebmeier (2006) used the *n*-back task to research depression-related working memory impairment. They used the National Adult Reading Test (NART) to estimate IQ. They found that, while IQ was a mediator for working memory across both the depressed and non-depressed groups, it did not contribute to the depression-related working memory impairment observed in their sample. These results are difficult to interpret given that Rose and Ebmeier should not have used ANCOVA to analyse their results, because of the inability to randomly assign depression or IQ. There is little in previous research to account for the combination of high IQ and high depression having better

working memory performance found in the present study. Even if this is related to a higher IQ being associated with greater processing and capacity resources that are better able to manage the cognitive symptoms of depression and still attend to memory tasks, this still does not explain the superior performance seen in those with the higher depression scores.

Secondly, there was a medium-sized interaction between Depression and IQ for the time-based prospective memory task. The pattern revealed in this analysis is that for time-based prospective memory, those in the lower IQ groups, irrespective of age, exhibited a decrease in task performance in the high depression group. Those in the high IQ groups showed a slight increase in performance with higher depression. The result is consistent with attentional resource and inhibitory control models of depression-related decline as intelligence relies on attention and cognitive capacity. Those with lower IQs are assumed to have fewer processing resources to rely on. These resources may become taxed by rumination in depression to the point of interfering with memory. Those in the higher IQ group may have had extra processing resources at their disposal and the memory task may not have reached their resource threshold.

Comparison of Depression, Anxiety, and Stress

The measures of depression, anxiety, and stress were all strongly related in the present study, sharing approximately 40% of the variance.

Similar to the Depression and Age two-way analysis, there was a small Anxiety x Age interaction with working memory in the letter-number sequencing task. Younger adults in the high anxiety group outperformed their low anxiety counterparts, while the older adults exhibited a slight impairment in performance in the high anxiety group. This pattern persisted into the Stress analysis, in which there was a small effect for the Stress x Age interaction.

While two-way interactions for the letter-number sequencing task were expected, the direction of the effects for the younger adults was not. The older adults showed poorer performance with higher depression, anxiety, and stress levels, whereas the younger adults showed relatively marked improvement in their scores. Previous research on anxiety and working memory found that increasing anxiety was associated with poorer working memory performance. Christopher and MacDonald (2005) also accounted for anxiety (using participants with Generalised Anxiety Disorder) in their research on the impact of depression on the components of working memory. They found their anxiety group performed similarly to their control group on working memory slave system tasks (involving the phonological loop and visuospatial sketchpad), while the depressed participants performed more poorly. When the central executive component of working memory was measured, both the anxious and depressed groups had impaired performance compared with the control participants. This is not consistent with the results of the present study which showed anxiety-related improvement in working memory performance for the younger adults. Rose and Ebmeier (2006) also measured both anxiety and state stress in their study of working memory in depression. Unfortunately, they did not present results of the influence of either stress or anxiety on their working memory task.

Qin, Hermans, van Marle, Luo, and Fernández (2009) investigated the effects of stress on working memory in a sample of young women. They used a stress induction paradigm in which an experimental group were exposed to violent video clips, while a control group watched neutrally themed clips. They used an *n*-back task towards the end of the videos to measure working memory, and also calculated brain activation during the task through the use of fMRI. Subjective stress was measured with the Positive and Negative Affect Scales (PANAS). Scores on the PANAS were different between the stress and neutral conditions, and indicated that the stress induction technique had been effective. They found no main effect for experimental condition on the *n*-back task, indicating stress did not have an impact on working memory as they had

predicted. Although, they did find stress was associated with a reduction in activity in the pre-frontal cortex which is known as an area of importance for working memory. They propose their results provide support for cognitive resources being allocated elsewhere, as well as to the memory task. The findings of this study are inconsistent with the results of the present study, in which young participants in the high stress scores outperformed their low stress counterparts.

Other studies have found stress-related impairment in working memory. The Stress x Age interaction in the present study identifies older adults had a slight stress-related impairment, yet, as noted above, the younger adults had stress-related enhancement of letter-number sequencing performance. A study of working memory and stress in young men also made use of the PANAS and *n*-back task (Schoofs, Preu, & Wolf, 2008). As with Qin et al. (2009), they used a stress induction paradigm. This required participants in the stress condition to perform a speech in front of a panel of judges, and to complete a mathematical task while being observed by the panel (the control condition did the same tasks alone in a room). They found that stressed participants had slower reaction times when responding to the *n*-back task probes. The *n*-back task was presented in blocks consisting of 24 trials, and rotating between a 2-back and a 3-back block. They found the stressed participants had poorer performance on the first block of each *n*-back task level they completed, but the difference was no longer significant on subsequent blocks. Again, this is in conflict with the results of the present study in which the high stress younger adults had superior performance on the letter-number sequencing task of working memory.

In the present investigation, interactions were also seen in the three-way analyses of Anxiety and Stress with Visual Processing Speed. The profiles in these three-way analyses had some similarities and some differences when compared with the Depression analyses. Firstly, there was a small Anxiety x Visual Processing Speed interaction for the recall task. In this interaction,

participants in the high anxiety groups had better recall performance with increasing visual processing speed, whereas those in the low anxiety groups had relatively stable recall performance across the visual processing speed groups. This is similar to the Depression results which also interacted with Age, in which the older adults in the high depression group showed a marked increase in recall performance in the high visual processing speed group. There was an expectation there would be a main effect for Visual Processing Speed with the recall task, but not that it would interact with Depression or Anxiety. As with depression, no research was found which has investigated processing speed as a mediator of short-term memory function and anxiety.

There was a medium effect for the Stress x Visual Processing Speed interaction for the reading span task, and a small Depression x Visual Processing Speed interaction on this same task. A similar pattern was observed in both of these interactions in which reading span task performance increased with higher visual processing speed for those in the low depression and low stress groups. At the same time, performance decreased with higher visual processing speed for those in the high depression and high stress groups. This effect would not make sense in traditional processing speed studies in which higher processing speed scores are associated with better working memory performance, especially in ageing (Salthouse, 1991, 2000).

Research on stress, working memory, and processing speed in older adults (aged 65-95) was conducted by Stawski, Sliwinski, and Smyth (2006). They used confirmatory factor analysis to investigate the role of perceived stress and cognitive interference (intrusive thoughts and rumination) from stress on working memory and processing speed performance. They found that both measures of working memory used (variations of an operation span task and a 2-back task) and the measure of cognitive interference were related, but that working memory was not related to perceived stress. They also found cognitive interference was negatively related to their measures of processing speed (a number matching task and a serial counting task). They entered

perceived stress, age, depression (CES-D score) and cognitive interference into their model and found only age and cognitive interference made unique contributions to performance on the cognitive tasks (working memory, processing speed, and recall). They propose that cognitive interference from stress affects cognitive tasks such as working memory and processing speed because fewer attentional resources are available to be allocated to the tasks. It is curious to note the measure of perceived stress was not related to working memory performance in the Stawski et al. study, yet cognitive interference from stress was. In the present study, only perceived stress was measured, and not rumination or cognitive interference. While this Stawski et al. study does take processing speed into account, it does not investigate it as a mediating factor for the effects of stress on working memory. This makes it difficult to compare with the results of the present study, although it does provide support for stress having an impact on processing speed. The above study supports the general trend of a stress-related decline in working memory performance in the letter-number sequencing task observed in the present study. It also supports stress-related impairment of processing speed (through stress-related cognitive interference).

It may be that the deterioration in performance seen in the high depression and stress groups with increasing visual processing speed are due to those with higher processing speeds having greater difficulty in inhibiting competing information. With higher visual processing speed, they may be taking in more extraneous information (either from the external environment, the task itself, or from their own internal ruminative thoughts), and this in turn may have a greater impact on their reading span performance. Those with higher visual processing speed may have comparatively more difficulty filtering out competing stimuli, and greater difficulty allocating full attention to the memory task. This is conjecture at this stage; further experimentation is required to explore the visual processing speed, depression, and stress links on working memory.

Implications

The implications of the present research suggest there may be other factors associated with memory that are impacting on relationships between depression and memory for both younger and older adults. The advantage of studying both age-groups together is the differing effects of depression on memory can be observed and compared. There were trends towards age-based interactions for some tasks in which factors such as depression, anxiety, stress, and visual processing speed had different effects for the younger and older adults. Research focussing on these differences is important as it cannot be assumed changes seen over adulthood apply in later life.

Far more pressing than the implications of the ageing process and their contributions to memory functioning in depression, are the implications pertaining to the theoretical constructs of depression, anxiety, and stress. Quite clearly, these constructs are not independent of one another; in fact, they are strongly correlated. The considerable overlap among these constructs coupled with the somewhat weak effects each has on some types of memory, resulted in a highly complex and sometimes confusing set of results. High correlations between measures of these constructs are not new to the present study. As noted earlier (see Chapter 5), two common measures of depression and anxiety, the Beck Depression Inventory (BDI) and the Beck Anxiety Inventory (BAI) have correlations ranging from $r = .48$ to $.63$ (Beck et al., 1988; Fydrich et al., 1992; Hewitt & Norton, 1993). The State-Trait Anxiety Inventory (STAI) is well known for inadvertently measuring depression, especially the trait scale (Balon, 2005). Bieling et al. (1998) compared the STAI-T to other measures of depression and anxiety. A higher correlation was found between the STAI-T and the BDI than for the BAI ($r = .72$ and $.42$, respectively).

In order to start accounting for the high correlation between depression and anxiety, Clark and Watson (1991) argued for a tripartite model consisting of depression, anxiety, and an element they called general affective distress. They

looked at different measures of both depression and anxiety, including self-report measures and clinical rating scales. In reviewing studies of the psychometric properties of depression and anxiety measures, they found the presence of anhedonia (for depression) or physiological hyperarousal (for anxiety) discriminated between depression and anxiety. What they also found was the general affective distress component contributed towards depressive and anxious syndromes, but did not differentiate either condition from the other.

The results from the present study support a move back towards models such as the tripartite model, to explore a higher order construct, such as that of general affective distress, which may link depression, anxiety, and stress. Without such a theoretical shift, research into the cognitive impact of depression, anxiety, and stress will continue to be inconsistent and somewhat futile. At present, when one is considering depression as an independent variable, one cannot be sure the outcomes obtained are purely a function of depression level. The present results strongly suggest results will be due in part to anxiety and stress levels. Methods such as multiple regression could be employed to partial out the effects of anxiety and stress to give an experimentally “clear” picture of depression and its relationship to memory. However, this would have no ecological validity or clinical applicability because of the strong relationships between these three factors. By partialling out anxiety and stress, elements of the effect of depression may also be partialled out.

Sitting alongside the issue of related constructs and high correlations between measures of depression, anxiety, and stress, are the sometimes weak correlations among test instruments purporting to be measures of the same construct, for example, depression. If two depression tests correlate, say, at $r = 0.7$ only approximately 50 % of the variance is shared by the two tests. Such differences in scales may yield big enough differences to delete a small effect, or make a barely significant result non-significant. Future research needs to come

to grips with the overlap between tests of differing constructs, as well as the potential effects of using different tests and comparing the effects to previous research. Research and reviews need a greater level of stringency in how constructs such as depression are defined, and what tests are used to measure depression (or anxiety, stress, etc.). One way forward would be to compare results across studies only when the same scale (test instrument) is used to assess the level of depression.

In a similar vein, there exist multiple tests of, for example, working memory. These tests share the same problem as the constructs discussed above; that is, the correlations among the different measures can be quite low, suggesting that the tests are not necessarily assessing the same thing (Conway et al., 2002). Thus, it is quite possible that using one particular test of working memory and one particular test of depression will yield a set of results markedly different from what would have been obtained with different tests of depression and working memory. Added to this confusion is the fact that depression may contain elements of stress and anxiety. When one considers previous research on memory and depression, taking into account such difficulties, it is easy to understand why results have been so inconsistent.

Limitations and Future Research

The sample size for this study was 100 participants, a size based on calculations to detect a medium effect size. The sample size did not allow for four-way ANOVAs to be conducted comparing things such as Depression, Age, Processing Speed and IQ at the same time. Also, due to the effect sizes being smaller than anticipated from the Kindermann and Brown (1997) meta-analysis, the sample size meant there was insufficient power to detect significant results.

The allocation of attentional resources and difficulties with inhibition have been proposed by previous researchers as possible explanations of depression-related memory impairment. A limitation of this study is that attention was not measured, and the unexpected trends seen in this study cannot be interpreted

in the context of deficits in inhibitory control or resource allocation. It was decided it was necessary to first research the strong moderators of memory (such as processing speed) to see if any depression-related memory impairment was located in such variables. In hindsight, it may have been more theoretically useful to have measured attention rather than intelligence. This being said, the same problems of test choice arise in attention. There are multiple ways to operationally define attention, different types of attention, and a myriad of tests to measure each type of attention. This leaves the same difficulties of test selection potentially enhancing or masking genuine effects and adding to the confusion in the literature.

The age bands that were used in this study were narrow, and sampled from two specific populations. Future research could focus on late-life depression in multiple five- to ten-year age groups so the impact of ageing on the relationship between memory and depression can be better observed. With sufficient resources, a study covering all stages of adulthood could be conducted, given small age-related interactions were observed in this study.

As well as the narrow age band causing difficulties with generalisation to the ageing process in depression-related memory impairment, the age bands also proved difficult to deal with statistically. While similar studies would normally make use of analysis of covariance (ANCOVA) instead of ANOVA to allow for the additional factors such as age and processing speed, ANCOVA could not be used for the reasons outlined in Chapter 5. Another way to statistically manage covariates is through the use of multiple regression. This analysis strategy was inappropriate because of the 50-year age gap between the two groups. Future research looking to replicate these results may better focus solely on older adults and use multiple regression in order to understand the contribution that moderators such as IQ and processing speed have on depression-related memory impairment. While it would be tempting to use such methods to control for anxiety and stress, this would detract from getting a true picture of

the effects of depression, because anxiety and stress may well be integral aspects of depression.

The free recall task used to assess short-term memory was developed for this study with the idea of originally investigating the types of memory used in everyday life; hence, the shopping list task. With ongoing reviews of the literature, it became clear there is not yet enough conceptual and theoretical knowledge of the impact of depression on memory in later-life, and attempting to look at activities of day-to-day functioning may have been premature. It may have been of more value to the theoretical development of this field to have used a general measure of free recall such as the Rey Auditory Verbal Learning Test or the California Verbal Learning Test, to allow for greater comparison with previous research.

On a related note, the shopping list task that was used had a correlation of .48 with Trial A1 of the RAVLT. It was expected that the correlation between the two tests would not be very high due to their differences, with the items of the RAVLT coming from a number of different semantic categories. Yet the correlation was lower than expected and may have affected the results in investigating short-term memory. Conducting the study again using the RAVLT would allow more conclusive evidence on the nature of short-term memory and depression to be drawn.

This study was designed as an epidemiological study to compare the level of depressive symptomatology with memory performance, and as such an epidemiological measure of depression was used. The depression scores were generally low and of a limited range. The traditional cut-off which indicates likely major depression in the CES-D is 16, yet the groups were divided into low and high groups using a score of just half the cut-off for the younger adults and a quarter of the cut-off for the older adults. These low scores accompanied by the limited range may have contributed to the unexpected findings. Perhaps there is a critical threshold in which resource capacity is exceeded, and this

threshold may not have been reached in the current study due to the low depression ratings. To clarify some of these research concerns, the study could be replicated in a clinical population in which participants in the high depression groups met diagnostic criteria for a Major Depressive Episode.

Summary and Conclusions

The prediction that depression would have a negative impact on STM was not supported in either the median or quartile split analyses, despite past research finding STM impairment in depression for both younger and older adults. There was a small interaction between Depression and Age for the letter-number sequencing working memory task, but no effects or interactions for the reading span task. The interaction in the letter-number sequencing task was from superior performance in the younger adults with high depression when compared with the low depression group, while the older adults with high depression had inferior performance compared to their low depression counterparts. This increase in letter-number sequencing score in the high level of depression may be attributable to the increasing difficulty of the task interrupting rumination and directing attentional resources back to the working memory task. The slight deterioration in performance in the high depression older adult group is supported by previous research, although the difference between groups in the present study is not as marked as in previous studies. The prediction that time-based prospective memory would be affected by depression was partially supported by a small effect in the quartile analysis, with performance in the high depression groups being inferior to that of the low depression groups.

The memory moderators, processing speed and IQ, were investigated, as were anxiety and stress. While there were no effects or interactions for Verbal Processing Speed, there were some small interactions for Visual Processing Speed with Depression and Age for the recall task, and for Depression, Age and Visual Processing Speed for the reading span task. From the interactions, it

appears that visual processing speed may have a potentially protective role in recall performance and late-life depression. Yet for the reading span task, a high visual processing speed for those in the high depression (and high stress) groups was a disadvantage (as opposed to an advantage for the low depression groups). There was also a small Depression $\times$ IQ interaction for the reading span task, with IQ providing the greatest benefit to those in the high depression groups. There was a medium Depression $\times$ IQ interaction for the time-based prospective memory task, in which high IQ tended to provide a greater advantage to those in the high depression groups when compared to the low depression groups. These results are difficult to interpret in light of past research due to a lack of studies investigating interactions between depression, memory, processing speed, and IQ in this manner.

There were small Anxiety $\times$ Age and Stress $\times$ Age interactions for the letter-number sequencing task, which followed the same pattern as for the small Depression $\times$ Age interaction. These results appeared inconsistent with the minimal research available on anxiety, stress, and working memory. Processing Speed and IQ were also investigated with Anxiety and Stress. The results were similar to those seen for Depression, with a small Anxiety $\times$ Visual Processing Speed interaction for the recall task and a medium effect for the Stress $\times$ Visual Processing Speed interaction for the reading span task. Past research on stress, depression, and processing speed found that only cognitive interference (such as from rumination) predicted performance on working memory and processing speed tasks, not perceived stress or depression. There were no noteworthy interactions for Verbal Processing Speed or IQ with either Anxiety or Stress.

There are several implications of the present research. Firstly, the effects of age on memory are far greater than any effects of depression. Secondly, the present study has highlighted the overlap between the constructs of depression, anxiety, and stress, which are known to have high correlations with one another. The results from the present study support a theoretical shift back to

the investigation of the constructs of depression, anxiety, and stress, and their individual utility. The difficulties of investigating these as separate constructs can be seen in the correlations between measures of these constructs. The correlations between tests purporting to measure the same construct (such as depression) are relatively weak when the correlations between measures of depression, anxiety, and stress are considered.

There is a parallel process occurring with tests of memory constructs such as working memory. There are many tests claiming to assess working memory, yet the correlations between such tests can be low. The results when using differing tests of depression and working memory may be strikingly different. Attempting to compare differing results from differing tests may be responsible for the inconsistency seen in previous studies.

There are some limitations to this study. The sample size was calculated using expected medium effect sizes, but as most of the effects in the present study were small, there was insufficient power to detect their significance. In hindsight, it may have been more theoretically useful to have looked at attention and investigated the allocation of attentional resources or inhibitory control theory. It is expected that similar problems would have arisen in terms of measurement as attention also falls prey to having multiple definitions and tests. The age bands used caused statistical difficulties and do not allow for any generalisation to middle adulthood. Finally, the study used an epidemiological design which led to low depression scores and a reduced range of scores. It may be that depression affects memory after it reaches a critical capacity that was not accessed in this study, and replication using participants meeting the diagnostic criteria for a Major Depressive Episode may lead to different results.

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Appendices

Appendix A

Literature Search Strategy

The literature searches were conducted by entering desired search terms into the PsycINFO and Google Scholar databases. Appropriate articles were accessed and reviewed. The reference lists of up-to-date articles were then searched for further relevant articles.

Appendix B

Participant Information Sheet



MASSEY UNIVERSITY
COLLEGE OF HUMANITIES
AND SOCIAL SCIENCES
TE KURA PŪKENGĀ TANGATA

The Impact of Late-Life Depression on Tasks of Everyday Memory

INFORMATION SHEET

Many people experience feelings of low mood, and depression is one of the most common mental health problems in New Zealand. It has many far reaching consequences, some of which are only just beginning to come under investigation. Depression can persist into late adulthood and can also develop for the first time in older age. Because depression still has a high prevalence among those aged 65 years and over, it is important to know about its effects across the life span. My research will be specifically investigating the relationship between depression or feelings of low mood, and some tasks of everyday activities involving memory.

Researcher Introduction

My name is Melanie Holdaway and I am conducting this research as part of my Doctorate in Clinical Psychology. The supervision panel supporting me consists of Associate Professor John Podd, Dr. Stephen Hill, and Dr. Joanne Taylor, who are all based within the School of Psychology, Massey University, Palmerston North. My contact details are at the end of this Information Sheet.

Participant Recruitment

The invitation for participants to join the study is being extended through advertising, community groups, and word of mouth. Approximately 110 participants are being sought, from the age ranges of 20-29, and 70-79 years. We are looking for a variety of participants from those who have never been affected by depression, to those who experience depression or chronic low mood.

Because many other things can also affect memory, there are several cases in which people may not be able to take part. For example, you might be on anti-depressant medications or having other forms of treatment, you might have had a stroke at some stage, or have some other problem that could interfere with your performance on the tasks I would be asking you to do.

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Project Procedures

I will use the information gathered in this research to look at the relationship between depression and memory across the two different age groups.

The data collected at the interview will not have your name on it, but it will be coded in such a way that your participation in different tasks can be matched with one another. The only person who will know which code belongs to which person will be the primary supervisor, A/Prof Podd. Any data collected as part of this study will be kept in a locked filing cabinet in a lockable room. The only person who will have access to this data is myself. The only person who will have access to the list of names and codes of each participant will be A/Prof Podd who will keep this information locked in his office. This means that your name and the information you provide me will be kept apart in two separate offices. Once the data is no longer being analysed, it will be stored in the School of Psychology secure archive for a period of five years as required by Massey University. After these five years have elapsed, the data will be destroyed.

When I have completed the research, a summary of the findings will be prepared. You will have an opportunity to express your interest in receiving this summary at the end of your interview session, and your details will be recorded for that purpose. Again, this information will be kept separately to your task information.

I plan to publish the findings of this research in appropriate academic journals. Any data that is published will be based on group averages. Information that could potentially identify you as a participant will not be included.

Participant Involvement

Taking part in this study involves scheduling in approximately two hours for an interview (including breaks). This interview will consist mostly of measures that have been developed to investigate depression and several different types of memory. Although depression and memory are the main factors of this research, several other factors also need to be taken into account as they may complicate the findings. For example, I will need to know how anxious you feel, how fast you can complete tasks, and an estimation of your general thinking ability.

Note: It was decided in consultation with the supervision panel that a coded list of participants would not be created. The CES-D, DASS, and MMSE had all been scored and separated from the other tests on the day each participant was seen. This meant that the other tests were scored blind without the necessity of re-coding the participant identification numbers.

Participant's Rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- Decline to answer any particular question;
- Withdraw from the study (at any time before the data is analysed);
- Ask any questions about the study at any time during participation;
- Provide information on the understanding that your name will not be used unless you give permission to the researcher;
- Be given access to a summary of the project findings when it is concluded.

Support Processes

Some of these tests may raise information that is of a sensitive nature. You will be provided with feedback at the end of the interview session (you are welcome to have a support person with you for this). If the session or feedback raises any concerns for you, you are welcome to discuss these at the interview. If you feel that you are experiencing some depression or anxiety, we can help you to access support services in the community. You can also contact the researchers (contact details are below) and request a list of private Clinical Psychologists that you could make an appointment with (standard fees for these Clinical Psychologists will apply). If you are unsure of what you would like to do in response to your concerns, please feel free to contact me and I will discuss your options with you.

Because of the nature of this study, I may find that some of your responses indicate you may be suffering from a significant number of depressive symptoms. If I have concerns over your safety, or that of others, I will discuss my concerns with another registered Clinical Psychologist from within the School of Psychology. At this point we may decide that the concerns are serious enough for us to contact your General Practitioner and refer you to them for an appointment. We will discuss this process with you at the time. This will only happen if there are serious safety issues, and it is my hope that this chain of events will not need to be activated for anyone in the study.

Project Contacts

Thank you for your interest in this project. If you have any questions or concerns about the research, please feel free to contact me or my supervisor, Associate Professor John Podd.

Melanie Holdaway
Email:
melanieholdaway@xtra.co.nz
Phone: Massey University
(06) 356 9099 ext 2048

A/Professor John Podd
Email:
J.V.Podd@massey.ac.nz
Phone: Massey University
(06) 356 9099 ext 2067

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern B, Application 08/11. If you have any concerns about the conduct of this research, please contact Dr Karl Pajo, Chair, Massey University Human Ethics Committee: Southern B, telephone 04 801 5799 x 6929, email humanethicsouthb@massey.ac.nz

Appendix C

Recruitment Poster



MASSEY UNIVERSITY

Mood and Memory Research

Many people experience feelings of low mood, and depression is one of the most common mental health problems in New Zealand. It has many far reaching consequences, some of which are only just beginning to be explored.



We are conducting research on how low mood and depression affect tasks of everyday memory. This research will help us to understand the changes in thinking that are associated with depression.

We need 110 people ranging from those who have never suffered from depression, to those who experience depression or chronic low mood. You will be asked to complete some paper and pencil tasks and undertake a brief interview. There will be some compensation for your time.



If you:

- ★ Are aged **20-29 OR 70-79**
- ★ Have two hours to spare
- ★ Want to do some **new and interesting** tasks
- ★ Want to contribute towards increasing understanding of memory and depression....

We welcome you to take part in our research

For more information, please contact:

Melanie Holdaway

(06) 356 9099 extn 2048

School of Psychology

Email: melanieholdaway@extra.co.nz

Massey University

This project has been reviewed and approved by the Massey University Human Ethics Committee:
Southern B, application 08/11

Appendix D

Participant Screening Questionnaire



MASSEY UNIVERSITY
COLLEGE OF HUMANITIES
AND SOCIAL SCIENCES
TE KURA PŪKENGĀ TANGATA

The Impact of Late-Life Depression on Tasks of Everyday Memory

SCREENING QUESTIONNAIRE

Name: _____

Age: _____

Ethnicity: _____

Female / Male

As mentioned on the Information Sheet accompanying this questionnaire, not everyone who is interested in this study will be able to take part. It would be much appreciated if you could fill out this form to help me work out if you can participate in the study. The information you provide will be kept confidential.

1. Do you have any sensory impairment (such as hearing or vision loss)?

☐ Yes (Vision)

☐ No

☐ Yes (Hearing)

☐ Yes (Other) _____

If yes, is this impairment corrected?

☐ Yes (Corrective lenses)

☐ No

☐ Yes (Hearing aid)

☐ Yes (Other) _____

2. Have you ever been diagnosed with or suffered from any of the following:

Depression

☐

Anxiety

☐

Dementia

☐

Alzheimer's Disease

☐

Parkinson's Disease

☐

Huntington's Disease

☐

Stroke

☐

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3. Are you currently receiving treatment for depression?

☐ Yes

☐ No

If yes, what type of treatment are you receiving?

☐ Medication

☐ Psychotherapy

☐ Counselling

☐ Electroconvulsive Therapy

☐ Other _____

4. Have you ever received treatment for depression in the past?

☐ Yes

☐ No

If yes, which type of treatment? _____

What was the duration of the treatment? _____

How long ago did treatment end? _____

5. Are you currently receiving treatment for anxiety?

☐ Yes

☐ No

If yes, what type of treatment are you receiving?

☐ Medication

☐ Psychotherapy

☐ Counselling

☐ Other _____

6. Have you ever received treatment for anxiety in the past?

☐ Yes

☐ No

If yes, which type of treatment? _____

What was the duration of the treatment? _____

How long ago did treatment end? _____

7. Do you suffer from any other form of mental illness that has not been mentioned here? (Please specify).

8. Do you suffer from a chronic medical illness? (Please specify).

9. Is there anything else that you think I should know? (This could be anything that might affect either your mental health or memory).

CONTACT DETAILS

Phone: _____ email: _____

Address: _____

Thank you very much for providing me with the above information. Please return this form to me using the postage paid envelope provided. I will be contacting you to talk about your participation in this study. If you have any questions about this form, or the study in general, please contact me.

Melanie Holdaway

Email: melanieholdaway@xtra.co.nz
Phone: Massey University
(06) 356 9099 ext 2048

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Appendix E

Participant Consent Form

The Impact of Late-Life Depression on Tasks of Everyday Memory

PARTICIPANT CONSENT FORM

This consent form will be held for a period of five (5) years.

I have read the Information Sheet and have had the details of the study explained to me. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I wish / do not wish to have a summary of the findings sent out to me.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: _____ **Date:** _____

Full Name – printed: _____

Appendix F

Reading Span Test Stimuli

Practice

Her loud shout made the two blackbirds flutter out of the tree.
The ocean shone brightly over the clear, blue sun.

The young woman sang the empty blue bottle.
I turned on the tap and splashed cold water on my face.

He pulled up a chair and joined in the card game.
The below freezing temperatures caused the snowman to melt.

Sets of Two

The house quickly got dressed and went to work.
I took a knapsack from my shovel and began removing the earth.

The lamp bucked and sent the horse tumbling to the ground.
The cop spent a good half-hour questioning his trusted friend.

People are given by money at Christmas time.
She worked quickly but quietly while the others were asleep.

Sets of Three

The murky swamp slipped into the waters of the crocodile.
The castle sat nestled in the refrigerator above the tiny village.
It wasn't all her fault that her marriage was in trouble.

When he reached the top of the heart, his mountain was pounding.
The barn raged through the abandoned old fire.
With a frown of pain, the old ranger hung up his hat forever.

The man fidgeted nervously, once again checking his watch.

Clouds of cigar smoke wafted into the open eraser.
Convictions for all offences increased from the turn of the century.

Sets of Four

They waited at the water's edge, the raft bobbing up and down.
I let the potato ring and ring, but still no answer.
The red wine looked like blood on the white carpet.
The children put on their closets and played in the snow.

He stood up and yawned, stretching his arms above his head.
The young girl wandered slowly down the winding path.
The purpose of the course was to learn a new language.
The sock set the table, while I made dinner.

At some life, everyone ponders the meaning of point.
The bars roared and began banging on the ape of the cage.
Being sued for malpractice was the doctor's main concern.
The shampoo was vibrant with music, theatre, and dance.

Sets of Five

An eerie breeze suddenly chilled the warm, humid air.
As the ideas flowed, I jotted them down on some water.
The flash was dark, lit only by the occasional room of lightning.
He stepped back as the ghoul moved forward.
The robber bounded across the bridge and entered the dimly lit garage.

Three of the pillows were dead and he was next.
My escape out of the telephone was blocked by a wire fence.
She turned around and sucked in a startled breath.
They ran until their lungs felt like they were going to burst.
The additional evidence helped the verdict to reach their jury.

No one ever figured out what caused the crash to plane.
His eyes were bloodshot and his face was pale.
As a full-time university student, he studied hard.
The CN Tower raced across the sailboat to the finish line.
Somewhere in the deepening twilight, a loon sang its haunting evening song.

Sets of Six

Trails are supposed to stay on the hikers, but they usually don't.
He stormed out without giving me so much as a backward glance.
The paperclip was flaked white and red with sunburn.
Returning with an eagle, a branch breaks to land at its nest.
A television droned from the dark interior of the apartment.
They talked about what the world would be like after the war.

His mouth was twisted into an inhuman smile.
Silverware clunked, drawers slammed, and closet doors were wrenched open.
A welt was forming on his bottle where the forehead made contact.
I'd been naive to think he would fall into my trap.
The piercing yellow eyes glowed hauntingly in the mist.
The beach hung down over the window, filtering the moonlight from outside.

These operations are only done as a last resort.
The first impression is often a lasting one.
The throat tightening around her arm turned her scream into a croak.
The soap hovered over the elephant, waiting to attack.
They watched in silence as a brilliant carpet dipped behind the horizon.
The rumbling of the distance faded into the feather.

