The impact of sleep on cognition in older adults: does depression matter?

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Declaration

I declare that this thesis is my own composition; all sources have been acknowledged and my contribution is clearly identified in the thesis. For any work that has been co-published (or prepared for publication), I have the permission of all co-authors to include this work in my thesis. The contribution of each author is noted at the commencement of each paper.

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Abstract

It is now well recognised that poor sleep affects cognition, yet this knowledge has been mostly acquired in younger or middle-aged adults. Studies in older adults are lacking, despite sleep problems and cognitive deficits with ageing being well documented. Similarly, depression has independent effects on both sleep and cognition but its impact on their relationship has been largely unexplored in older adults. The broad objectives of this thesis were to examine how sleep relates to cognition in the context of ageing and how depression impacts on this relationship. Methods of investigation included: self-reports using questionnaires, portable-polysomnography (PSG), wrist-mounted actigraphy monitoring, and neuropsychological tasks.

The first aim of this thesis (Chapter 2) was to investigate how self-reported sleep quality changes across the lifespan, controlling for subclinical symptoms of depression and risk of sleep-disordered breathing (SDB). Participants comprised a large sample of community adults \( (n = 582) \) aged 18–89 years. Results indicate that there were modest, age-related changes in self-reported sleep quality, and that subclinical depression and SDB-risk impacted on subjective sleep quality, but did not have a marked impact on the relationship between age and sleep.

The second aim of this thesis (Chapter 3) was to examine the pattern of association between self-reported sleep quality and cognitive complaints in a community cohort \( (n = 205) \) aged 19–89 years. Standardised questionnaires assessed subjective problems related to memory, attention, and executive functioning. Results indicate that low arousal levels (daytime effects of poor sleep) affected self-reported cognition, largely via effects on attention. In addition, depression was a significant covariate, and explained the relationship between the sleep efficiency factor and executive functioning.

The third and fourth aims of this thesis were to characterise the sleep of older adults with clinical depression, and to investigate the relationship between sleep and cognition in the context of depression in ageing. Chapters 4 and 5 report on studies using objective measures of sleep and cognition in a sample of older adults with and without a diagnosis of Major Depressive Disorder (MDD). In Chapter 4, the sleep of 43 older adults with a current \( (n = 10) \), and a past diagnosis of MDD \( (n = 14) \), and no history of clinical depression \( (n = 19) \), was characterised using PSG, actigraphy, and sleep
questionnaires. Results indicate that sleep problems were a common feature of current depression (‘state-related’), rather than persisting after remission (‘trait-related’).

Chapter 5 investigated the links between sleep-wake patterns (actigraphy) and sleep architecture (PSG) and cognition in older adults with and without current MDD. Results show that sleep-wake patterns, but not sleep architecture, were linked to cognitive performance. Furthermore, there was a distinct pattern of sleep-related cognitive deficits in individuals with current MDD relative to those without current MDD.

Overall, this thesis found that:
1) Ageing had a modest impact on self-reported sleep, complementing our understanding of sleep across the lifespan.
2) The relationship between sleep and cognition varied depending on the aspect of sleep being assessed. Specifically: (i) in adults across the lifespan, daytime effects of poor sleep (indices of arousal) impacted on attention, which in turn affected subjective cognition; (ii) in older adults, disturbed sleep-wake patterns were linked to poorer cognition; however, (iii) sleep architecture was largely unrelated to cognitive performance.
3) Depression (subclinical and clinical levels) had a measurable impact on sleep, and on the relationship between sleep and cognition.

In conclusion, sleep-wake patterns played a critical role in the cognitive profile of older adults. However, sleep stages in this population group did not appear to be significantly associated with cognitive performance. This stands in contrast to neurodevelopmental theories, mostly based on early-life studies, which suggest that sleep architecture is closely linked to cognition. Instead, the current findings suggest that the relationship between sleep stages and cognition may weaken with age. Another key finding in this thesis was that depression, even at subclinical levels, impacted on the relationship between sleep and cognition. Depression is common in older adults and should remain a key clinical target with causal implications for healthy ageing.

Overall, using rigorous methodologies, this thesis reveals new knowledge about the relationship of sleep and cognition in the context of ageing, and the role of depression in this relationship.
Contents

Abstract .................................................................................................................. i
Contents ................................................................................................................ iii
Acknowledgements ............................................................................................... vi
Statement of original contribution ......................................................................... viii
Publications arising from thesis ........................................................................... ix
List of tables and figures ......................................................................................... x
List of abbreviations ............................................................................................... xii

Chapter 1: General introduction ........................................................................... 1
  1.1 Introduction overview ...................................................................................... 1
  1.2 Normal ageing and associated cognitive changes .......................................... 1
      1.2.1 Attention .................................................................................................. 2
      1.2.2 Executive functions ................................................................................ 2
      1.2.3 Memory .................................................................................................. 3
      1.2.4 Other theories of cognitive ageing ......................................................... 4
  1.3 Age-related cognitive decline – a role of sleep? ............................................ 5
      1.3.1 Sleep as a family of interrelated processes .......................................... 6
      1.3.2 Methods of sleep assessment ................................................................. 6
          1.3.2.1 Subjective measures of sleep .......................................................... 6
          1.3.2.2 Objective measures of sleep ............................................................ 7
      1.3.3 Sleep – a developmental perspective .................................................... 9
      1.3.4 Age-related changes in sleep ................................................................. 10
          1.3.4.1 Changes in subjective sleep ............................................................ 10
          1.3.4.2 Changes in objective sleep .............................................................. 10
      1.3.5 Health consequences of poor sleep in older adults ............................... 12
      1.3.6 The association of sleep and cognition ................................................. 13
          1.3.6.1 Sleep and attention ......................................................................... 15
          1.3.6.2 Sleep and executive functioning ...................................................... 17
          1.3.6.3 Sleep and memory ......................................................................... 19
      1.3.7 The role of sleep in age-related changes in cognition ............................ 21
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Statement of Original Contribution

This thesis or any of its components have not previously been submitted for a degree or diploma at any other higher education institution. This thesis is an original contribution and has been accomplished during enrolment in the degree of Doctor of Philosophy (School of Psychiatry and Clinical Neurosciences).

Components of this project, including study design, data collection, data analysis, and the writing of papers and chapters, have been completed by Alix Mellor, with support from her supervisors.

The following tasks were completed over the course of the PhD: research proposal; ethics committee application and amendments; advertising in community centres; attendance at fortnightly meetings and presentations at Selby Lodge and Osborne Park North Metropolitan Area Older Adult Mental Health Services to recruit the depressed sample; liaison with expert staff in the analysis of sleep studies and cortisol analysis; design of the procedure for contacting and testing participants; training for administration of the CogState test battery; writing of a neuropsychological assessment manual and information brochures for participants; creation and administration of an online survey; collection of objective data, including neuropsychological assessments, clinical interviews, and sleep studies for 43 participants over a two-week timeframe per participant (~200 hours of testing); data entry and analysis; and writing of papers.

For any work that has been co-published (or prepared for publication), I have the permission of all co-authors to include this work in my thesis. The contribution of each author is noted at the commencement of each paper.
Publications arising from this thesis

Peer reviewed publications


Components of thesis presented at conferences


Abstracts co-authored by the candidate, describing research beyond this thesis


List of tables and figures

Chapter 1:

Figure 1. Portable-PSG (Somté) ......................................................... 8
Figure 2. Actiwatch .......................................................... 9
Figure 3. The two-process model of sleep ........................................... 10
Figure 4. Example of sleep architecture patterns of a young adult compared to an older adult ................................................................. 12
Figure 5. Direction of predicted relationships in this thesis .................... 34

Chapter 2:

Table 1. Descriptive statistics for the demographics questionnaire, including Berlin Questionnaire and Depression Anxiety and Stress Scale 21 (DASS-21) .......................................................... 49
Table 2. Descriptive statistics for the Pittsburgh Sleep Quality Index (PSQI) ..... 50
Table 3. Correlation matrix (Spearman correlation coefficients) for age, sleep quality (PSQI), and psychological symptoms (DASS-21) ........ 50
Table 4. Regression analyses for all indices of sleep quality (PSQI) ............. 51

Chapter 3:

Table 1. Descriptive statistics for the demographics questionnaire, including DASS-21 .......................................................... 63
Table 2. Descriptive statistics for sleep data ........................................ 66
Table 3. Correlation matrix (Spearman correlation coefficients) for all study variables .......................................................... 67
Table 4. The impact of sleep on attention, memory and executive functioning .......................................................... 69

Chapter 4:

Table 1. Descriptive statistics for all study variables ........................................ 89
Table 2. Correlations for all study variables ........................................ 91
Figure 1. Scatterplot of the relationship between PHQ-9 and PSQI Global score ... 92
Figure 2. Scatterplot of the relationship between PHQ-9 and cortisol (AUC_G) ... 93
Figure 3. Scatterplot of the relationship between cortisol AUC_G and %NREM ... 93

Chapter 5:

Table 1. Cognitive Composites derived from CogState tasks and paper and pencil measures .......................................................... 114
Table 2. Descriptive statistics for demographic and sleep variables for currently depressed versus not currently depressed participants ........ 117
Table 3. Descriptive statistics for cognitive variables (z scores) for currently depressed and not currently depressed participants .............................. 118
Table 4. Correlation matrix (Pearson correlation coefficients) for all study variables .......................................................... 119
<table>
<thead>
<tr>
<th>Figure</th>
<th>Interaction of depression and sleep latency predicting</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Delayed recall accuracy (z scores)</td>
</tr>
<tr>
<td></td>
<td>........................................................................ 122</td>
</tr>
<tr>
<td>Figure</td>
<td>Interaction of depression and total sleep time predicting</td>
</tr>
<tr>
<td></td>
<td>working memory speed (z scores)</td>
</tr>
<tr>
<td></td>
<td>........................................................................ 123</td>
</tr>
<tr>
<td>Figure</td>
<td>Interaction of depression and WASO predicting</td>
</tr>
<tr>
<td></td>
<td>executive functioning speed (z scores)</td>
</tr>
<tr>
<td></td>
<td>........................................................................ 124</td>
</tr>
<tr>
<td>Figure</td>
<td>Interaction of depression and total sleep time predicting</td>
</tr>
<tr>
<td></td>
<td>executive functioning accuracy (z scores)</td>
</tr>
<tr>
<td></td>
<td>........................................................................ 125</td>
</tr>
<tr>
<td>Figure</td>
<td>Interaction of depression and sleep latency predicting</td>
</tr>
<tr>
<td></td>
<td>executive functioning accuracy (z scores)</td>
</tr>
<tr>
<td></td>
<td>........................................................................ 126</td>
</tr>
</tbody>
</table>
List of abbreviations

AUCG – Area Under the Curve with respect to ground
BQ – Berlin Questionnaire
CDS – Cognitive Difficulties Scale
DASS-21 – Depression Anxiety Stress Scale (21 items)
DSM – Diagnostic and Statistical Manual of Mental Disorders
EEG – Electroencephalogram
ESS – Epworth Sleepiness Scale
FrSBe – Frontal Systems Behavior Scale
HPA – Hypothalamic Pituitary Adrenal Axis
HSCT – Hayling Sentence Completion Test
IQ – Intelligence Quotient
LOI – Length of Illness
MDD – Major Depressive Disorder
MEQ – Morningness-Eveningness Questionnaire
MINI – The Mini International Neuropsychiatric Interview
MMQ – Multifactorial Memory Questionnaire
mps – moves per second
ms – milliseconds
NART-R – National Adult Reading Test Revised
NREM – Non-Rapid Eye Movement
ODI-3 – Oxygen Desaturation Index (3%)
OSA – Obstructive Sleep Apnoea
PFC – Prefrontal Cortex
PHQ-9 – Patient Health Questionnaire 9 items
PSG – Polysomnography
PSQI – Pittsburgh Sleep Quality Index
PVT – Psychomotor Vigilance Task
RDI – Respiratory Disturbance Index
REM – Rapid Eye Movement
SDB – Sleep-Disordered Breathing
SDMC – Sleep Dependent Memory Consolidation
SWS – Slow Wave Sleep
TST – Total Sleep Time
WASO – Wake After Sleep Onset (minutes)
Chapter 1
General introduction

1.1 Introduction overview

This thesis starts with a comprehensive literature review. First, the impact of ageing on cognition is discussed, with a summary of the major theories of age-related cognitive decline. Second, changes in sleep with age are reviewed, as well as methods of sleep assessment, and the health and cognitive consequences of poor sleep in the context of ageing. Third, a review of depression in ageing and how it relates to sleep and cognition is presented. Finally, the inter-relationships between sleep, cognition and depression are explored, and proposed mechanisms for these associations are discussed. The chapter ends with a summary of the objectives of this thesis and the research methodologies used.

1.2 Normal ageing and associated cognitive changes

Normal ageing is characterised by a ‘progressive loss of physiological integrity, leading to impaired function, and increased vulnerability to death’ (Lopez-Otin, Blasco, Partridge, Serrano, & Kroemer, 2013). While the definitions of what constitutes ‘ageing’ and ‘older adults’ vary from study to study (e.g., Bastien et al., 2003; 55+ years), (Ohayon & Vecchierini, 2002; 60+ years) (Oosterman, van Someren, Vogels, van Harten, & Scherder, 2009; 50+ years), in this thesis these terms pertain to individuals of 50 years of age and over (the older adult cohort of the current project was 50-79 years).

Decline in cognition is part of normal ageing, however decline is not uniform across all functions. Rather, some cognitive domains and abilities are more affected than others (Bruce & Aloia, 2006). For example, tasks tapping verbal ability (e.g., vocabulary), general knowledge or ‘wisdom’, referred to as ‘crystallised intelligence’\(^1\), remain stable and may even improve with age (Birren & Morrison, 1961; Hedden & Gabrieli, 2004; Horn & Cattell, 1967). On the other hand, ‘fluid intelligence’, a general capacity for problem-solving and abstract intelligence, generally declines with age (Horn & Cattell,

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\(^1\) The concepts of crystallised and fluid intelligence form part of the Cattell-Horn-Carroll theory of intelligence, which suggests that intelligence is composed of cognitive abilities that can be grouped into various domains (McGrew, 2005).
Major domains such as attention, executive functioning, and memory are particularly vulnerable to age-related decline. Moreover, within these domains there is variability in the extent of change in normal ageing.

### 1.2.1 Attention

There are various types of attention, which are differentially affected by ageing. Divided attention (e.g., Zanto & Gazzaley, 2014) and selective attention are particularly susceptible to age-related decline (McDowd & Shaw, 2000; Zanto & Gazzaley, 2014), especially on complex tasks (Gilsky, 2007; Wright, 1981). It is important to note that several neuropsychological tests used to assess attention also assess components of executive functioning (Perry & Hodges, 1999). For example, the Stroop task measures selective attention, but is also considered a measure of inhibition and working memory, which are components of executive functioning (Strauss, Sherman, & Spreen, 2006). On the other hand, other types of attention, such as sustained attention, appear relatively resilient to age effects (e.g., Berardi, 2001; Carriere, Cheyne, Solman, & Smilek, 2010), at least until the age of 70 when deficits begin to emerge (Filley & Cullum, 1994).

### 1.2.2 Executive functions

Executive functions encompass a variety of higher order cognitive processes involved in self-regulated goal-directed behaviour (Drag & Bieliauskas, 2010). There are multiple components of executive functioning, including working memory, task-switching, planning, inhibition, verbal fluency and problem-solving, all of which are susceptible to age-related cognitive decline (Brink & McDowd, 1999; Craik & Grady, 2002; Drag & Bieliauskas, 2010; Phillips & Henry, 2008; Rodríguez-Aranda & Sundet, 2006; Spieler, Balota, & Faust, 1996; Van Hooren et al., 2007; Verhaeghen & Cerella, 2002). Although diverse, these processes are united in their reliance on the prefrontal cortex (PFC), which is particularly vulnerable to neurological change and accordingly, to age-related decline.

One explanation for the deficits in executive functioning in normal ageing suggests that they result from age-related changes in frontal areas, namely the PFC (Braver & Barch, 2002; Cabeza & Dennis, 2012). This theory, referred to as the ‘frontal lobe hypothesis’, suggests that the wide range of cognitive deficits observed in older adults results from
altered prefrontal lobe activity. Support for this theory derives from neuro-imaging studies in older adults, which indicate disproportionate changes in the structure and functioning of the frontal lobes, including neurochemical changes; decrease in brain volume; and white and grey matter changes (e.g., Head et al., 2004; Li, Lindenberger, & Sikström, 2001; Raz, Briggs, Marks, & Acker, 1999; Raz et al., 2005).

Furthermore, functional imaging studies point to changes in PFC activation observed during neuropsychological tasks in older adults (e.g., Rypma, Prabhakaran, Desmond, & Gabrieli, 2001). For example, some studies have reported decreased activation of frontal areas, which corresponds to age-related deficits in cognitive tasks assessing divided and selective attention (e.g., West, Murphy, Armilio, Craik, & Stuss, 2002), working memory (e.g., Persson et al., 2006; Rypma & D'Esposito, 2000; Rypma et al., 2001) and episodic memory (e.g., Grady et al., 1995; Stebbins et al., 2002). In fact, biological explanations for age-related memory impairment propose that deficits in frontal-lobe processes such as working memory may mediate age-related decline in episodic memory (e.g., Brickman & Stern, 2009; Salthouse, Atkinson, & Berish, 2003; Salthouse & Babcock, 1991).

In contrast, other studies report over-activation in specific frontal areas, which correlate with some age-related cognitive deficits (e.g., Greenwood, 2007; Park & Reuter-Lorenz, 2009). These findings may suggest dysfunction of the frontal lobes or, alternatively, underlying compensatory processes.

### 1.2.3 Memory

Memory is one of the most commonly reported cognitive domains that changes with age. Subjective memory complaints, such as forgetfulness, are widely reported by older adults (Jonker, Geerlings, & Schmand, 2000; Ponds, Commissaris, & Jolles, 1997) and have been linked to future cognitive decline (Jessen et al., 2010; Jorm, Christensen, Korten, Jacomb, & Henderson, 2001; Reid & MacLullich, 2006).

Studies of objective memory functioning often validate these subjective complaints, and show that older adults consistently under-perform relative to younger adults on memory tasks, which are hippocampal-dependent (Troyer, D'Souza, Vandermorris, & Murphy, 2011).
Specific types of memory are differently affected by ageing. For example, there are minimal changes in procedural memory, whereas on average, working memory, prospective memory, context memory, and long-term declarative memory, including episodic memory, decline significantly with age (e.g., Henry, MacLeod, Phillips, & Crawford, 2004; Hornung, Danker-Hopfe, & Heuser, 2005; Nilsson, 2003). Of note, declines in episodic memory occur across various tasks, including verbal and non-verbal learning (Brickman & Stern, 2009).

Age-related deficits in memory are often explained by changes in the frontal lobes and medial temporal cortices, especially the hippocampus (e.g., Raz & Rodrigue, 2006; Raz, Rodrigue, Head, Kennedy, & Acker, 2004). Accordingly, the ‘temporal lobe hypothesis’ suggests that age-related memory deficits are related to structural and functional changes in the medial temporal lobes as people age (e.g., Raz et al., 2005). In support, neuronal loss and reduced hippocampal volume have been associated with memory loss in healthy ageing (Head, Rodrigue, Kennedy, & Raz, 2008; Morrison & Hof, 1997; Persson et al., 2006; Raz, Gunning-Dixon, Head, Dupuis, & Acker, 1998). In fact, a recent structural MRI study found that medial temporal lobe volume, but not prefrontal lobe volume, predicted episodic, semantic, and working memory (Bailey et al., 2013).

1.2.4 Other theories of cognitive ageing

Several theories of cognitive ageing have been developed, each of which proposes that one or more underlying mechanisms is primarily responsible for age-related deficits in specific domains of cognition. Some explanations focus on neurochemical changes (e.g., the dopamine theory of cognitive ageing; Bäckman & Farde, 2004), and are beyond the scope of this thesis; others propose more localised changes in brain structure and function (e.g., changes in the frontal lobes and medial temporal lobes) and have been reviewed above; others, still, focus on more diffuse changes in cognitive functions (e.g., deficits in processing speed; inhibition filters), and are reviewed briefly here for the sake of completeness, but are not specifically addressed in this thesis. These theories are not necessarily mutually exclusive (Drag & Bieliauskas, 2010).

An earlier theory that relates to age-related deficits in executive functioning is the ‘theory of inhibition’. Hasher and Zacks (1988) proposed that brain changes associated
with ageing led to fundamental failures in inhibiting irrelevant information, leading to broad deficits in all cognitive domains. For example, deficits in memory processing were suggested to result from the failure to filter out irrelevant information during memory retrieval. Evidence presented in support includes the finding that free recall is affected by age more than recognition memory, where all of the relevant information is provided (e.g., Craik & McDowd, 1987; Drag & Bieliauskas, 2010). However, while there is evidence that older adults are more distracted by irrelevant information (Hedden & Park, 2001), not all findings support this theory (e.g., McDowd & Shaw, 2000).

The ‘theory of limited processing capacity’ suggests that cognitive deficits in older adults are due to a decrease in attention-processing resources available with ageing (Verhaeghen & Cerella, 2002). Craik (1986) describes a reduction in mental energy with age, suggesting that effortful tasks requiring high attentional demands cause the greatest deficits in older adults, whereas automatic tasks are more robust. The ‘speed of processing hypothesis’ proposes that a general slowing of all mental processes in older adults can account for age-related deficits in various cognitive tasks, including working memory and long-term memory (Salthouse, 1994, 1996; Salthouse & Miles, 2002). These abilities appear to be differentially affected depending on task complexity. For example, Verhaeghen, Cerella, and Basak (2006) showed that speed of processing on simple verbal tasks was relatively robust to age-related decline, but that slowed information-processing led to decreases in performance on more complicated verbal and visuospatial tasks. Although there is widespread agreement that processing speed declines with age, some research suggests that this theory cannot explain the significant variance across different cognitive domains (e.g., Bieliauskas, 2001). In addition, this account cannot explain the age-related changes in neural activation that have been observed in neuroimaging studies during higher-order cognitive processes in older adults, hence the development of neurobiological theories.

1.3 Age-related cognitive decline – a role of sleep?

While there is much evidence that ageing is linked to changes in cognition, substantial inter-individual variability has been noted (e.g., Ardila, 2007). That is, while some individuals experience substantial age-related decline in functioning, other individuals experience relatively subtle declines (e.g., Fillit et al., 2002). Some accounts propose that this variability could be due to differences in brain structure and function, such as
increasing atrophy (Raz et al., 1998) and white matter pathology (Gunning-Dixon & Raz, 2000). Other accounts suggest that differences in compensatory mechanisms, cognitive reserve, psychosocial variables (e.g., family support), physical health, and psychological health, are responsible (Shaie, 1990; Vandenberghhe & Tournoy, 2005).

More recent developments suggest that sleep is a potentially important variable that may account for individual variability in age-related cognitive change (e.g., Nebes, Buysse, Halligan, Houck, & Monk, 2009).

In the following sections, a comprehensive review of sleep in ageing is presented; then the links between sleep and cognition are reviewed; and a summary of the evidence of the role of sleep in age-related cognitive decline is given.

1.3.1 Sleep as a family of interrelated processes

Sleep is best understood as a family of processes, each with separate but interrelated components. Sleep physiology, or sleep architecture, refers to the organisation of sleep stages, whereas sleep cycles refer to circadian rhythms and the pattern of sleep-wake activity. Subjective measures of sleep include self-reports about sleep quality, but also the consequences of sleep on daytime functions. This configuration is helpful when explaining sleep changes in older adults.

1.3.2 Methods of sleep assessment

1.3.2.1 Subjective measures of sleep

Subjective sleep quality is an important component of sleep assessment, and is based on self-reports. It is often undervalued, as it is reliant on individual perceptions, which can be biased, however self-reports are typically used in clinical settings to determine the need and type of treatment required, and thus are of significant clinical importance. Methods of subjective measures of sleep include sleep logs or diaries, which can accompany actigraphy measures. Sleep diaries are usually completed over one to two weeks, and provide information about sleep time, sleep latency, total sleep duration and sleep efficiency. Subjective sleep measures also include standardised questionnaires designed to assess sleep quality, such as the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI assesses sleep quality in
the previous month and provides information about seven different aspects of sleep quality such as night-time sleep efficiency, sleep latency, and daytime effects of poor sleep. The PSQI also yields a measure of overall sleep quality based on a total score, which is often used to discern between ‘good’ versus ‘poor sleepers’ (using a cut-off score of 5 on the Global PSQI; Buysse et al., 1989). Another example of a subjective measure of sleep is the Epworth Sleepiness Questionnaire, which assesses self-reported daytime sleepiness (ESS; Johns, 1991).

1.3.2.2 Objective measures of sleep

Objective measures of sleep include polysomnography (PSG) and actigraphy.

(a) Polysomnography

PSG (see Figure 1) is commonly referred to as an ‘overnight sleep study’, which involves attaching electrodes to the face and body to permit the recording of overnight brain and respiratory functions and blood oxygen levels (Butkov, 2007). This test is used to investigate sleep stages and is also used in the diagnosis of sleep disorders such as Obstructive Sleep Apnoea (OSA).

Sleep stages: Briefly, sleep can be divided into two types – Rapid Eye Movement (REM) and Non-REM sleep (NREM; e.g., Carskadon & Dement, 2005) – that alternate in four or five cycles each night (Pace-Schott & Hobson, 2002). REM sleep has been referred to as active sleep and is associated with dreaming and mixed brain activity and eye movements (Carskadon & Dement, 2005). NREM sleep is broken down into stages 1–3, which represent a continuum of depth (Stanley, 2005): Stage N1 is the lightest stage of sleep, characterised by high amplitude brain waves; Stage N2 is also a shallow sleep stage, but includes rapid bursts of activity called sleep spindles; and Stage N3 is referred to as Slow Wave Sleep (SWS), which is characterised by high altitude delta waves (Edwards et al., 2010). Previously, a fourth stage, N4, was described, however this stage of sleep has now been combined with Stage N3, due to revised scoring by the American Academy of Sleep Medicine (AASM; Iber, Ancoli-Israel, Chesson, & Quan, 2007b). There is a greater amount of SWS in the first half of the night, whereas the proportion of REM sleep increases in the later part of the sleep cycle (Pace-Schott & Hobson, 2002).

Overnight PSG conducted in a sleep laboratory is considered the gold-standard method of assessment for sleep disorders (Kryger, 2010). However, portable-PSG enables
investigation of sleep stages in contexts more familiar to the participant, and is increasingly accepted as a valid alternative when it is difficult to conduct laboratory-based studies because of patient and practical requirements. Portable-PSG also offers benefits over laboratory-based PSG in that it allows a more natural testing environment in which participants may sleep in their usual home environment. A portable sleep device, called the Somté, has been found to be effective in identifying sleep and respiratory variables, and has good agreement with full PSG (Ferré et al., 2012), and is used in the current study. It includes electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), airflow, body position, thoracic and respiratory belts, and blood oxygen saturation. The data from the sleep study is typically analysed by a trained sleep technician (Jennifer Maul in this research project).

Figure 1. Portable Somté device

(b) Actigraphy

Whereas PSG assesses sleep stages (or sleep architecture), actigraphy (Figure 2) is used when examination of sleep-wake patterns is required. An ‘actiwatch’ is a portable device, worn on the wrist, which is used to record movement, from which sleep-wake patterns are inferred. It is a popular device, which has been found to be a reliable and valid instrument to assess sleep-wake patterns (Berger et al., 2008; Morgenthaler et al., 2007). While actigraphy monitoring alone is not valid for the diagnosis of insomnia (Neubauer, 1999), some of the advantages include the fact that it permits 24-hour online recording and may be worn for an extended period of time (up to 30 days), and that it allows assessment of sleep-wake patterns while the individual goes about their normal routine (Sadeh & Acebo, 2002). Seven days of actigraphy recording is sufficient to obtain a valuable ‘snapshot’ of an individual’s sleep quality (Knutson, Rathouz, Yan, Liu, & Lauderdale, 2007; Tworoger, Davis, Vitiello, Lentz, & McTiernan, 2005).
Actigraphy output measures include total sleep time (TST; mins), sleep efficiency (%), sleep latency (mins), and minutes spent awake after sleep onset (WASO; mins). Actigraphy has been used in healthy (e.g., Blackwell et al., 2011) and clinical populations (e.g., Lichstein et al., 2006). An understanding of the limitations of actigraphy monitoring (it records movement rather than sleep per se) helps in the interpretation of the results. Typically, interpretation is aided by a sleep log, which is completed by the participant over seven days, and measures sleep onset and offset, interruptions to sleep, and the times when the actiwatch was taken off. The combination of actigraphy and sleep logs, as well as cross-rater validity checks by experienced raters (myself and A/Prof Waters in this research) helps to maximise the quality of the data generated.

Figure 2. Actiwatch

1.3.3 Sleep – a developmental perspective

Sleep changes across the lifespan. For example, infants spend a significantly greater proportion of the night in Rapid Eye Movement (REM) sleep compared to adults (D’Ambrosio & Redline, 2014). This stage of sleep is thought to assist with brain development and learning in early life (Mirmiran & Van Someren, 1993). In older age, sleep becomes less consolidated and REM sleep gradually declines (D’Ambrosio & Redline, 2014). Similarly, the proportion of Slow Wave Sleep (SWS) undergoes dramatic reductions with ageing (e.g., Stanley, 2005). Hence, it has been speculated that sleep physiology might adapt to human requirements for learning and cognition (Geiger, Achermann, & Jenni, 2010).
1.3.4 Age-related changes in sleep

1.3.4.1 Changes in subjective sleep

Studies indicate that sleep complaints are common in older adults. Estimates range from 50–60% in adults over the age of 65 (Ancoli-Israel, 2005; Foley et al., 1995; Staner, 2010; Vitiello, Foley, Stratton, & White, 2004). Subjective reports frequently include problems with sleep onset, duration and maintenance, as well as associated daytime consequences such as sleepiness, waking feeling un-refreshed, functional disability and fatigue (Nebes et al., 2009; Ohayon & Vecchierini, 2005; Ohayon, Zulley, Guilleminault, Smirne, & Priest, 2001; Vaz Fragoso & Gill, 2007). However, not all studies of older adults have found that sleep complaints increase with age (Middelkoop, Smilde-van den Doel, Neven, Kamphuisen, & Springer, 1996). In fact, some studies suggest that older adults do not necessarily rate their sleep quality as poor and are largely satisfied with their sleep once health conditions linked to ageing have been controlled for (Foley et al., 1995; Vitiello, Moe, & Prinz, 2002).

1.3.4.2 Changes in objective sleep

In ageing, sleep changes include alterations to sleep-wake patterns and the organisation of sleep stages (Bliwise, 1993; D’Ambrosio & Redline, 2014; Dijk, Duffy, & Czeisler, 2000; Espiritu, 2008; Vitiello, 2006).

(a) Sleep-wake cycle changes with age

The sleep-wake cycle is regulated by the circadian rhythm system (the ‘wake promoting system’, referred to as ‘Process C’), which interacts with homeostatic processes (the ‘sleep promoting system’, referred to as ‘Process S’; Ancoli-Israel, Ayalon, & Salzman, 2008; Gillette & Abott, 2006). This has been referred to as the two-process model of sleep-wake regulation (Borbely, 1982; see Figure 3). Both components of this model have been implicated in age-related changes in sleep.

![Figure 3. The two-process model of sleep](image-url)
Circadian rhythms are endogenous biological rhythms that control physiological and behavioural processes such as the sleep-wake cycle. The superchiasmatic nucleus (SCN) in the hypothalamus is the centre for the circadian system and the cells within it contain ‘molecular clocks’ that are synchronised to 24-hour days (Pace-Schott & Hobson, 2002). The SCN also modulates secretion of melatonin, a hormone secreted from the pineal gland, which is involved in regulation of sleep (Kunz & Hermann, 2000). There is evidence of biological changes in the SCN with ageing, such as decreases in the number of cells (e.g., Naylor & Zee, 2006) and decreased melatonin secretion (e.g., Touitou, 2001). As a result, there are significant changes in circadian rhythms, which may be partly responsible for age-related changes in sleep (e.g., Espiritu, 2008). On average, ageing is associated with less robust and synchronised circadian rhythms resulting in less consistent periods of sleep/wake across the day/night (Ancoli-Israel et al., 2008). Phase advancement of circadian rhythm in older adults is also common, which results in earlier sleep onset and earlier waking (Crowley, 2011; Harrington & Lee-Chiong, 2007; Naylor & Zee, 2006). Problems of sleep fragmentation seen in older adults might also be due to continuing decreases in sleep homeostasis or the drive to sleep (Bliwise, 2005; Dijk et al., 2000).

Age-related disturbances to sleep-wake patterns include delayed sleep onset, increased napping, increased night-time awakenings, decreased sleep efficiency (the time spent asleep as a proportion of the time in bed), and decreases in total sleep time, although this last point has been disputed in some studies (e.g., Bliwise, 1993; Huang et al., 2002; McCrae, 2009).

Studies using actigraphy provide evidence of disrupted sleep-wake patterns in older adults, indicating increased time spent awake after sleep onset (WASO), longer sleep onset latency, decreased sleep efficiency, and reduced total sleep time, with increasing age (e.g., Hoch et al., 1997; Hoch et al., 1994; Huang et al., 2002).

(b) Sleep architecture changes with age

Sleep architecture changes with age. As can be seen from Figure 4, a reduction in the percentage of SWS (Stage N3) is perhaps the most pronounced change² (Knowles & MacLean, 1990; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004; Stanley, 2005).
Figure 4. Example of sleep architecture patterns of a young adult compared to an older adult. Older adults typically have more fragmented sleep and decreased SWS compared to younger adults (Stage 3 and 4 represent SWS in this diagram, however Stage 4 no longer exists according to modern scoring criteria). This image was reproduced with permission from American Family Physician (1999).

Some studies suggest that there is relative preservation of REM sleep in ageing (e.g., Raju & Radtke, 2012), however others suggest that there is still some reduction (D’Ambrosio & Redline, 2014; Floyd, Janisse, Jenuwine, & Ager, 2007; Ohayon et al., 2004; Stanley, 2005). Increases in Stages N1 and N2 have also been reported, which might represent compensation for the reduction in other sleep stages (Bliwise, 1993; Feinsilver & Hertz, 1993; Ohayon et al., 2004). Finally, there is also a decrease in the number of sleep cycles in older adults (Feinsilver & Hertz, 1993).

1.3.5 Health consequences of poor sleep in older adults

There are important life-sustaining and restorative functions of sleep, including, but not limited to, restoration of brain energy (Kunz & Hermann, 2000), cleansing of

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2 Some studies have suggested that age-related reductions in SWS are due to the fact that while the EEG frequency of SWS is maintained, the amplitude of the wave decreases and no longer meets the scoring criteria for SWS (e.g., Bliwise, 1993; Feinberg, 1976). It has been suggested that if scoring criteria are modified, older adults have an equivalent amount of SWS to younger adults (Feinberg, Fein, Floyd, & Aminoff, 1983; Smith, Karacan, & Yang, 1977). However, the majority of published studies investigate age-related changes in sleep architecture follow standard practice and adhere to the criteria recommended by the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events (Iber et al., 2007b).
cerebrospinal fluid and maintenance of metabolic homeostasis (Xie et al., 2013), and cell synthesis and repair (Bellesi et al., 2013).

Evidence regarding the pivotal role of sleep derives from clinical (sleep-disordered) populations and community older adults, indicating that poor sleep is frequently associated with problems with daytime functioning, reduced health-related quality of life, alongside reduced energy, mental, social and physical functioning (Alapin et al., 2000; Ancoli-Israel, 2009). There is a strong association of sleep disorders in the older population and decreased ability to carry out activities of daily living, and increased morbidity and mortality (McCrae, 2009).

Sleep problems can also affect mental and physical health in older adults (Avidan et al., 2005; Schubert et al., 2002), and can increase the severity of existing conditions such as diabetes (Reid et al., 2006). They also have been linked to an increased rate in falls and a reduction in quality of life (Ancoli-Israel & Cooke, 2005). Reid et al. (2006) found that physical and mental health problems were directly correlated with the number and type of self-reported sleep disturbances in a dose-dependent way, especially on domains such as daytime somnolence, difficulty sleeping and feeling un-refreshed on awakening.

### 1.3.6 The association of sleep and cognition

Accumulating evidence suggests that sleep plays an important role in cognition. Evidence derives from several different methodological approaches.

Experimental sleep deprivation paradigms are commonly used to investigate the effect of sleep loss on cognition (e.g., Drummond et al., 2000), although sample sizes are typically small and there are limited studies in older adults. The second approach refers to cross-sectional experimental studies, contrasting the cognitive profiles of ‘good sleepers’ to ‘poor sleepers’ (e.g., Nebes et al., 2009). In these studies, sleep may be assessed using objective measurements (e.g., PSG, actigraphy) or subjective reports (questionnaires), and cognition is assessed using neuropsychological tests. Another related approach comprises epidemiological methods which rely on questionnaires.

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3 The link between sleep and mental health, specifically depression, will be discussed in detail in subsequent sections.
regarding perceived sleep quality and cognition complaints, and which have benefits of large sample sizes and greater statistical power (e.g., Kronholm et al., 2009; \( N = 5177 \)).

A different style of experimental paradigm examines how sleep exerts effects on learning, and the longer-term benefits of sleep on cognition. The role of sleep on cognition is assessed either by correlating specific features of sleep (e.g., sleep duration or sleep stages) with cognitive performance (‘waking cognitive performance’), or by directly comparing the effects of a period of time asleep versus a period of time awake on the consolidation of information (‘consolidation paradigms’). The latter approach refers to studies demonstrating that sleep plays an active role of system consolidation (e.g., Backhaus et al., 2007).

The final approach involves clinical populations with a primary diagnosis of sleep disorders, and provides important information regarding effects of poor sleep. For example, research in individuals with sleep-disordered breathing disorders (SDB) such as obstructive sleep apnoea (OSA), and circadian rhythm disorders, provides rich information regarding mechanistic processes of sleep disorders and specific cognitive deficits in comparison to healthy controls (e.g., Bucks, Olaithé, & Eastwood, 2013; Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012; Salorio, White, Piccirillo, Dunley, & Uhles, 2002). Throughout this thesis, studies using individuals with SDB are cited in support of a close link between sleep disturbance and cognitive deficits. However, it is important to acknowledge that the mechanisms by which SDB impacts on cognition may be due to sleep disturbance as a result of repeated arousals from sleep (Verstraeten, 2007), and/or to hypoxic effects on the brain (Beebe & Gozal, 2002). Therefore, studies investigating the impact of sleep disturbance on cognitive functioning must control for potential effects of SDB, and the associated hypoxia, which is often underdiagnosed (Peppard, Szklo-Coxe, Hla, & Young, 2006).

Three domains of cognition are typically affected by sleep in adults and older adults: attention, memory and executive functioning. Because the literature in adults is extensive, but less so in older adults, available evidence pertaining to both population groups is reviewed here.
1.3.6.1 Sleep and attention

(a) Attention deficits in adults

Vigilance, or sustained attention, has been described as “the fundamental process affected by sleep loss” (Lim & Dinges, 2008). In support, sleep deprivation studies in adults show that restricted sleep duration (< 6 hours a night) causes lapses in attention on attentional tasks such as the Psychomotor Vigilance Task (PVT; e.g., Drake et al., 2001; Van Dongen, Maislin, Mullington, & Dinges, 2003). Similarly, epidemiological studies in adults have linked self-reported sleep problems to attention deficits (e.g., Kronholm et al., 2009), and deficits in attention and vigilance are widely reported in adults with sleep disorders such as insomnia (e.g., Hauri, 1997), and sleep-disordered breathing (SDB; Aloia, Arnedt, Davis, Riggs, & Byrd, 2004; Beebe, Groesz, Wells, Nichols, & McGee, 2003; Bucks et al., 2013; Verstraeten, 2007).

Little is known about the mechanisms by which sleep impacts on attention. One explanation for decreased performance on attention tasks following sleep deprivation has been referred to as the ‘vigilance hypothesis’ (Doran, Van Dongen, & Dinges, 2001; Williams, Lubin, & Goodnow, 1959). This model suggests that circadian rhythms and the homeostatic drive for sleep impact on arousal levels and lead to ‘lapses’ in attention.

Regarding the effect of sleep architecture on attention, one study indicated a strong relationship between more SWS and faster reaction times on vigilance tasks in young adults (Jurado, Lunavillegas, & Buelacasal, 1989). This suggests that SWS is important for vigilance performance, at least in younger adults.

(b) Attention deficits in older adults

Studies in older adults are less common and the majority of evidence for the association of sleep and attention in this age group derives from epidemiological studies. Studies are supportive of the suggestion that sleep plays a key role in attention, although evidence largely derives from self-reports and assessment of sleep-wake patterns.

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4 The ‘vigilance hypothesis’ is also referred to as the ‘lapse hypothesis’ (Williams et al., 1959), and the ‘wake-state instability hypothesis’ (Doran et al., 2001). While the ‘lapse hypothesis’ suggests that brief periods of low arousal or ‘lapses’ disrupt cognitive performance, an expansion of this hypothesis (the ‘wake-state instability hypothesis’) suggests that increased variability in responses after sleep deprivation is due to competing influences of increasing homeostatic drive for sleep and the need to stay alert. These conceptually similar hypotheses can be collectively referred to as the ‘vigilance hypothesis’ given their mutual focus on decreased arousal and vigilance as predictors of cognitive deficits following sleep deprivation.
while there is inconsistent evidence regarding PSG.

For example, Amer, Hamza, El Akkad, and Galeel (2013) investigated 100 adults over 60 years of age from elderly care homes and found that ‘poor sleepers’ (as defined by the PSQI) had more attention deficits on the Mini Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975), compared to ‘good sleepers’. Similarly, self-reported insomnia symptoms have been linked to problems with attention on digit span tests (Bastien et al., 2003, N = 60, 55+ years); (Vignola, Lamoureux, Bastien, & Morin, 2000, N = 60, 55+ years). Furthermore, sleepiness on the Epworth Sleepiness Scale (ESS; Johns, 1991) has been associated with self-reported attention problems in 1026 community older adults (Ohayon & Vecchierini, 2002, 60+ years). Finally, Haimov, Hanuka, and Horowitz (2008) found older adults with self-reported insomnia (also supported by actigraphy measures indicating poor sleep quality) had significant deficits in sustained attention compared to older adults without insomnia.

Studies in older adults with SDB also indicate pronounced deficits in attention and vigilance (see Zimmerman & Aloia, 2012 for review). For example, a study of 611 adults (35–74 years) reported an association of severity of SDB, as assessed with the Apnoea Hypopnia Index (AHI), and deficits on the PVT in the 65–74 year old age-group (Kim, Dinges, & Young, 2007).

Sleep deprivation studies in older adults have produced interesting results and suggest that the impact of restricted sleep on attention might be moderated by age (e.g., Duffy, Willson, Wang, & Czeisler, 2009). Several studies have shown that vigilance in older adults is more resistant to sleep deprivation than in younger adults (e.g., Adam, Retey, Khatami, & Landolt, 2006; Philip et al., 2004; Stenuit & Kerkhofs, 2005). This suggests that the relationship between sleep and attention might change, and even weaken, with age.

There are very few studies investigating the relationship between sleep architecture and sustained attention in adults and the existing studies are somewhat inconsistent. For example, Crenshaw and Edinger (1999) found an association of greater SWS and faster reaction time on reaction time tasks, including a vigilance task, in 64 older adults (60+ years) with insomnia, but not in those without. In contrast, Van der Werf, Altena, Vis, Koene, and Van Someren (2011) found that selective SWS reduction using acoustic
stimuli was linked to poorer vigilance performance the next day in 13 older adults (mean age = 60.1 years) without sleep complaints, but not to response time. Taken together, these studies provide mixed support of a link between SWS and vigilance in older adults.

1.3.6.2 Sleep and executive functioning

(a) Executive functioning deficits in adults

According to the ‘frontal lobe hypothesis’, sleep loss produces deficits in executive functioning as a result of changes in the Prefrontal Cortex (PFC), which is particularly vulnerable to the effects of sleep loss (e.g., Beebe & Gozal, 2002; Horne, 1993). In support, sleep deprivation studies in adults show that sleep loss leads to decreased performance on PFC-dependent neuropsychological tasks assessing planning (e.g., Killgore et al., 2008), decision making (e.g., Harrison & Horne, 2000), problem-solving (e.g., Killgore et al., 2008), inhibition (e.g., Drummond, Paulus, & Tapert, 2006; Harrison, Jones, & Waterhouse, 2007), divided attention (e.g., Jackson et al., 2011), mental flexibility (e.g., Heuer, Kleinsorge, Klein, & Kohlisch, 2004; Van Dongen et al., 2003), verbal fluency (e.g., Harrison & Horne, 1997), and working memory (e.g., Chee & Choo, 2004; Jones & Harrison, 2001; Pilcher, Band, Odle-Dusseau, & Muth, 2007).

Clinical studies also support a crucial role of sleep in executive functioning, showing that adults with OSA exhibit greater problems with planning, inhibition, shifting, generativity, and working memory compared to healthy controls (e.g., Olaithe & Bucks, 2013).

Finally, research in community samples demonstrates effects of poor self-reported sleep quality on components of executive functioning. For example, perceived insufficient sleep was linked to poorer performance on working memory tasks in 143 adolescents (Gradisar, Terrill, Johnston, & Douglas, 2008), and greater insomnia severity was linked to poorer cognitive control on a thought control questionnaire in 310 individuals aged 17–65 years (Schmidt, Gay, Ghiisletta, & Van der Linden, 2010).

Overall, there is evidence that sleep affects executive functioning. Furthermore, it has been suggested that the impact of sleep on executive functioning in adults is driven by specific sleep stages. For example, REM sleep (Cai, Mednick, Harrison, Kanady, &
Mednick, 2009; Walker, Liston, Hobson, & Stickgold, 2002) and SWS (Beijamini, Ribeiro Pereira, Cini, & Louzada, 2014) have both been positively associated with performance on problem-solving tasks in younger adults.

(b) Executive functioning deficits in older adults

Although studies regarding sleep and executive functioning are less common in older adults, there are similarities to the findings in adults, using both self-reports and actigraphy monitoring, although little evidence exists from PSG.

With regard to questionnaire studies, Nebes et al. (2009) compared ‘good’ versus ‘poor sleepers’ (using a cut-off score of 5 on the Global PSQI) in 157 community older adults (65–80 years). They found that ‘poor sleepers’ had poorer executive functioning performance, including in working memory, attentional set shifting and problem-solving, compared to ‘good sleepers’.

Actigraphy studies support the link between sleep and executive functioning in older adults. For example, Blackwell, Yaffe, Ancoli-Israel, Schneider, and et al. (2006) found an association between sleep-wake pattern disturbance and performance on the Trails Making Test – Part B, which primarily assesses shifting (Trails B; Reitan, 1958) in 2932 older women (65+ years). Specifically, greater time spent awake after sleep onset (WASO), longer sleep latency, and less sleep efficiency, were associated with poorer performance. A later study in 3132 older men (67+ years) partly replicated these findings, demonstrating an association of greater WASO and poorer performance on Trails B (Blackwell et al., 2011). Finally, Oosterman et al. (2009) found an association of actigraphic sleep-wake rhythm disturbance and deficits on standardised tasks of executive functioning, including working memory and inhibition, in 144 older community adults (50–91 years).

Research using PSG in older adults is rare, and somewhat contradictory. One study pointed to a role of SWS in executive functioning, showing that the proportion of SWS activity in the frontal lobes was positively correlated with waking performance on the Wisconsin Card Sorting Test (WCST; Heaton, 1981), and Tower of London task (TOL; Shallice, 1982) in 24 healthy older adults (61–75 years; Anderson & Horne, 2003). By contrast, a recent study found no association between REM sleep duration and performance on any tasks of executive functioning (Lafortune et al., 2014). There is a
need for further studies examining the link between sleep physiology and executive functioning in older adults.

1.3.6.3 Sleep and memory

(a) Memory deficits in adults

The past ten years have witnessed a considerable increase in studies providing evidence that sleep benefits memory. Two types of studies have been conducted. One approach assesses memory before and after a period of sleep, and demonstrates that information is strengthened or consolidated during sleep compared to an equivalent period of wake ('sleep-dependent memory consolidation paradigms'; see Payne, 2011 for review). The other approach assesses whether an individual’s usual sleep profile correlates with memory functioning (‘waking cognitive performance’). There is now considerable evidence in adults using both types of studies.

Using sleep-dependent memory consolidation (SDMC) paradigms, studies have demonstrated an active role of sleep in consolidating memories. The literature is vast and complex (see Walker & Stickgold, 2004 for a comprehensive review), and beyond the immediate scope of this thesis. Briefly, many aspects of memory and learning are dependent on brain plasticity, and there is evidence of such plasticity during sleep. The literature shows that sleep mediates many aspects of learning and memory consolidation, although the processes by which this occurs remain poorly understood.

Sleep deprivation paradigms provide support for the proposed role of sleep in memory. Studies consistently show impaired recall of information (Walker & Stickgold, 2006; Walker & van der Helm, 2009) and performance decrements after restricted sleep in list-learning, paired-associates, and semantic memory (Drake et al., 2001; Drummond et al., 2000). Neuroimaging studies also point to the effects of poor sleep on the medial temporal lobes, with functional effects on memory performance (Gais, Molle, Helms, & Born, 2002; Spencer, Gouw, & Ivry, 2007). For example, Drummond et al. (2000) found that sleep deprivation affected learning on a verbal memory task and was associated with dysfunctional activity in the hippocampus and medial temporal regions.

Much of the early sleep physiology research focused on REM sleep, although recent work has revealed an important role of NREM sleep, including SWS, in cognition.
Studies using PSG have indicated that REM sleep plays a role in procedural and emotional memory consolidation, whereas SWS is involved in the consolidation of declarative memory, including episodic memory (e.g., Gais & Born, 2004; Maquet, 2001). Some studies suggest that this distinction may be too simplistic, as REM sleep theta activity has been implicated in declarative memory consolidation (Fogel, Smith, & Cote, 2007), and Rauchs et al. (2004) provided evidence of a complementary role of SWS and REM sleep in tasks of episodic memory. However, the majority of previous research supports a role of REM sleep in the context emotional memory consolidation. Overall, much remains to be learned about the precise role of sleep stages in memory.

(b) Memory deficits in older adults

While the association of sleep and memory is well documented in adults, there is surprisingly little research in healthy older adults. Existing research, however, supports an association of sleep and memory in older populations.

For example, epidemiological studies point to the importance of self-reported sleep timing and quality in perceived memory ability in older adults (e.g., Foley, Ancoli-Israel, Britz, & Walsh, 2004). One study linked poor self-reported sleep quality (longer PSQI sleep latency) to greater deficits on tasks assessing long-term memory and verbal knowledge in older adults (Schmutte et al., 2007). A study using objective measures of sleep-wake patterns and cognition provides further support. Oosterman et al. (2009) found that community older adults ($N = 144$; 50–91 years) with sleep-wake cycle disturbance, investigated using seven nights of actigraphy monitoring, exhibited poorer performance on three standardised tasks of memory functioning, including verbal and visual memory, over and above the effects of age. In addition, clinical studies in older adults with OSA have shown that increased severity of sleep apnoea is associated with increased cognitive impairment, including memory decline (e.g., Aloia et al., 2003; Ju et al., 2012), albeit a dose-response relationship is not always found (Olaithe, Skinner, Hillman, Eastwood, & Bucks, 2014).

Studies using PSG in older adults are more mixed. Two SDMC studies (Backhaus et al., 2007; Mander et al., 2013) found positive associations of SWS and declarative memory. Consistent with these findings, (Van der Werf et al., 2009) found artificially induced disruption of SWS decreased memory encoding the next day, which was associated with
reduced hippocampal activation. By contrast, Scullin (2013) found no evidence of SDMC in older adults. Furthermore, there was a negative association of SWS and memory in older adults. That is, greater SWS was linked to poorer episodic memory in the older adults, but better memory in the younger adults. This suggests that the relationship between sleep and cognition might change with age, which Spiegel, Koberle, and Allen (1986) referred to as ‘functional-dissociation’.

Some studies have suggested a role of REM sleep in declarative memory, as opposed to emotional or procedural learning, in older adults. For example, Schredl, Weber, Leins, and Heuser (2001) implicated REM sleep in overnight declarative memory consolidation, and a recent study assessing ‘waking cognitive performance’ found that REM sleep duration was linked to better verbal learning in older adults the next day (Lafortune et al., 2014). However, overwhelmingly, previous studies point to a role of SWS over REM sleep in declarative memory.

Finally, sleep disorders, such as OSA, which increase with age (e.g., Harris, Glozier, Ratnavadivel, & Grunstein, 2009) have been shown to affect overnight memory consolidation (e.g., Kloepfer et al., 2009).

In summary, there are fewer studies in older adults relative to younger adults investigating the impact of sleep on cognition, and studies using PSG are lacking and inconsistent.

**1.3.7 The role of sleep in age-related changes in cognition**

In view of the rich evidence linking sleep to cognitive performance in ageing, as reviewed above, recent studies suggest that part of what we regard as age-related cognitive decline could be due to poor sleep (e.g., Nebes et al., 2009). Altena, Ramautar, Van Der Werf, and Van Someren (2010) reviewed the available evidence derived from studies using various sleep methodologies. The researchers observed that sleep deprivation effects on performance on cognitive tasks appear to mirror cognitive changes in old age. Similarly, insomnia-related changes in cognition and brain structure and function show a profile of performance, which relates to the integrity of the prefrontal cortical system, and which is closely linked to age-related changes. Altena et al. (2010), therefore, proposed that age-related changes might be due to poor sleep.
There is broad evidence for this proposal. Longitudinal studies implicate the role of sleep in future cognitive decline in older adults (Ferrie et al., 2011; Foley, Monjan, Simonsick, Wallace, & Blazer, 1999; Haimov et al., 2008; Jelicic et al., 2002). One study by Blackwell et al. (2014) found that sleep quality, assessed using the PSQI and actigraphy, was linked to subsequent cognitive decline on the Modified Mini-Mental State Examination (3MS; Teng & Chui, 1987) over a three to five year period in 2,822 older men (67+ years). Specifically, greater WASO and more long-wake episodes, as well as lower sleep efficiency, were associated with an increased risk of decline in executive functioning.

Further evidence derives from neurobiological studies that have shown that brain areas involved in age-related cognitive decline, such as the PFC and medial temporal lobes, are also affected by sleep (Altena et al., 2008; Hornung et al., 2005).

Finally, variability in cognitive performance in older adults has been linked to individual differences in self-reported sleep quality, over and above other potential moderators (e.g., educational level, the use of medications, smoking, psychiatric illness, substance abuse; Amer et al., 2013; Nebes et al., 2009). Studies using objective measures of sleep, such as actigraphy monitoring, also provide evidence that sleep is related to cognitive deficits in ageing (Miyata et al., 2013; Oosterman et al., 2009), and may even contribute to age-related cognitive decline. Given that these cognitive changes in memory and executive functioning are typical of a profile observed in ‘age-related cognitive decline’, sleep problems provide a plausible explanation for at least some of the cognitive changes observed in older age.

The mechanisms by which sleep is linked to age-related cognitive deficits are not fully understood. It is possible that poor sleep accelerates cognitive decline by increasing brain ageing, such as changes in brain structure and function, including changes in neurotransmitters, increased brain atrophy, and decreased synapses (Bruce & Aloia, 2006; Cabeza, 2001; Ferrie et al., 2011). Similarly, age-related deterioration of the PFC has been shown to lead to reductions in SWS and REM sleep seen in ageing (Mander et al., 2013). These stages also play a crucial role in the transferal of short-term memories to longer-term storage (Hornung et al., 2005). That these sleep stages show significant reductions with age thus provides a possible explanation for the link between sleep and age-related changes in cognition (Hornung et al., 2005; Mander et al., 2013; Neikrug &
It is, therefore, surprising that so few studies have investigated the link between changes in sleep architecture and cognition in older adults.

In summary, there is accumulating evidence that sleep contributes to age-related cognitive decline. However, PSG evidence is incomplete, and few studies have specifically addressed the question of whether sleep in older individuals is related to their cognitive profile. It is generally understood that the cognitive performance of older adults represents a decline associated with ageing. It is important to clarify at this point, that while age-related cognitive declines are well documented, this thesis does not investigate change in cognition over time. Rather, it assesses the cognitive performance of older adults at one point in time, which is assumed as a decline from previous function.

1.4 Sleep and age-related changes in cognition – a role of depression?

Few studies have specifically considered the role of clinical depression in the association between sleep and cognitive functioning in older adults, despite the fact that depression and sleep problems are closely linked, and that both sleep problems and depression are associated with cognitive deficits. In the following sections, a review of depression in ageing is presented; the links between depression and sleep are presented; followed by a review of the cognitive deficits associated with depression. Finally, a summary of the impact of depression on the relationship between sleep and age-related changes in cognition is offered.

1.4.1 Prevalence and presentation of depression in ageing

Some studies have reported that the susceptibility to depression tends to decrease, rather than increase, with age (Henderson et al., 1998; Jorm, 2000). However, as O’Connor (2006) suggests, findings of an apparent reduction in the prevalence of depression in older adults could be due to sampling bias. While formal diagnoses of depression are less common in older people (Dow, Tinney, & Bryant, 2009), when more inclusive measures of depression are employed, which do not exclude bereavement or dementia, estimates of between 6–20% in older adults in the community (Baldwin, 2008; Blazer, 2003; Chiu, Ames, Draper, & Snowdon, 1999), 48% for inpatients of a hospital (Bryant, Jackson, & Ames, 2009), and up to 50% in older adults in aged-care facilities
(Cummings, 2002) have been quoted. Therefore, depression is actually common in aged individuals (Fiske, Wetherell, & Gatz, 2009) and has even been described as the most common mental health problem in older adults (Garcia, 2008). Depression is cited as the leading cause of disability worldwide (WHO, 2008). Depression causes suffering, impairments in social and occupational functioning, decreased quality of life, and is associated with increased healthcare costs and higher rates of chronic medical conditions (Reddy, 2010). Early diagnosis and treatment of depression in older adults improves quality of life and functional status, and may even help prevent premature death (Blazer, 2003; Fiske et al., 2009).

Depression in older adults differs from depression in younger adults in terms of presentation (Fiske et al., 2009). Older adults are less likely to display affective symptoms and are more likely to present with sleep disturbance, psychomotor retardation, loss of interest in living, and cognitive changes (Fiske et al., 2009).

1.4.2 Sleep and depression

There is a large body of literature indicating a strong relationship between depression and sleep problems. Insomnia frequently co-occurs with Major Depressive Disorder (MDD; Riemann & Voderholzer, 2003; Tsuno, Besset, & Ritchie, 2005). Sleep impairment occurs in almost all individuals with MDD, with approximately 80% of patients reporting insomnia-like symptoms and daytime sleepiness (Armitage, 2007). MDD has also been associated with irregular sleep-wake times and circadian rhythm abnormalities, such as sleep fragmentation and phase advancement (Germain & Kupfer, 2008; Jagannath, Peirson, & Foster, 2013; Mendlewicz, 2008; Monteleone & Maj, 2008). Sleep disturbance has even been cited as one of the most debilitating features of the disorder (Sbarra & Allen, 2009).

Studies support a close association of sleep and depression in both younger and older adults. Evidence relating to younger adults is reviewed, before a more detailed review in older adults is presented.
1.4.2.1 The relationship between sleep and depression in adults

There are several lines of enquiry, which support the relationship between sleep and depression in adults. One type of evidence derives from cross-sectional studies (questionnaires, actigraphy and PSG), and another from longitudinal studies.

Cross-sectional studies show a link between depression and sleep (Kaneita et al., 2006; Lundt, 2005; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005; van Mill, Hoogendijk, Vogelzangs, van Dyck, & Penninx, 2010). Studies using actigraphy in both clinical and community samples of adults show disturbed sleep-wake patterns in depression (Korszun et al., 2002; Mendlowicz, Jean-Louis, von Gizycki, Zizi, & Nunes, 1999; Robillard et al., 2014). Furthermore, there are significant sleep architecture abnormalities associated with MDD. These include less SWS (Stage N3), shorter REM sleep latency, and more REM sleep, compared to healthy controls (e.g., Armitage, 2007; Buysse, Frank, Lowe, Cherry, & Kupfer, 1997; Modell & Lauer, 2007; Palagini, 2012; Pillai, Kalmbach, & Ciesla, 2011; Steiger, Dresler, Kluge, & Schuessler, 2013; Steiger & Kimura, 2010). In fact, a recent meta-analysis of 31 studies suggests that such sleep architecture alterations (e.g., REM latency and SWS), might prove useful in detecting MDD in adults (Arfken et al., 2014). Furthermore, other studies have pointed to a role of sleep architecture abnormalities in predicting future episodes of depression in remitted individuals (e.g., Buysse et al., 1997).

Longitudinal studies are also useful in examining the relationship between sleep and depression. However, the direction of the association of sleep and depression is not straightforward. Insomnia is a symptom of depression in the DSM-IV, which encourages a uni-directional interpretation of depression; that is, of depression impacting on normal sleep processes. This was the dominant view of the past and, accordingly, insomnia was seen simply as a symptom of psychopathology (see Riemann, Berger, & Voderholzer, 2001; Staner, 2010 for reviews). Longitudinal studies in adults have supported this proposition, demonstrating that depressive symptoms increase the risk of subsequent insomnia symptoms (Jansson & Linton, 2006; LeBlanc et al., 2009).

This view, however, is now outdated and studies show that sleep problems can precede depression (e.g., Ohayon & Roth, 2003) and predict subsequent depressive episodes (Breslau, Roth, Rosenthal, & Andreski, 1996; Buysse et al., 2008; Chang, Ford, Mead,
Cooper Patrick, & Klag, 1997; Eaton, Badawi, & Melton, 1995; Ford & Kamerow, 1989; Jackson, Szendur, Diamond, Byles, & Bruck, 2014; Jansson-Frojmark & Linton, 2008; Mallon, Broman, & Hetta, 2000; Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Neckelmann, Mykletun, & Dahl, 2007; Okajima, Komada, Nomura, Nakashima, & Inoue, 2012; Salo et al., 2012; Suh et al., 2013; Szklo-Coxe, Young, Peppard, Finn, & Benca, 2010). These studies indicate that sleep problems and/or insomnia symptoms are linked to increased depression risk (for meta-analyses see Baglioni et al., 2011; Riemann & Voderholzer, 2003).

Studies have also found that sleep disturbance can be a prodromal symptom of recurrent depression, preceding relapse (Ohayon & Roth, 2003; Perlis et al., 2006).

Overall, these studies suggest that the relationship between sleep and depression is complex and reciprocal (Krishnan & Hawranik, 2008; Sbarra & Allen, 2009), and is likely to be bidirectional (e.g., Alvaro, Roberts, & Harris, 2013; Jansson-Frojmark & Linton, 2008; Morphy et al., 2007; Siversten et al., 2012).

1.4.2.2 The relationship between sleep and depression in older adults

There is a strong relationship of sleep and depression in older adults, which may even be more pronounced compared to other age groups (Siversten et al., 2012). Evidence derives from different sources:

There is a plethora of cross-sectional studies supporting the close relationship of self-reported insomnia symptoms and/or sleep problems and depressive symptoms in community older adults (Almeida & Pfaff, 2005; Foley et al., 1995; Ito et al., 2000; Newman, Enright, Manolio, Haponik, & Wahl, 1997; Schechtman, Kutner, Wallace, Buchner, & Ory, 1997; Wu, Su, Fang, & Chang, 2012; Zimmerman, Bigal, Katz, Derby, & Lipton, 2013). For example, two studies have demonstrated a dose-dependent relationship between depressive symptoms on the 15-item Geriatric Depression Scale (GDS; Almeida & Almeida, 1999; Yesavage et al., 1983) and self-reported sleep disturbance on the PSQI and daytime sleepiness on the ESS in older men (N = 3051, 67+ years; Paudel et al., 2008), and older women (N = 3045, 70+ years; Maglione et al., 2012).
Studies reporting on the relationship between depression and sleep duration, however, are less consistent. Foley et al. (2004) failed to report an association of sleep duration and depression. Van den Berg et al. (2009), however, found that both short (< 6 hours per night) and long (≥ 9 hours per night) sleep durations on the PSQI, compared to durations of 7–8 hours, were linked to increased risk of a depressive disorder in 5019 older adults (58+ years).

There are fewer cross-sectional studies using objective measures of sleep disturbance, such as actigraphy, in older adults. One existing study by Maglione et al. (2012) reported a graded association between subclinical depressive symptoms on the GDS and both wake after sleep onset (WASO) and wake episodes longer than five minutes in 3045 older women (70+ years). Similarly, a study in 3051 older men (67+ years) reported an association of greater depressive symptoms on the GDS and longer sleep latency (Paudel et al., 2008). Furthermore, Naismith et al. (2011) found older men and women (46–86 years) with a history of lifetime depression (n = 44), including those with current depression, had greater WASO and poorer sleep efficiency compared to controls (n = 22). In addition, longer sleep latency was associated with greater depressive symptomatology on the GDS. A recent study investigating sleep-wake patterns in 238 individuals (12–90 years) with a lifetime history of depression (current and past MDD and bipolar disorder), however, did not replicate these findings (Robillard et al., 2014). While circadian rhythm disruption was found in the older age bracket (60+ years), alterations to sleep continuity (WASO and sleep efficiency) were associated with younger age and greater depression severity on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). This was an unexpected result, which the authors suggest may have resulted from the over-representation of older participants who were asymptomatic compared to symptomatic (and vice versa in the younger age bracket). Overall, actigraphy studies are lacking in older adults with depression, but the majority support disturbance of sleep-wake patterns.

Studies using PSG indicate that sleep architecture abnormalities in older adults with clinical depression are similar to those observed in younger adults, but may show additional age-related decline (e.g., Knowles & MacLean, 1990). The majority of these studies have linked depression to sleep architecture changes, such as shorter REM sleep latency (Kupfer, Reynolds, & Ehlers, 1989; Lauer, Riemann, Wiegand, & Berger, 1991; Reynolds et al., 1991), decreased SWS (Gillin et al., 1981), and increased REM sleep
(Reynolds et al., 1991; Reynolds III et al., 1985). Furthermore, a recent study of 2853 older community adults (65+ years) suggested an association of subclinical depression (assessed with the GDS) and sleep architecture abnormalities, such as less REM (Smagula et al., 2013). The authors conclude that depressed mood might be associated with accelerated age-related changes in sleep architecture.

Longitudinal studies in older adults support a reciprocal association of depression and sleep problems. Depression has been associated with the development of insomnia in older adults (Foley et al., 1999; Kim et al., 2009), but the majority of research focuses on insomnia symptoms and/or sleep problems as risk factors for depression (e.g., Martin, Fiorentino, Jouldjian, Josephson, & Alessi, 2010; Roberts, Shema, Kaplan, & Strawbridge, 2000). For example, Perlis et al. (2006) found that older women with insomnia had significantly elevated risk of developing their first episode of depression one year later compared to those with no insomnia. Similarly, Livingston, Blizard, and Mann (1993) found that sleep disturbance at baseline predicted presence of depression in healthy older adults two to three years later, despite controlling for other common risk factors of depression, such as older age and living alone, and Almeida, Alfonso, Yeap, Hankey, and Flicker (2011) found that longer self-reported sleep latency in older adults was associated with increased risk of depression even six years later.

In summary, evidence suggests that sleep problems and depression remain closely related in older adults. In fact, this association may even be stronger in older individuals. This has important clinical implications as the treatment of sleep problems may provide a means of reducing the risk of future depression (Cole & Dendukuri, 2003).

**1.4.3 Cognitive deficits in depression**

Cognitive deficits are common in individuals with depression, and form part of the DSM-IV criteria for MDD, describing a possible symptom as “diminished ability to think or concentrate, or indecisiveness” (American Psychiatric Association, 2000, in Snyder, 2013).

The main cognitive deficits in depression mirror the deficits in ageing, including problems with executive functioning, attention, memory, and processing speed.
1.4.3.1 Cognitive deficits associated with depression in adults

Most of the literature in adults with depression reports executive dysfunction (Fossati, Coyette, Ergis, & Allilaire, 2002; Harvey et al., 2004), including deficits in planning and problem-solving (Fossati, Ergis, & Allilaire, 2001; Naismith et al., 2003), inhibition (Gohier et al., 2009), verbal fluency (Ravnkilde et al., 2002), and working memory (Naismith et al., 2003; Rose & Ebmeier, 2006). In support, neuroimaging studies have linked cognitive deficits in depression to altered functioning of fronto-subcortical circuitry (Elliott, Rubinsztein, Sahakian, & Dolan, 2002; Schatzberg, 2002). For instance, cognitive deficits in depression have been associated with changes in the structure and function of the prefrontal areas, including over- and under-activation (Diener et al., 2012; Fitzgerald et al., 2006; Pizzagalli, 2011). The anterior cingulate cortex (ACC) has also been implicated in tasks of cognitive control, as well as subcortical structures such as the thalamus (Diener et al., 2012).

Problems with memory and learning are commonly reported in depression (see Goodwin, 1997 for review). For example, a meta-analysis of 14 studies reported a significant association of episodic memory deficits and greater self-reported depressive symptoms in adults with DSM-defined minor or major depression (McDermott & Ebmeier, 2009). Deficits are evident across multiple types of memory tasks, including visual memory (Naismith et al., 2003) and verbal recall and recognition (Bearden et al., 2006; Landrø, Stiles, & Sletvold, 2001; Vythilingam et al., 2004). In support, neuroimaging studies consistently demonstrate temporal lobe changes in depression, including dysfunction of the hippocampus, as well as reductions in hippocampal volume (Femenia, Gomez-Galan, Lindskog, & Magara, 2012; McKinnon, Yucel, Nazarov, & MacQueen, 2009; Sheline, Wang, Gado, Csernansky, & Vannier, 1996).

Problems with psychomotor functioning and a general slowing of information processing are reported extensively in the depression literature (Lee, Hermens, Porter, & Redoblado-Hodge, 2012; McIntyre et al., 2013; Porter, Gallagher, Thompson, & Young, 2003). These include both reduced motor and cognitive speed (Caligiuri & Ellwanger, 2000). A meta-analysis linked depression severity to slower processing speed in depressed adults (McDermott & Ebmeier, 2009).

Problems with concentration form part of the diagnostic criteria for depression (Potter & Steffens, 2007). Accordingly, deficits in attention are widely reported in adults with
depression (Gualtieri, Johnson, & Benedict, 2006; Huang et al., 2002; Landrø et al., 2001; Liu et al., 2002; Porter et al., 2003). Hammar (2003) has suggested that these deficits are especially pronounced in tasks requiring effortful attention.

### 1.4.3.2 Cognitive deficits associated with depression in older adults

Older adults with depression are an important population group to investigate given that both ageing and depression are associated with cognitive decline (Austin, Mitchell, & Goodwin, 2001). For example, depressive symptoms have been linked to future cognitive decline in older community adults (60+ years; Koehler, Thomas, Barnett, & O'Brien, 2010), and depression has even been identified as a risk factor for dementia (Jorm et al., 1991).

There are significant deficits in executive functioning in older adults with depression, including problems with selective attention, inhibition, response monitoring, and working memory (e.g., Alexopoulos et al., 2000; Baudic, Tzortzis, Dalla Barba, & Traykov, 2004; Beats, Sahakian, & Levy, 1996; Boone et al., 1995; Kiosses, Klimstra, Murphy, & Alexopoulos, 2001; Lockwood, Alexopoulos, & van Gorp, 2002; Nebes et al., 2000; Snyder, 2013). In support, neuroimaging studies have implicated prefrontal areas in depression in older adults (e.g., Lai, Payne, Byrum, Steffens, & Krishnan, 2000; Taylor, MacFall, et al., 2005).

Episodic memory impairment is common in depressed older adults (Baudic et al., 2004; Butters et al., 2004; Kramer-Ginsberg et al., 1999; Portella et al., 2003). However, some research has suggested that memory deficits may be secondary to executive dysfunction (e.g., Elderkin-Thompson, Mintz, Haroon, Lavretsky, & Kumar, 2007).

General slowing of cognitive processes is also one of the most consistent findings in the depression literature in older adults (Baune, Suslow, Engelien, Arolt, & Berger, 2006; Butters et al., 2004; Kramer-Ginsberg et al., 1999; Nebes et al., 2000; Portella et al., 2003; Sheline et al., 2006).

Finally, complaints of poor concentration are very common in older adults with depression (Fiske et al., 2009), and greater depressive symptoms have been linked to poorer performance on standardised tasks of attention in older community adults (Baune et al., 2006).
Overall, therefore, depression is associated with cognitive impairment, which may be even more pronounced in older adults compared to younger adults.

1.4.4 The impact of depression on the relationship between sleep and age-related changes in cognition

As reviewed in previous sections, sleep, ageing and depression appear to be closely related. In fact, it appears that there is an overlap in the brain mechanisms underlying depression and sleep, particularly the PFC, which impact on cognitive functions. Importantly, prefrontal cortical circuitry is also significantly affected in ageing (Cabeza & Dennis, 2012). This suggests that depression, sleep problems, and cognitive impairment in older adults might share common neural circuitry (Naismith et al., 2011). However, very few studies have specifically considered the role of clinical depression in the association between sleep and cognitive functioning, especially in older adults.

The existing studies that have considered depression have either controlled for subclinical levels of depression (e.g., Blackwell et al., 2011; Nebes et al., 2009; Schmutte et al., 2007), and/or have excluded older adults with clinical levels of depression (e.g., Amer et al., 2013; Bastien et al., 2003; Nebes et al., 2009; Oosterman et al., 2009; Tworoger, Lee, Schernhammer, & Grodstein, 2006; Vignola et al., 2000).

Some of these studies suggest that subclinical depression has significant effects on sleep and cognition, and their relationship. For example, Jelicic et al. (2002) found that controlling for depressive symptoms on the depression subscale of the Symptoms Checklist–90 (SCL–90; Arrindell & Ettema, 1986) removed a previously significant association between self-reported sleep on the SCL–90 and cognitive decline on the Mini Mental State Examination (MMSE; Folstein et al., 1975) in a sample of 838 community older adults (50+ years), suggesting that the relationship between sleep and cognition was explained by depression.

By contrast, other researchers have suggested that the contribution of depressive symptoms is negligible in the relationship between sleep and cognition in healthy older adults. For example, Nebes et al. (2009) found that levels of depression on the Geriatric Depression Scale (GDS; Yesavage et al., 1983) in 157 community older adults (65–80 years of age) were not responsible for the differences in working memory and
components of executive functioning between self-reported ‘good sleepers’ and ‘poor sleepers’ as defined by the PSQI. They concluded that sleep had independent effects on cognition in older adults. However, this study excluded participants with clinical levels of depression (GDS ≥ 15). It is therefore possible that while the relationship between sleep and cognition was not affected by sub-clinical depression, results may not apply to individuals with more severe levels of depression. Similarly, Vignola et al. (2000) found that subclinical depressive symptoms on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) did not explain self-reported attention deficits in 40 older adults with insomnia. However, they also excluded clinical levels of depression. Schmutte et al. (2007) found that self-reported sleep problems were associated with various cognitive deficits, including long-term memory and visual-spatial reasoning, regardless of depressive symptoms on The Zung Self-Rating Depression Inventory (Zung, 1965) in 375 community older adults (75–85 years). One study, also in older adults, reported a significant relationship between self-reported sleepiness and global cognitive function, independent of depressive symptoms (Jaussent et al., 2012). Furthermore, Ohayon and Vecchierini (2002) found that self-reported sleepiness predicted various cognitive functions (e.g., attention, delayed recall, prospective memory) on all domains of the Cognitive Difficulties Scale (CDS; McNair & Kahn, 1983) after controlling for psychological well-being.

Studies using objective measures of sleep have also reported significant associations of sleep and cognition after controlling for subclinical depression. Two actigraphy studies found independent associations of sleep-wake cycle disturbance and global cognitive impairment on the Mini-Mental State Examination (MMSE; Folstein et al., 1975) in older women (Blackwell et al., 2006), and older men (Blackwell et al., 2011), which were not explained by depressive symptoms on the GDS. Finally, studies investigating sleep architecture and cognition have typically excluded individuals with clinical depression (e.g., Backhaus et al., 2007).

Two studies support a close relationship between sleep and cognition in samples of depressed older adults. For example, Naismith et al. (2009) found that self-reported insomnia symptoms in 48 older adults (mean age = 59.6 years) with MDD were associated with poorer cognition (verbal fluency and memory), and that depression severity did not account for these relationships. Furthermore, a study by Naismith et al. (2011) assessed sleep-wake patterns using actigraphy in older adults with a lifetime
history of clinical depression (\(n = 44\)) compared to healthy controls (\(n = 22\)). In clinically depressed individuals, there were significant associations between sleep-wake pattern disturbances and cognitive deficits (including in memory and executive functioning), and these associations remained significant after controlling for depression severity. This research suggests that sleep problems are related to cognitive impairment in depression. However, Naismith et al. (2011) included participants with low levels of depression severity and did not compare the cognitive performance of the depressed group with the healthy control group.

Only one study has specifically investigated whether depression alters the relationship between sleep and cognition in older adults, which was published after the commencement of this thesis. Sutter, Zöllig, Allemand, and Martin (2012) tested whether depressive symptoms on the GDS moderated the relationship between self-reported sleep quality on the PSQI and objective measures of cognition, including processing speed, memory, and executive functioning, in 107 community older adults (61+ years of age). Results indicate that the relationship between poor overall self-reported sleep (Global PSQI scores) and performance on standardised tasks of executive functioning, including verbal fluency, reasoning and set-shifting, was limited to those participants with high levels of depressive symptoms. That is, poor sleep alone did not independently cause changes in cognitive performance, but the combined effect of poor sleep and depression was linked to deficits in executive functioning. Therefore, one hypothesis is that clinical depression alters, or exacerbates the relationship between sleep and cognitive impairment. The functional mechanism of this association is not well understood, but a possible explanation is that depression lowers the threshold of the brain’s coping mechanisms for sleep problems (Sutter et al., 2012).

Another explanation is that depression exacerbates sleep-related cognitive deficits. This is plausible given that sleep-wake cycle disturbances and sleep architecture changes, which are linked to cognitive deficits even in healthy older adults, are particularly pronounced in depression. To provide an example, research indicates that depression is associated with less REM sleep in older adults beyond the effects of normal ageing (Smagula et al., 2013). Given that REM sleep is important for memory processes (Gais & Born, 2004), it follows that depression might exacerbate sleep-related cognitive deficits in ageing, although evidence is lacking.
In summary, only one known study has directly addressed the question of whether depression impacts on the relationship between sleep and cognition (Sutter et al., 2012), but it was limited by its use of subjective measures of sleep and subclinical levels of depression. Hence, an overarching aim of this thesis was to investigate whether the presence of MDD changes the association between sleep and cognition in older adults, using objective measures (see Figure 5).

![Diagram showing relationships between Depression, Sleep, and Cognition](image)

*Figure 5. Direction of predicted relationships in this thesis*

### 1.5 Objectives of this thesis

The first objective of this thesis was to investigate how sleep changes with age. While there is a rich body of literature documenting age-related changes in objective measures of sleep, more inconsistent findings exist in questionnaire studies. This research project was particularly interested in investigating whether the lifespan changes in self-reported sleep were affected by subclinical levels of depression. Hence, a large community sample comprising adults ranging in age, using self-reports, seemed the most appropriate choice to answer this research question.

The second objective was to examine the impact of sleep on cognition (‘waking cognitive performance’), with a focus on older adults, an under-studied population group compared to younger adults. For the first time, this was done using a combination of self-reports and objective measures of sleep in the one sample. Special attention was given to the cognitive domains of attention, memory and executive functions, based on the cognitive changes that occur with ageing. Given that sleep has been found to contribute to variations in age-related cognitive decline, this thesis investigated the role of sleep in the cognitive profile associated with ageing.
Finally, since depression is common in ageing, and since depression and sleep have overlapping brain circuitry, the third objective of this thesis was to examine whether depression impacts on the relationship between sleep and cognition in older adults. For the first time, objective measures of sleep were used to investigate whether the presence of MDD would alter the relationship between sleep and cognition in older adults.

1.6 Thesis organisation

This thesis uses data from two population groups in order to address the study hypotheses. The first two studies report on data from a large sample of community adults of varying ages, with a range of subclinical levels of depression (Chapters 2 and 3). Consistent with methods most suitable for epidemiological samples, self-report measures of sleep and cognition were used in this sample.

Chapter 2 (published in the journal, *Behavioral Sleep Medicine*) presents the results of an investigation of how self-reported sleep quality changes with age and the influence of depressive symptoms and risk of sleep-disordered breathing (SDB).

Chapter 3 investigates the relationship between sleep and cognition, using standardised questionnaires.

The last two studies (Chapters 4 and 5) report on data from a smaller cohort of older adults (collected at the same time), comprising individuals with and without a diagnosis of current MDD. In this sample, objective measures of sleep (actigraphy, PSG) and neuropsychological tests were used.

Chapter 4 provides a comprehensive investigation into the sleep of older adults with a current versus past diagnosis of MDD, compared to healthy controls.

Chapter 5 examines the association of multiple aspects of objectively assessed sleep (sleep architecture and sleep-wake patterns) and cognitive performance, including attention, memory, and executive functioning in older adults. It also investigates whether the presence of current MDD impacts on the relationship of sleep and cognition using moderation analysis.
The final chapter (Chapter 6) provides a general discussion of the main results, strengths and limitations, implications and original contributions of this thesis, and suggestions for future research.

1.7 Summary of research methodologies used in this project

The empirical chapters are written for publication in peer-reviewed journals, with associated limitations on word counts. Accordingly, the methods used in this thesis are described below to provide a little more detail for the interested reader.

Chapters 1 and 2

These studies included standardised self-reports of sleep and cognition. Measures were selected based on their previous reliability and validity, and ease of administration for assessing sleep and cognition in a large community sample.

Sleep measures
The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) assesses sleep quality in the previous month.
The Berlin Questionnaire (BQ; Netzer, Stoohs, Netzer, Clark, & Strohl, 1999) assesses risk of sleep-disordered breathing (SDB).
The Epworth Sleepiness Scale (ESS; Johns, 1991) assesses daytime sleepiness.

Cognitive measures
The Cognitive Difficulties Scale (CDS; McNair & Kahn, 1983) assesses self-rated cognitive deficits, including attention-concentration deficits (the sole subscale analysed in this study).
The Multifactorial Memory Questionnaire (MMQ; Troyer & Rich, 2002) assesses self-reported memory functioning.
The Frontal Systems Behavior Scale (FrSBe; Grace & Malloy, 2001) assesses self-rated behaviours linked to deficits in prefrontal functioning, including apathy, disinhibition, and executive dysfunction (Total score = overall measure of executive functioning).
Clinical questionnaires
The Depression Anxiety and Stress Scale (DASS-21; Lovibond & Lovibond, 1995) measures the frequency of these core negative symptoms over the past week. The Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001) assesses levels of depression in the past 2 weeks. The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) is a short, diagnostic interview used to assess psychiatric conditions. It was selected to exclude co-morbid psychiatric disorders and to confirm diagnosis of Major Depression.

Chapters 3 and 4
These studies used a combination of subjectively and objectively assessed sleep and cognition. The use of several measures enabled a comprehensive investigation of sleep, including quality and duration, sleep-wake patterns, and sleep architecture in healthy and depressed older adults, and how specific aspects of sleep impacted on cognition.

Sleep measures
Actigraphy monitoring (worn for two weeks to ensure seven nights of viable data were collected) is a non-invasive method of measuring sleep-wake patterns (total sleep time, sleep efficiency, sleep latency, and wake after sleep onset; WASO). Portable-PSG enables investigation of sleep stages and has benefits over PSG conducted in a sleep clinic, as it is more naturalistic. There were two sleep studies in order to reduce the effect of unfamiliarity with the equipment (‘the first night effect’; Agnew, Webb, & Williams, 1966). The Somté device is effective in identifying sleep (sleep stages such as %REM, %SWS) and respiratory variables, and has good agreement with full PSG (Ferré et al., 2012).

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

Cognitive measures
The CogState is a computerised battery of neuropsychological assessment (http://www.cogstate.com.au) selected due to its widespread use, ease of administration, reliability and validity, and suitability for an older population (Collie, Maruff, Darby, & McStephen, 2003). There is also evidence that the CogState battery is sensitive to subtle cognitive impairment (e.g., fatigue and mild intoxication; Falleti, Maruff, Collie, Darby,
It includes measures of speed and accuracy covering various cognitive domains including attention, memory and executive functioning.

The Hayling Sentence Completion Test (HSCT; Burgess & Shallice, 1997) was included as an additional measure of executive functioning.

The National Adult Reading Test Revised (NART-R; Nelson & Willison, 1991) was selected as a measure of premorbid intellectual ability. The NART error scores were converted to the revised Wechsler Adult Intelligence Scale (WAIS-R) in order to estimate current intelligence level (IQ) according to this equation: Predicted WAIS-R Full Scale IQ = 130.6 – 1.24 x NART error score (S.E. est = 8.6; Nelson & Willison, 1991).

Clinical interviews and questionnaires
The MINI and the PHQ-9 were used to assess depression, as described above.

Cortisol
In addition to a diagnosis by the participant’s mental health practitioner, clinical interviews and questionnaires, collection of morning cortisol samples provided a biological measure of depression. Elevated waking cortisol is an index of the hypothalamic pituitary adrenal axis (HPA), which is proposed to play an important role in the pathophysiology of depression (Riemann et al., 2001).
Foreword to Chapter 2

Before investigating the associations of self-reported sleep and cognition, this chapter examines how self-reported sleep changes across the lifespan in a large sample of community adults aged 18–89 years. Objective studies show that sleep changes with age, however reports regarding subjective measures of sleep are less consistent. This could be because many studies have failed to consider the impact of depression and sleep-disordered breathing (SDB). Hence, this chapter investigates the association of age and self-reported sleep quality, controlling for depression and risk of SDB.
Chapter 2
Sleep and ageing: Examining the effect of psychological symptoms and risk of sleep-disordered breathing

Mellor, A., Waters, F., Olaithe, M., McGowan, H., & Bucks, R. S.

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This paper is included in its published form in the appendix.

Author contributions:
A. Mellor, F. Waters and R. S. Bucks developed the study concept, hypotheses, and design. A. Mellor selected the questionnaires. A. Mellor coded, analysed and interpreted the data under the supervision of R. S. Bucks and F. Waters. Michelle Olaithe and Helen McGowan assisted with participant recruitment and data collection and provided feedback on the manuscript. All authors approved the final version of the manuscript.
Abstract

Controversy exists as to whether self-reported sleep quality declines with age, despite changes in sleep being accepted as part of normal ageing. This study sought to investigate age-related differences in self-reported sleep quality, after controlling for conditions that are common with age, such as psychological symptoms and increased risk of sleep-disordered breathing (SDB). The Pittsburgh Sleep Quality Index (PSQI) was administered to a sample of 582 community adults (18–89 years), and the association between age and three factors of the PSQI (Sleep Efficiency, Perceived Sleep Quality, Daily Disturbance), and global scores, was examined controlling for depression, anxiety, stress, gender and SDB-risk. Results indicate that: (i) Before controlling for covariates, there was no significant relationship between age and all indices of self-reported sleep quality, with the exception of Sleep Efficiency. However, once depression, gender and SDB-risk were controlled for, a significant, yet small relationship was revealed between older age and poorer global sleep quality; (ii) there was no association between age and Perceived Sleep Quality, or Daily Disturbances, before or after controlling for relevant covariates; and (iii) depression, gender and SDB-risk were significant predictors of poorer sleep quality across the indices, but in general, did not have a marked impact on the relationship between age and sleep quality. In conclusion, results suggest that sleep problems are common across the lifespan, and that there were modest age-related differences in self-reported sleep quality, which were not due to depressed mood, gender, or risk of SDB.
Changes in sleep are part of the normal ageing process, and include alterations to sleep architecture and sleep-wake patterns, which have been reported using objective measures of sleep such as polysomnography (PSG) and actigraphy. The timing of sleep is typically shifted towards early bed and rise times, and there are increased night-time awakenings. In addition, sleep efficiency and the percentage of slow wave sleep are reduced (e.g., Knowles & MacLean, 1990; McCrae, 2009; Ohayon et al., 2004). However, controversy exists as to whether self-reported sleep quality, commonly assessed using questionnaires such as the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), also declines with age.

Several large epidemiological studies that assessed sleep across the lifespan (18–65+ years of age) have reported that on average, age is linked to an increase in subjective sleep complaints. These include difficulties with sleep maintenance and reduced sleep duration (Stein, Belik, Jacobi, & Sareen, 2008; Zeitlhofer et al., 2000). By contrast, other studies of older adults (60+ years of age), with equally large samples, have found that older adults do not necessarily rate their sleep quality as poor and are largely satisfied with their sleep once health conditions linked to ageing have been controlled for (Foley et al., 1995; Vitiello et al., 2002). Indeed, several studies have reported that subjective sleep quality in older adults was not significantly different from that of middle-aged adults (e.g., Middelkoop et al., 1996), and a recent study of adults aged 18–80+ years even found that older adults were less likely to report poorer sleep quality and tiredness than younger adults (Grandner et al., 2012).

One reason for such mixed findings could be that older people adjust their expectations about their sleep as they age (Buysse et al., 1991; Zilli, Ficca, & Salzarulo, 2009). Methodological issues might also explain these seemingly contradictory findings. For example, differences exist between studies in the aspect of sleep quality being investigated, and in the instruments employed to assess it. Sleep quality is a multifaceted concept that includes individual components such as satisfaction with sleep, sleep efficiency, and impact on daytime functioning (Magee, Caputi, Iverson, & Huang, 2008). Many epidemiological studies have used global scores from the PSQI to demonstrate age-related decline in sleep quality (e.g., Stein et al., 2008; Wong & Fielding, 2011; Zeitlhofer et al., 2000), although recent factor analytic studies suggest that the PSQI has a multidimensional structure that is better characterised by a two- or three-factor model (Cole et al., 2006; Magee et al., 2008). Specific associations might,
therefore, exist between age and PSQI sleep factors, but not with global scores. This suggests that the characterisation of different dimensions of sleep must remain an important focus in studies of sleep quality across the lifespan.

Psychological factors, such as depression, have also been cited as important factors that can affect the relationship between sleep quality and age. A large study by Roberts, Shema, and Kaplan (1999) for instance, showed that the trend for increased sleep disturbance across the lifespan (specifically assessed with symptoms of insomnia and hypersomnia) disappeared when levels of negative mood were controlled for. This finding is consistent with cross-sectional and longitudinal studies documenting the reciprocal relationship between sleep problems and psychological problems such as depression (Krishnan & Hawranik, 2008; Sbarra & Allen, 2009; Taylor, Lichstein, et al., 2005; Walker & van der Helm, 2009).

One possibility exists, therefore, that sleep problems in older adults may be directly linked to psychological symptoms such as depression, anxiety, or stress, rather than ageing per se. However, a recent study by Grandner et al. (2012) contradicts the suggestion that controlling for depression might impact on the way that age impacts on sleep quality. The authors adjusted their results for depression and found that age-related changes in self-reported sleep quality remained, despite the trend being in the opposite direction to the results reported by Roberts et al. (1999). That is, sleep disturbances decreased, rather than increased, across the lifespan despite controlling for depression. These findings suggest that depression might not have a direct impact on the relationship between age and self-reported sleep quality.

Given these discrepant results, an independent examination of this issue is warranted. In addition, with the exception of Grandner et al., few studies have examined the effect of depression, or indeed other psychological symptoms such as anxiety or stress, on sleep quality and age (Doi, Minowa, & Tango, 2003; Hall et al., 2000). In the current study, we sought to examine whether depression, anxiety and stress contributed to age-related differences in sleep quality as measured with the PSQI.

It is important to note that the relationship between sleep, depression and age is complex. Some studies have reported that the susceptibility to depression tends to decrease, rather than increase, with age (Henderson et al., 1998; Jorm, 2000). This
challenges our understanding that poor sleep in older adults should be linked to greater levels of depression and suggests that there could be differential associations between depression and sleep at different ages (Knowles & MacLean, 1990). However, as O’Connor (2006) suggests, findings of an apparent reduction in the prevalence of depression in older adults could be due to sampling biases, such that depression is actually highly prevalent in aged individuals (Fiske et al., 2009). Investigation into self-reported sleep quality across the lifespan, taking into account the possible effects of psychological symptoms such as depression, anxiety and stress, is therefore urgently needed to advance knowledge in this area.

Another potential confound that is often overlooked is the risk of sleep-disordered breathing (SDB). This under-diagnosed disorder includes a range of sleep-related respiratory abnormalities such as repeated episodes of high-resistance breathing, reduction in airflow and temporary cessation of breathing (Peppard et al., 2006). These symptoms increase with age and can dramatically affect sleep quality, with reports of restless and un-refreshed sleep (Amra, Farajzadegan, Golshan, Fietze, & Penzel, 2011; Flemons & Tsai, 1997). In addition, the association between SDB and depression, although complex, has been well established (Harris et al., 2009). Therefore, given its links to sleep quality, age, and depression, risk of SDB should be accounted for when investigating the relationship between age and sleep quality.

In summary, the current study aimed to investigate age-related differences in sleep quality as assessed using global PSQI scores and PSQI factors, controlling for depression, anxiety and stress, and risk of SDB. In addition, gender was controlled for, since women tend to report more sleep problems than men (Voderholzer, Al-Shajlawi, Weske, Feige, & Riemann, 2003).

Method

Participants
Recruitment was conducted via online advertising through universities, international survey and social networking sites, through advertisements in Australian community centres, and advertisement emails sent to individuals from volunteer groups (Western Australian Participant Pool, Director RS Bucks; and Older Adult Clinical Research Unit, H McGowan, F Waters). Once interest was expressed, participants were either
mailed a questionnaire or sent a link to the questionnaire online. Completion of the questionnaire was taken to indicate consent. All mailed questionnaires were completed and returned. In total, 615 participants completed the questionnaires. Only questionnaires completed by individuals over the age of 18 and from English-speaking and industrialised countries were used in the study. Exclusion criteria for all participants included: chronic infectious illness; neurological or neurodegenerative conditions; a history of moderate or severe traumatic brain injury; previous loss of consciousness > 30 minutes duration; treatment for substance abuse; or any other severe psychiatric disorder. After excluding participants according to the above criteria, data were analysed for 582 adults aged 18–89 years. The majority (559; 96.0%) of participants completed the online version of the questionnaire, whilst 23 (4.0%) completed the hardcopy version. Participants were recruited from three countries: Australia (n = 471); the United Kingdom (n = 31); and the United States of America (n = 80). There were no significant differences on BMI (p = .205) or gender (p = .320) between participants from different countries of origin. However, there were significant differences on global sleep quality scores (p = .004), hence country of origin was initially included as a covariate in the analyses. Approval was granted by both the North Metropolitan Mental Health Services and the University of Western Australia Human Research Ethics Committees.

Materials

Participants completed a demographics questionnaire, which asked about age, gender, medical history, education and occupation. They also completed the following scales:

The Pittsburgh Sleep Quality Index (PSQI; (Buysse et al., 1989), which assesses sleep quality in the previous month across seven domains (sleep duration, sleep disturbance, sleep latency, daytime dysfunction, sleep efficiency, subjective sleep quality, and use of sleep medication). Component scores are calculated on the basis of raw scores (scores range from 0–3, with higher scores indicating poorer sleep). Scores > 5 indicate significant sleep problems (Buysse et al., 1989). The PSQI has good estimates of reliability in a range of populations, including older adults, with alpha values quoted between .77–.83 (Beck, Schwartz, Towsley, Dudley, & Barsevick, 2004; Carpenter & Andrykowski, 1998; Gentili, Weiner, Kuchibhatla, & Edinger, 1995; Spira et al., 2012). In this study, Cronbach’s alpha was .71 for the global score and .61 for the 9-item sleep disturbance subscale. As reported by Buysse et al. (1989), alpha values were not
calculated for the other subscales due to the small number of items, rather correlations between each of the seven subscales and the global score were computed. These were all significant (all \( p < .001 \)), ranging from .37–.68, which compared well to the original article that reported coefficients between .35–.76. This suggests that although the PSQI has good reliability overall, the global score may not be a robust measure of sleep quality, as suggested by Cole et al. (2006). Accordingly, Cole and colleagues derived a three-factor model of the PSQI: Sleep Efficiency (comprising PSQI components: sleep efficiency and sleep duration), Perceived Sleep Quality (comprising PSQI components: use of sleep medication, sleep latency, and subjective sleep quality), and Daily Disturbances (comprising PSQI components: daytime dysfunction and sleep disturbance). This model has been used in a range of community studies with young and older adults, and in people with chronic fatigue syndrome (Aloba, Adewuya, Ola, & Mapayi, 2007; Cole et al., 2006; Mariman et al., 2012).

The Depression Anxiety Stress Scale (Lovibond & Lovibond, 1995), which measures the frequency of these core negative symptoms over the past week. There are seven questions per subscale and scores for each subscale range from 0–21, with higher scores indicating more symptoms. Internal consistency was high, \( \alpha = .94 \) overall; Depression: \( \alpha = .91 \); Anxiety: \( \alpha = .82 \); and Stress: \( \alpha = .87 \). These values are consistent with previous studies using community samples, which also have excellent reliability (Crawford et al., 2009; Crawford & Henry, 2005).

The Berlin Questionnaire (BQ; Netzer et al., 1999) is a self-report measure, which assesses risk of SDB. It includes questions about snoring and breathing during sleep (Category 1), feelings of fatigue and tiredness after sleep (Category 2), and BMI (which is calculated from height and weight), and whether people experience high blood pressure (Category 3). Risk of SDB was based on the following criteria: For Category 1, persistent symptoms (3–4 times a week) for two or more questions about snoring; for Category 2, persistent feelings of sleepiness during the day, and/or drowsiness during driving (3–4 times a week); and for Category 3, a history of hypertension (‘yes’, ‘no’, ‘don’t know’), or a BMI > 30. Only one response to either of these questions is needed to score this category as positive, and ‘don’t know’ responses are scored as negative. Whilst some participants did not know their height and weight (\( n = 50 \)), these individuals all scored at risk for Category 3 on the basis of the other question. Two or more positive responses across the three categories indicate a high risk of SDB. The BQ
has high internal validity. Cronbach’s alpha estimates range from .86–.92 (Netzer et al., 1999), and $\alpha = .59$ in this study.

**Statistical analysis**

Data were analysed using SPSS Version 20 (IBM, Inc). There were few missing PSQI data (3.3%; number of missing values / number of variables * number of participants), which came from the hardcopy questionnaires. Formatting of the online questionnaire did not allow participants to miss questions. Because the data was missing at random, Estimation Maximisation was used to replace missing values on PSQI subscales (Field, 2009). Relationships between study variables were investigated using Spearman’s correlation coefficients. The analyses included the PSQI global score and PSQI factors (Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances; Cole et al., 2006). Factor scores were calculated for each participant by multiplying the relevant component scores comprising each factor by the factor loadings presented by Cole et al. (2006), and summing them.

Multiple Regression Analyses were used to examine the effect of age on PSQI variables (global scores and the three factors). Given that gender, depression, anxiety and stress correlated significantly with age and PSQI factors, initially these variables were entered as covariates in the analyses. Similarly, given a significant age-difference between those at risk, and those not at risk of SDB, as indicated by a $t$-test, SDB-risk was entered as a covariate in the analyses. Anxiety and stress (measured with the DASS-21) were not significant predictors of the PSQI factors and were not included in the final regression models. Similarly, country of origin (dummy coded as AUS vs UK/US and US vs AUS/UK) was not a significant predictor of any sleep variables and was therefore not included in the final regression models.

**Results**

Table 1 reports descriptive statistics for the demographics and clinical questionnaire for the sample. Over 20% of the sample was characterised as ‘at risk’ of SDB as assessed by the Berlin Questionnaire (BQ; see Table 1).
Table 1. Descriptive statistics for the demographics questionnaire, including Berlin Questionnaire and Depression Anxiety and Stress Scale 21 (DASS-21; N = 582)

<table>
<thead>
<tr>
<th>Variable</th>
<th>N: %, Median [IQR], range or %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>33.30 [30], 18-89</td>
</tr>
<tr>
<td>18-29</td>
<td>257: 43.6%</td>
</tr>
<tr>
<td>30-39</td>
<td>78: 13.4%</td>
</tr>
<tr>
<td>40-49</td>
<td>71: 12.2%</td>
</tr>
<tr>
<td>50-59</td>
<td>69: 11.9%</td>
</tr>
<tr>
<td>60-69</td>
<td>63: 10.8%</td>
</tr>
<tr>
<td>70 +</td>
<td>47: 8.1%</td>
</tr>
<tr>
<td>Gender: Males</td>
<td>154: 26.5%</td>
</tr>
<tr>
<td>Body Mass Index(^1)</td>
<td>23.66 [6.02], 15.62-44.41</td>
</tr>
<tr>
<td>Risk of Sleep-Disordered Breathing (Berlin Questionnaire)</td>
<td>125: 21.1%</td>
</tr>
<tr>
<td>Completed tertiary education</td>
<td>391: 67.2%</td>
</tr>
<tr>
<td>Currently employed</td>
<td>316: 54.3%</td>
</tr>
<tr>
<td>Student</td>
<td>164: 28.2%</td>
</tr>
<tr>
<td>Seen a Doctor for mental health issues</td>
<td>127: 21.8%</td>
</tr>
<tr>
<td>DASS-21 - Depression</td>
<td>4 [8.5], 0-42</td>
</tr>
<tr>
<td>DASS-21 - Anxiety</td>
<td>2 [6], 0-42</td>
</tr>
<tr>
<td>DASS-21 - Stress</td>
<td>8 [10], 0-36</td>
</tr>
</tbody>
</table>

Note: \(^1\)N = 532 due to missing data; DASS-21 = Depression Anxiety Stress Scale- 21 item version.

Table 2 presents the descriptive statistics for the PSQI global and factor scores for the whole sample, and by decade of life\(^5\). Half the respondents (N = 306; 52.3%) scored > 5 on the global PSQI score, indicating significant sleep problems (Buysse et al., 1989).

\(^5\) One reviewer expressed concern about the disproportionate number of participants in the 18–29 year age bracket, which could have skewed results. Accordingly, we randomly selected 80 participants from the 18–29 year age bracket and reran all analyses; the results did not change.
Table 2. Descriptive statistics for the Pittsburgh Sleep Quality Index (PSQI; N=582)

<table>
<thead>
<tr>
<th></th>
<th>Cole et al.’s factor 1: Sleep Efficiency\textsuperscript{1}</th>
<th>Cole et al.’s factor 2: Perceived Sleep Quality\textsuperscript{1}</th>
<th>Cole et al.’s factor 3: Daily Disturbances\textsuperscript{1}</th>
</tr>
</thead>
<tbody>
<tr>
<td>M (SD), range</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All ages</td>
<td>6.4 (3.3), 0-17</td>
<td>2.0 (1.2), 0-6</td>
<td>1.4 (0.6), 0-3.6</td>
</tr>
<tr>
<td>18-29</td>
<td>6.1 (2.9), 0-14</td>
<td>2.0 (1.1), 0-5.1</td>
<td>1.3 (0.5), 0-3.6</td>
</tr>
<tr>
<td>30-39</td>
<td>6.6 (3.5), 1-15</td>
<td>2.2 (1.2), 0-5.5</td>
<td>1.4 (0.6), 0-2.9</td>
</tr>
<tr>
<td>40-49</td>
<td>6.4 (3.7), 1-16</td>
<td>1.9 (1.2), 0-4.4</td>
<td>1.4 (0.6), 0-3.1</td>
</tr>
<tr>
<td>50-59</td>
<td>6.8 (3.4), 2-17</td>
<td>2.1 (1.2), 0-6</td>
<td>1.4 (0.5), 0-2.4</td>
</tr>
<tr>
<td>60-69</td>
<td>6.4 (3.7), 2-16</td>
<td>1.8 (1.2), 0-5.1</td>
<td>1.3 (0.5), 0-3</td>
</tr>
<tr>
<td>70+</td>
<td>6.9 (3.8), 2-15</td>
<td>1.9 (1.3), 0-5.1</td>
<td>1.4 (0.5), 0.7-2.9</td>
</tr>
</tbody>
</table>

Note: \textsuperscript{1}Cole et al. (2006)

Table 3 reports bivariate Spearman’s correlations between PSQI global and factor scores, and age, depression, anxiety and stress (DASS-21). There was no significant correlation between age and global PSQI scores. Similarly, there was no relationship between age and Perceived Sleep Quality, or Daily Disturbances. However, there was a significant positive, yet weak, correlation between age and Sleep Efficiency (higher scores indicate more disturbance; $\rho = .16, p < .01$). In addition, increasing age was associated with less depression, lower anxiety and less stress, although those at risk of SDB were likely to be older, $t(578) = -5.43, p < .001$.

Table 3. Correlation matrix (Spearman correlation coefficients) for age, sleep quality (PSQI), and psychological symptoms (DASS-21)

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>DASS-Depression</th>
<th>DASS-Anxiety</th>
<th>DASS-Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-.17\textsuperscript{‡}</td>
<td>-.09\textsuperscript{†}</td>
<td>-.12\textsuperscript{‡}</td>
<td></td>
</tr>
<tr>
<td>PSQI Global</td>
<td>.03</td>
<td>.33\textsuperscript{‡}</td>
<td>.12\textsuperscript{‡}</td>
<td>.11\textsuperscript{‡}</td>
</tr>
<tr>
<td>Cole’s Factor 1: Sleep efficiency\textsuperscript{1}</td>
<td>.16\textsuperscript{‡}</td>
<td>.15\textsuperscript{‡}</td>
<td>.04</td>
<td>.02</td>
</tr>
<tr>
<td>Cole’s Factor 2: Perceived sleep quality\textsuperscript{1}</td>
<td>-.05</td>
<td>.23\textsuperscript{‡}</td>
<td>.07</td>
<td>.07</td>
</tr>
<tr>
<td>Cole’s Factor 3: Daily disturbances\textsuperscript{1}</td>
<td>-.02</td>
<td>.40\textsuperscript{‡}</td>
<td>.18\textsuperscript{‡}</td>
<td>.15\textsuperscript{‡}</td>
</tr>
</tbody>
</table>

Note: \textsuperscript{†} $p < .05$, \textsuperscript{‡} $p < .01$; Higher scores on all measures indicate greater disturbance; DASS = Depression Anxiety and Stress Scale; \textsuperscript{1} Cole et al. (2006).
We conducted four separate multiple regression analyses and as expected, gender, depression and risk of SDB were significant covariates across the sleep quality indices. Depression was linked to poorer sleep quality on global scores, Sleep Efficiency, Perceived Sleep Quality, and Daily Disturbances (β = .18 to .37, p < .01). Anxiety and stress were not related to sleep quality. Risk of SDB was linked to poorer sleep quality on global scores, Sleep Efficiency, and Daily Disturbances (β = .13 to .18, p < .01). Similarly, gender was a significant predictor of global sleep quality, Perceived Sleep Quality, and Daily Disturbances, with females reporting poorer sleep quality than males (β = .11 to .13, p < .01). Therefore, these results confirm the need to control for gender, depression and risk of SDB in the regression analyses (Table 4).

### Table 4. Regression analyses for all indices of sleep quality (PSQI)

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Covariate(s)</th>
<th>Predictor</th>
<th>Beta (95% CI) for predictor</th>
<th>R square covariates only (Step 1)</th>
<th>R square change after predictor added (Step 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSQI Global†</td>
<td>Gender†, Risk of SDB‡, DASS-Depression‡</td>
<td>Age</td>
<td>1.00 (0 - .03)†</td>
<td>.15‡</td>
<td>.01†</td>
</tr>
<tr>
<td>Factor 1: Sleep Efficiency†, ‡</td>
<td>Risk of SDB†, DASS-Depression‡</td>
<td>Age</td>
<td>.17 (.01 - .02)‡</td>
<td>.06‡</td>
<td>.03‡</td>
</tr>
<tr>
<td>Factor 2: Perceived Sleep Quality†, ‡</td>
<td>Gender†, DASS-Depression‡</td>
<td>Age</td>
<td>-.02 (-.01 - .01)</td>
<td>.06‡</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>Factor 3: Daily Disturbances†, ‡</td>
<td>Gender†, Risk of SDB‡, DASS-Depression‡</td>
<td>Age</td>
<td>.01 (0)</td>
<td>.19‡</td>
<td>&lt; .01</td>
</tr>
</tbody>
</table>

**Note.** † = p < .05, ‡ = p < .01; Significance values for the covariates are reported from the final model (Step 2); †: Higher scores indicate greater disturbance; DASS = Depression Anxiety and Stress Scale; ‡ Cole et al. (2006).

Despite the significant impact of depression, SDB-risk and gender on sleep quality, there was no substantive effect of these covariates on the relationship between age and the indices of sleep quality (see Table 4). Relationships between age and the three factors of the PSQI did not change once gender, depression, and SDB-risk were controlled for. That is, there was no relationship between age and Perceived Sleep Quality, or Daily Disturbances before or after controlling for relevant covariates. Further, there was a significant relationship between age and Sleep Efficiency before and after controlling for risk of SDB and depression, however the magnitude of this effect was very small and must not be over-interpreted; (R values between .01–.089 were considered small effects, those between .09–.249 as medium, and those greater
than .25 as large effects; Cohen, 1988). For PSQI global scores, a significant age-effect was revealed after controlling for relevant covariates, such that older age was associated with poorer overall sleep quality (gender, depression and risk of SDB; see Table 4) – however, the association between age and global sleep scores was of very modest strength and must, therefore, be interpreted with caution (Cohen, 1988).

Discussion

This study investigated the relationship between age and self-reported sleep quality, and whether factors such as depression, anxiety, stress and risk of SDB affected this relationship. Overall, findings indicate that: (i) after controlling for gender, depression and SDB-risk, there was a modest association between age and global sleep scores, which was largely driven by age differences in Sleep Efficiency, (ii) there was no relationship between age and Perceived Sleep Quality, and Daily Disturbances before and after controlling for relevant covariates; (iii) depression, gender and risk of SDB were significant predictors of some indices of sleep quality, but did not have a marked impact on the relationship between age and self-reported sleep quality. In summary, results suggest little age-related difference in self-reported sleep quality.

Prevalence of sleep problems in our sample

Overall, the prevalence of sleep problems in our sample was relatively high (52.3%). The global PSQI score in the current study (M = 6.4, SD = 3.3) was slightly higher than in other community studies, although still within one standard deviation of larger studies, e.g., (Wong & Fielding, 2011 M = 5.3, SD = 3.3); (Stein et al., 2008 M = 5, SD = 3); (Zeitlhofer et al., 2000 M = 4.6, SD = 3.7), suggesting that our sample was broadly representative of community adults.

The effect of psychological factors on the relationship between age and sleep quality

This study investigated how psychological factors, including depression, anxiety and stress, affected the relationship between age and sleep quality. Firstly, results indicate that depression, anxiety and stress decreased with age. These results are consistent with other studies of older adults recruited from the community (rather than care homes) that show that the risk of depression decreases with age (Jorm, 2000). Secondly, contrary to previous reports reporting that psychological factors such as depression may explain
sleep difficulties associated with ageing (e.g., Roberts et al., 1999), current results do not support this proposition. While depression was associated with reduced sleep quality, it was not linked to age-related sleep problems. This suggests that controlling for symptoms of depression had little impact on the relationship between age and sleep quality, as recently reported by Grandner et al. (2012).

The effect of SDB-risk on the relationship between age and sleep quality
In the current study, SDB-risk increased with age and depression, as previously reported (Amra et al., 2011). Increased risk of SDB was also associated with poorer sleep quality, however results indicate that it had largely no impact on the relationship between age and sleep quality. While this suggests that SDB-risk had little explanatory power in the relationship between sleep and age, the current study requires independent replication in a clinical sample of individuals with SDB, given the low alpha value for the Berlin Questionnaire (.59) in this community sample.

Age and sleep quality, and association with covariates (gender, psychological factors and SDB-risk)
Initial analysis of PSQI global scores revealed no significant relationship with age. This finding initially appears to support studies showing that sleep quality in older adults is not significantly different from middle-aged adults (e.g., Middelkoop et al., 1996). However, after controlling for gender, depression and SDB-risk, a significant relationship between age and global sleep quality emerged, such that increased age was associated with higher global scores on the PSQI (indicating poorer sleep quality). This would appear to suggest that these variables actually concealed age-related differences in global sleep scores, rather than contributed to them. In other words, age-related differences in global sleep quality were only apparent after depression, gender and SDB risk were controlled for. The size of the effect was small (Cohen, 1988), although significant. This finding is in direct contrast to previous research indicating that age differences in sleep quality disappear when controlling for variables such as depression (Roberts et al., 1999). However, Robert et al.’s sample comprised an older cohort only and not individuals across the lifespan. Therefore, the relationship between sleep and depression may change further as people reach older age. An examination of the relationship between age and PSQI factor scores (Cole et al., 2006), after controlling for gender, psychological factors and risk of SDB, also revealed interesting findings:
Firstly, Sleep Efficiency scores were positively associated with age, indicating increased time spent awake during the night in older participants. This is consistent with studies reporting reduced sleep efficiency and sleep duration as common sleep problems in older adults (Reid et al., 2006). However, again, this was not a robust finding given the small effect size. Secondly, Perceived Sleep Quality was not significantly associated with age, suggesting that participants’ ratings of their sleep quality did not differ much across the lifespan. Buysse et al. (1991) provided an explanation for this finding, suggesting that it could be because older adults adjust their expectations about their sleep quality, and are largely non-complaining about sleep changes. Thirdly, there was no relationship between age and Daily Disturbances, indicating that older adults did not report increased disruption to their daily lives as a result of poor sleep. This could be because older adults re-evaluate their expectations about how sleep impacts on their daily lives (Zilli et al., 2009), or they have reduced stressors and responsibilities, such as full-time work, study and childrearing, compared to younger adults, for whom the effects of poor sleep would, perhaps, be more pronounced.

In summary, current results revealed that sleep problems were common across the lifespan, and there was relatively little age-related difference in self-reported sleep quality across all indices of the PSQI. We also found that sleep-related risk factors such as gender, psychological symptoms and risk of SDB, although related to sleep quality, largely did not impact on the relationship between age and sleep quality. In other words, the modest age-related differences in self-reported sleep quality were not due to depressed mood, gender, or risk of SDB.

This study had some limitations. Firstly, given that this was a cross-sectional study, the direction of the relationships studied must be interpreted with caution. Future studies may wish to consider a longitudinal design, which would enable investigation of sleep changes with age. Secondly, while the use of the PSQI has its strengths (it is a well-validated measure and results are easily compared to previous studies), it relies on the respondent’s assessment of sleep quality over the previous month and therefore may be susceptible to recall bias. Future studies may wish to consider the use of sleep diaries, which might be less susceptible to recall bias and would provide information about sleep quality on a day-to-day basis.
Altogether, these findings stand in contrast to studies of *objective* sleep that consistently report poorer sleep quality with advancing age (Espiritu, 2008; Unruh et al., 2008). Self-reports, however, are an important dimension of sleep because, firstly, clinicians use subjective complaints to determine the need and type of treatment used, and secondly, there is often a discrepancy between polysomnography and subjective sleep reports (Unruh et al., 2008). Given that there is a pervasive view that older adults typically experience more sleep difficulties than younger adults (Vitiello, 2006), our finding that poor self-reported sleep quality is not a prominent feature of older age suggests that reports of new sleep problems from older individuals merit further clinical investigation. This highlights the evaluation of self-reported sleep as a valuable and independent dimension of sleep assessment.
Foreword to Chapter 3

Chapter 2 demonstrated that there were little age-related changes in self-reported sleep quality. In addition, while subclinical depression impacted on sleep, it did not impact on this relationship. This chapter examined the relationship between self-reported sleep and cognition in a community cohort (19–89 years), controlling for important confounds identified in Chapter 2, including depression and risk of SDB. Contrasting hypotheses concerning the impact of sleep on cognition are examined in this chapter.
Chapter 3
Sleep and cognition across the lifespan: an examination of competing functional models

Mellor, A., Bucks, R. S., McGowan, H., & Waters, F.

Author Contributions:
A. Mellor, R. S. Bucks and F. Waters developed the study concept, hypotheses, and design. A. Mellor selected the questionnaires and coded, analysed and interpreted the data under the supervision of R. S. Bucks and F. Waters. H. McGowan assisted with participant recruitment. A. Mellor drafted the paper and all authors provided critical revisions. All authors approved the final version of the manuscript.
Abstract

Neurobiological hypotheses propose that poor sleep affects cognition via direct biological brain changes, which impact on memory and executive functioning. In contrast, the ‘vigilance model’ suggests that poor sleep affects cognition via its impact on arousal levels and decreased attention. These models were examined using self-report data from 205 participants. Overnight sleep duration/efficiency was assessed using the Sleep Efficiency factor of the PSQI and provided a proxy for neurobiological models; and daytime consequences of poor sleep, reflecting low arousal levels (the PSQI Daily Disturbances factor and sleepiness on the Epworth Sleepiness Scale; ESS), were used as a proxy for the vigilance model. Two contrasting hypotheses were that: (i) Poorer sleep duration/efficiency would directly predict poorer memory and executive functioning; and (ii) Lower daytime arousal (PSQI Daily Disturbances and ESS sleepiness) would predict attention deficits, which would, in turn, predict poorer memory and executive functioning. Regression analyses revealed support for the vigilance model in that attention largely explained the impact of sleep on cognition. Results provide evidence of the critical role of low arousal on self-reported cognition. Importantly, the impact of Sleep Efficiency on cognition was accounted for by depression. While indirect measures of neurobiological processes have limitations, information provided regarding the self-reported timing and efficiency of sleep is worthwhile. Objective studies are needed to extend these findings, taking into account the role of attention and depression.
Sleep problems are common and can cause obesity, cardiovascular disease, reduced quality of life, depression and cognitive impairment (Ancoli-Israel, 2009). Three cognitive domains particularly vulnerable to sleep disturbance are attention, memory and executive functions (see review, Waters & Bucks, 2011). Separate theoretical frameworks have been developed, each providing a different explanation of how sleep impacts on cognition:

**Neurobiological models**

This ‘frontal lobe hypothesis’ proposes that sleep loss affects the frontal lobes by producing temporary changes in cerebral metabolism, causing problems with executive functioning (e.g., Horne, 1993). Proposed mechanisms include altered synaptic homeostasis and inhibition of neural plasticity (see Gorgoni et al., 2013 for review). In support, some sleep-related changes in cognition are consistent with mild prefrontal cortex (PFC) dysfunction (Killgore et al., 2008). Sleep deprivation studies also show decreased performance on PFC-dependent neuropsychological tasks (Turner, Salamat, Drummond, & Brown, 2007). In addition, clinical studies in individuals with sleep disorders link Obstructive Sleep Apnoea (OSA) to problems with executive functioning (Beebe & Gozal, 2002; Wallace & Bucks, 2013). Finally, neuroimaging studies show abnormal activity in the PFC as a result of sleep loss (e.g., Drummond et al., 1999).

The medial temporal lobe hypothesis proposes that sleep affects the pattern of activity of the medial temporal lobes and hippocampus, affecting the formation of memories (Gais et al., 2002). Evidence derives from experimental studies showing that sleep deprivation impacts on learning and activity in the hippocampus (Drummond et al., 2000). Furthermore, individuals suffering from OSA are consistently impaired on memory tasks (Wallace & Bucks, 2013), including tasks of verbal encoding (Ayalon, Ancoli-Israel, & Drummond, 2010). Finally, epidemiologic studies highlight the importance of self-reported sleep timing and quality in perceived memory ability (e.g., Foley et al., 2004).

**The vigilance model**

A contrasting argument to the neurobiological models suggests that sleep impacts on cognition via its effect on attention, rather than affecting memory and executive functioning directly. The vigilance model (Doran et al., 2001; Williams et al., 1959) proposes that the interaction between the homeostatic drive for sleep and circadian
factors causes fluctuating arousal levels, which cause “lapses” in attention. These attention deficits, it is argued, cause the memory and executive problems outlined above because of the central importance of attention in many higher-order cognitive tasks (Sarter, Givens, & Bruno, 2001).

Evidence derives from studies in both healthy and clinical samples. For example, sleep deprivation studies show that restricted sleep duration (< 6 hours a night) causes lapses in attention on tasks such as the Psychomotor Vigilance Task (PVT; e.g., Lim & Dinges, 2008). Epidemiological studies also indicate that sleepiness, a common consequence of poor sleep, is associated with self-reported attention problems (e.g., Ohayon & Vecchierini, 2002). Furthermore, decreased alertness due to fragmented sleep and sleepiness has been found to explain many of the cognitive deficits associated with sleep apnoea (e.g., Ayalon, Ancoli-Israel, Aka, McKenna, & Drummond, 2009). In fact, it has been suggested that contradictory findings in the OSA literature may be due to the failure to control for attentional capacity (e.g., Verstraeten, 2007).

For the first time, this study tested two models of sleep and cognition using self-report data in the same sample of community participants. While self-reports have limitations given their reliance on participants' perception of their sleep, advantages include larger sample sizes and greater statistical power. Furthermore, studies have shown that self-reported sleep quality corresponds well with objective measures of sleep, such as actigraphy (e.g., Girschik, Fritschi, Heyworth, & Waters, 2012; Spira et al., 2012). Finally, self-reports represent a valid dimension of sleep assessment, as they are used clinically to assess the need for and impact of treatment.

Overnight sleep duration/efficiency was assessed using the Sleep Efficiency factor of the PSQI and provided a proxy for neurobiological models. Thus, we predicted that the Sleep Efficiency factor of the Pittsburgh Sleep Quality Index (PSQI) would have direct effects on self-reported memory and executive functioning.

Daytime consequences of poor sleep, reflecting low arousal levels (the PSQI Daily Disturbances factor and sleepiness on the Epworth Sleepiness Scale; ESS), were used as a proxy for the vigilance model. Thus, we predicted that the impact of poor sleep would predict attention deficits, which would then impact on memory and executive functions via the key role of attention in these higher order cognitive functions. In these analyses,
the age of participants was also considered because of age effects on both sleep and cognition (Rabbitt & Lowe, 2000; Unruh et al., 2008). Also, since depression, risk of sleep-disordered breathing (SDB) and being female are associated with reduced sleep quality (Ancoli-Israel, 2005; Malhotra & White, 2002; Voderholzer et al., 2003), these variables were controlled for.

Method

Participants

Participants were 43 males and 162 females aged 19–89 years (see Table 1). Recruitment was conducted via advertisements in community centres and mail outs to volunteer groups. Ethics approval was granted by the North Metropolitan Mental Health Service, and the University of Western Australia Human Research Ethics Committees. Participation was voluntary and questionnaire completion was taken to indicate consent.

The majority (183; 89.3%) completed the questionnaire online. The hardcopy-version respondents were older than the online respondents but there were no other significant group differences.

Table 1. Descriptive statistics for the demographics questionnaire, including DASS-21

<table>
<thead>
<tr>
<th>Variable</th>
<th>Whole sample %, M(SD), range or Median [IQR], range</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>41.6(16.35), 19–89</td>
<td>205</td>
</tr>
<tr>
<td>Gender: Males</td>
<td>21%</td>
<td>43</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>23.53[4.75], 16.92–35.63</td>
<td>193</td>
</tr>
<tr>
<td>Completed tertiary education</td>
<td>74.60%</td>
<td>153</td>
</tr>
<tr>
<td>Currently employed</td>
<td>78.50%</td>
<td>161</td>
</tr>
<tr>
<td>Employed full-time</td>
<td>51.50%</td>
<td>105</td>
</tr>
<tr>
<td>Shift work</td>
<td>9.80%</td>
<td>20</td>
</tr>
<tr>
<td>Seen a Doctor for mental health issues</td>
<td>34.60%</td>
<td>71</td>
</tr>
<tr>
<td>DASS: Depression</td>
<td>4[10], 0–42</td>
<td>205</td>
</tr>
<tr>
<td>MMQ: Ability (A)</td>
<td>53[13], 15–80</td>
<td>205</td>
</tr>
<tr>
<td>FrSBe: Total</td>
<td>81[28], 50–162</td>
<td>205</td>
</tr>
<tr>
<td>CDS: and concentration</td>
<td>11[8], 0–34</td>
<td>205</td>
</tr>
</tbody>
</table>

Note. DASS = Depression Anxiety Stress Scale; MMQ = Multifactorial Memory Questionnaire; FrSBe = Frontal Systems Behavior Scale; CDS = Cognitive Difficulties Scale.
Materials

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) assesses sleep quality in the previous month. Higher scores indicate poorer sleep and Global scores > 5 indicate significant sleep problems (Buysse et al., 1989). In this study, Cronbach’s alpha, a measure of internal consistency, was .76 for the global score. We also used the three-factor model by Cole et al. (2006): Sleep Efficiency (comprising PSQI components: sleep duration and efficiency), Perceived Sleep Quality (comprising PSQI components: use of sleep medication, sleep latency, and subjective sleep quality), and Daily Disturbances (comprising PSQI components: daytime dysfunction and sleep disturbance). Factor scores were calculated for each participant by multiplying the component scores comprising each factor by the factor loadings by Cole et al. (2006) and summing them. Only Sleep Efficiency and Daily Disturbances factor scores were used in this study as they mapped clearly onto the proposed explanatory models.

The Epworth Sleepiness Scale (ESS; Johns, 1991) measures self-reported daytime sleepiness. Items ask about the tendency to doze in different situations. Participants respond on a scale from 0 = no chance of dozing, to 3 = high chance of dozing. Total scores range from 0–24, with a score of ≥ 10 across the eight items suggesting clinical levels of sleepiness. Reliability was good in this study (α = .78).

The Berlin Questionnaire (BQ; Netzer et al., 1999) measures risk of sleep-disordered breathing (SDB). Items include questions about snoring and breathing during sleep. Participants are identified as at high or low risk of SDB. The BQ has good internal consistency (Netzer et al., 1999); α = .65 in this study, likely due to low levels of SDB symptoms in this community sample.

The Depression Anxiety Stress Scale (DASS-21; Lovibond & Lovibond, 1995) measures the frequency of these core negative symptoms over the past week. Items from the depression subscale (7-items) only were used in this study. Scores range from 0–21, with higher scores indicating more symptoms. Internal consistency was high; α = .91, for the depression subscale.

The Multifactorial Memory Questionnaire (MMQ; Troyer & Rich, 2002) assesses self-reported memory functioning. The ‘Ability subscale’ consists of 20 items assessing self-evaluation of the respondent’s memory abilities. Participants are asked to rate the frequency of memory problems over the past two weeks (e.g., ‘How often do you forget to pay a bill on time?’) on a 5-point scale (0–4; ‘all the time’ to ‘never’), with higher scores indicating fewer memory errors. The MMQ had good internal consistency (α = .91, for the Ability scale in this study).
The Cognitive Difficulties Scale (CDS; McNair & Kahn, 1983) includes 26 items that assess cognitive complaints. The ‘Attention and concentration’ subscale was used in this study (11 items, e.g., ‘I cannot keep my mind on one thing’). Respondents are asked to rate the frequency of each statement on a scale (0–4; ‘never’ to ‘all the time’). Higher scores indicate more cognitive difficulties. Internal consistency was good in this study; $\alpha = .90$, for the ‘Attention and concentration’ subscale.

The Frontal Systems Behavior Scale (FrSBe; Grace & Malloy, 2001) measures behaviours associated with alterations to prefrontal functioning, including apathy (e.g., ‘I sit around doing nothing’), disinhibition (e.g., ‘I do things impulsively’) and executive dysfunction (e.g., ‘I make the same mistakes over and over, and do not learn from past experience’). For each item, respondents rate the frequency of their behaviour on a scale of 1–5 (‘almost never’ to ‘almost always’). Scores were totalled to assess behaviours associated with frontal lobe dysfunction. Internal consistency was good in this study; $\alpha = .93$.

**Statistical analysis**

Data were analysed using SPSS Version 20. There were few missing data (4.7%$^6$). Because data were missing at random, Estimation Maximisation was used to replace missing values (Field, 2009).

Associations between all study variables were explored using Spearman’s correlations due to the non-normal distribution of the data. Transformation did not produce normally distributed data, however there was no systematic misfit from the statistical models being used. Hierarchical multiple regression analyses were used to investigate the relationship between sleep and cognition. Demographic variables including depression, which were related to the cognitive variables (as determined by correlations or $t$ tests), were entered as covariates in Step 1. At Step 2, the predictors were added. Only significant predictors were entered in the final models. There was no adjustment for multiple comparisons, given that the relationships were predicted _a priori_ (Rothman, 1990).

Mediation models were calculated to determine the effect of attention on the relationship between sleep and cognition. If there was evidence of mediation in the

\[6 \text{ (number of missing values / number of variables) } * \text{ number of participants}\]
standard regression analyses, further analysis using the Preacher and Hayes (2004) indirect regression method was used to explore the nature of the indirect effect on a predictor-to-predictor basis. Bootstrapping was used to calculate 95% bias corrected and accelerated confidence intervals (CI) using 1000 bootstrap samples (Preacher & Hayes, 2004). This method samples repeatedly from the dataset and estimates the indirect effect in each resampled dataset. If the CIs do not cross zero, the indirect effect is significant.
Alpha was .05.

Results

Descriptive statistics for all sleep variables are reported in Table 2.

<table>
<thead>
<tr>
<th>Variable</th>
<th>M(SD), range or %</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSQI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime dysfunction</td>
<td>1(0.8), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Use of sleep medication</td>
<td>0.4(0.8), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep disturbance</td>
<td>1.2(0.5), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep latency</td>
<td>1.1(0.9), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep latency (minutes)</td>
<td>24.02(32.32), 1–360</td>
<td>205</td>
</tr>
<tr>
<td>Subjective sleep quality</td>
<td>1.1(0.8), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep duration</td>
<td>0.5(0.8), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep duration (hours)</td>
<td>6.95(1.25), 3–12</td>
<td>205</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>0.7(0.9), 0–3</td>
<td>205</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>85.58(13.04), 26.67–100</td>
<td>205</td>
</tr>
<tr>
<td>Global score</td>
<td>5.9(3.6), 0–16</td>
<td>205</td>
</tr>
<tr>
<td>Cole’s Factor 1: Sleep Efficiency</td>
<td>1(1.4), 0–5</td>
<td>205</td>
</tr>
<tr>
<td>Cole’s Factor 2: Perceived Sleep Quality</td>
<td>1.8(1.3), 0–5</td>
<td>205</td>
</tr>
<tr>
<td>Cole’s Factor 3: Daily Disturbances</td>
<td>1.3(0.6), 0–3.6</td>
<td>205</td>
</tr>
<tr>
<td>ESS</td>
<td>6.52(4.07), 0–20</td>
<td>205</td>
</tr>
<tr>
<td>Risk of Sleep-Disordered Breathing (Berlin Questionnaire)</td>
<td>20%</td>
<td>41</td>
</tr>
<tr>
<td>Has been diagnosed with a sleep disorder</td>
<td>9.3%</td>
<td>19</td>
</tr>
</tbody>
</table>

Note. PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; ¹ Cole et al. (2006).
### Table 3. Correlation matrix (Spearman correlation coefficients) for all study variables

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>DASS-D</th>
<th>ESS</th>
<th>PSQI Global score</th>
<th>Cole’s Factor 1&lt;sup&gt;1&lt;/sup&gt;: Sleep Efficiency</th>
<th>Cole’s Factor 3&lt;sup&gt;1&lt;/sup&gt;: Daily disturbances</th>
<th>CDS Att</th>
<th>FrSBe total</th>
<th>MMQ-A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
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<tr>
<td>DASS-D</td>
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<td>ESS</td>
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<tr>
<td>PSQI Global score</td>
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<tr>
<td>Cole’s Factor 1&lt;sup&gt;1&lt;/sup&gt;: Sleep Efficiency</td>
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<td>Cole’s Factor 3&lt;sup&gt;1&lt;/sup&gt;: Daily disturbances</td>
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<td>CDS Att</td>
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<tr>
<td>FrSBe total</td>
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<tr>
<td>MMQ-A</td>
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</table>

Note. DASS-D = Depression Anxiety Stress Scale – Depression subscale; ESS = Epworth Sleepiness Scale; PSQI = Pittsburgh Sleep Quality Index; CDS Att = Cognitive Difficulties Scale – Attention subscale; FrSBe = Frontal Systems Behavior Scale; MMQ-A = Multifactorial Memory Questionnaire – Ability subscale; † p < .05, ‡ p < .01; Higher scores indicate greater disturbance on all measures except MMQ-A, where higher scores indicate greater memory ability; <sup>1</sup>Cole et al. (2006).
At the bivariate level, older age was associated with better self-reported executive functioning on the FrSBe, and less severe depression on the DASS-21 (see Table 3).

More severe depression was linked to more attention and concentration problems on the CDS, reduced memory ability on the MMQ-A, and poorer executive functioning on the FrSBe. There was no effect of gender on attention, memory or executive functioning, $t(203) = .12, p = .908, d = 0.03$; $t(203) = .29, p = .774, d = 0.05$; and $t(203) = 1.82, p = .071, d = 0.31$, respectively.

More severe depression was associated with poorer sleep efficiency, greater Daily Disturbances, and greater daytime sleepiness on the ESS (Table 3). There was no effect of gender, $t(203) = -.20, p = .844, t(203) = -1.20, p = .234, t(203) = .53, p = .418$, or age on Sleep Efficiency, Daily Disturbances or sleepiness, respectively. There was no effect of SDB-risk on attention, memory or executive functioning, $t(203) = -.17, p = .869, t(203) = 1.57, p = .118$, and $t(203) = -1.52, p = .129$ respectively.

Simple correlations revealed that sleepiness and Daily Disturbances were associated with more attention and executive functioning problems and poorer memory ability. Poorer Sleep Efficiency was associated with more executive functioning deficits (see Table 3).

Hierarchical multiple regression analysis was used to test whether daytime sleep variables predicted attention problems after controlling for depression (see Table 4). Depression accounted for 16% of the variance in attention. Further, and in line with predictions based on the vigilance model, sleepiness and Daily Disturbances were significant predictors of attention: see Table 4; Step 1: $F (2, 202) = 28.68, p < .001$; Step 2: $F (3, 201) = 23.57, p < .001$, explaining an additional 10% of the variance.
Table 4. The impact of sleep on attention, memory and executive functioning (final models, significant covariates only)

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Covariate(s)</th>
<th>Predictors</th>
<th>Beta (95% CI) for predictor</th>
<th>R square covariates only</th>
<th>R square change mediator only</th>
<th>R square change after predictors added</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attention: (CDS)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDS: Attention¹</td>
<td>Depression¹ ‡</td>
<td>PSQI Daily Disturbances¹,²</td>
<td>.23 (1.07–4.30) ‡</td>
<td>.16 ‡</td>
<td>N/A</td>
<td>.10 ‡</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ESS¹</td>
<td>.21 (0.14–0.57)‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Memory (MMQ-A) with mediator Attention (CDS)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MMQ-Ability¹</td>
<td>N/A</td>
<td>CDS Attention¹ (mediator)</td>
<td>-.70 (-1.21– -0.86)‡</td>
<td>N/A</td>
<td>.49 ‡</td>
<td>.02 †</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PSQI Daily Disturbances¹,²</td>
<td>-.14 (-0.63– -0.08)†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ESS¹</td>
<td>.01 (-1.78–2.13)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Executive Functioning (FrSBe)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FrSBe Total score¹</td>
<td>Depression¹ ‡</td>
<td>PSQI Sleep Efficiency¹,²</td>
<td>.06 (-0.98–2.65)</td>
<td>.25 ‡</td>
<td>N/A</td>
<td>0</td>
</tr>
<tr>
<td>FrSBe Total score¹</td>
<td>Depression¹ ‡</td>
<td>PSQI Daily Disturbances¹,²</td>
<td>.15 (0.23–9.41)†</td>
<td>.25 ‡</td>
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<td>.03 †</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ESS¹</td>
<td>.11 (-0.10–1.12)</td>
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<td><strong>Executive Functioning (FrSBe) with mediator (Attention; CDS)</strong></td>
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<tr>
<td>FrSBe Total score¹</td>
<td>Depression¹ ‡</td>
<td>CDS Attention¹ (mediator)</td>
<td>.51 (1.15–1.84)‡</td>
<td>.25 ‡</td>
<td>.23 ‡</td>
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<td></td>
<td></td>
<td>PSQI Daily Disturbances¹,²</td>
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<td></td>
<td>ESS¹</td>
<td>0 (-0.55–0.52)</td>
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</table>

Note. †p < .05, ‡p < .01; Significance values are reported from the final model; ¹Higher scores indicate greater disturbance; ²Cole et al. (2006); ³Higher scores indicate greater ability; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; CDS = Cognitive Difficulties Scale, MMQ-A = Multifactorial Memory Questionnaire -Ability Subscale; FrSBe = Frontal Systems Behavior Scale.
Contrary to predictions based on the neurobiological model, Sleep Efficiency was not related to memory ability at the bivariate level. Rather, Daily Disturbances and sleepiness correlated with memory (see Table 3). In order to test the vigilance model, these variables were included in the regression model (see Table 4).

A mediation model was used to investigate the effect of attention on the relationship between Daily Disturbances and sleepiness, and memory ability (Table 4). Attention was a significant predictor of memory, accounting for 49% of variance; Step 1: \( F(1, 203) = 197.23, p < .001 \). However, the mediation model revealed that once attention had been added (bootstrapped indirect path = -3.59, CI = -6.14 to -1.29), Daily Disturbances was no longer a significant predictor of memory (see Table 4). By contrast, sleepiness remained a significant predictor of memory ability, 2% variance explained, even though attention partially mediated the relationships between sleepiness and memory ability (bootstrapped indirect path = -.46, CI = -.76 to -.21), Step 2: \( F(3, 201) = 69.41, p < .001 \). Therefore, results indicate that attention partially mediated the relationship between sleepiness and memory ability, whilst fully mediating the effect of Daily Disturbances on memory.

Contrary to predictions based on the neurobiological model, analysis revealed that the effect of Sleep Efficiency on executive functioning revealed in the correlation analyses (Table 3) was explained by depression; see Table 4; Step 1: \( F(1, 203) = 66.17, p < .001 \); Step 2: \( F(2, 202) = 33.47, p < .001 \). Effect sizes revealed that 25% of the variance in executive functioning was explained by depression and that sleep efficiency accounted for no further variance.

After controlling for depression, Daily Disturbances remained a significant predictor of executive functioning: see Table 4; Step 1: \( F(1, 203) = 66.17, p < .001 \); Step 2: \( F(3, 201) = 25.76, p < .001 \), explaining an additional 3% of the variance in executive functioning. Sleepiness was not a significant predictor of executive functioning.

As with memory, it is possible that changes in attention due to Daily Disturbances and sleepiness were associated with executive functioning deficits. Again, depression was controlled for and was significant; see Table 4; Step 1: \( F(1, 203) = 66.17, p < .001 \). As with memory, attention was also a significant predictor of executive functioning: Step 2: \( F(2, 202) = 90.18, p < .001 \), accounting for an additional 23% of variance. However,
with the addition of attention, Daily Disturbances was no longer a significant predictor of executive functioning (zero variance explained; bootstrapped indirect path = 4.80, CI = 1.68 to 8.99). Similarly, sleepiness was no longer a significant predictor of executive functioning, and bootstrapping revealed a positive indirect effect (bootstrapped indirect path = 0.65, CI = 0.25 to 1.20), indicating full mediation: Step 3: \( F(4, 200) = 44.73, p < .001. \)

In summary, results indicate that Sleep Efficiency did not predict memory ability or executive functioning after controlling for depression. Rather, Daily Disturbances predicted memory and executive functioning deficits via effects on attention, and sleepiness had both direct and indirect effects on memory ability. Finally, Daily Disturbances and sleepiness predicted attention and concentration problems. Age did not impact on the relationship between sleep and cognition.

**Discussion**

This study investigated the relationship between self-reported sleep and cognition. We tested two models which predicted that: i) poor night-time sleep duration/efficiency (PSQI Sleep Efficiency) would have direct effects on self-reported memory and executive functioning (a proxy for the neurobiological models); and ii) the daytime consequences of poor sleep (PSQI Daily Disturbances and ESS Sleepiness) would predict self-reported attention and concentration problems, which in turn would impact on memory and executive functioning complaints as a consequence of such effects on attention (a proxy for the vigilance model). Current findings provide partial support for the vigilance model.

*The daytime consequences of poor sleep*

Results indicate that the daytime consequences of poor sleep (Daily Disturbances and sleepiness) predicted attention and concentration complaints, which is consistent with the vigilance model. Results underscore experimental studies linking sleep loss to reduced performance on attentional tasks (e.g., Lim & Dinges, 2008) and epidemiological studies linking sleepiness to deficits in attention (e.g., Ohayon & Vecchierini, 2002).
Daytime sleep variables not only predicted attention, but also predicted executive functioning and memory deficits, largely via their impact on attention. The effect of Daily Disturbances on memory and executive functioning was entirely due to their effect on attention, whereas the effect of sleepiness on memory was only partially explained by its effect on attention. This suggests that although Daily Disturbances and ESS are related (see Table 3), they represent distinct components of arousal. Beyond the role of attention, sleepiness explained a small but significant 2% of variance in memory. These findings are broadly consistent with predictions based on the vigilance model, which proposes that poor sleep impacts on cognition via reductions in arousal, and with the idea that attention plays a critical role in the impact of poor sleep on higher order cognitive processes (Verstraeten, 2007).

Other studies have demonstrated a link between low arousal and cognitive deficits. For example, a meta-analysis indicated that sleepiness was the strongest predictor of school performance in children and adolescents, followed by sleep quality and duration (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010). Furthermore, a study in adolescents linked executive functioning problems to self-reported sleepiness, but not sleep duration (Anderson, Storfer-Isser, Taylor, Rosen, & Redline, 2009). However, attention was not controlled for, so its involvement remains speculative.

Previous studies have linked sleepiness to memory deficits (Caselli et al., 2002), although again, basic attentional processes were not controlled for making it difficult to determine the relative contribution of sleepiness and attention on memory ability. The current findings show that sleepiness was directly associated with memory and that this could not be fully explained by attention or depression.

Taken together, these results suggest that the impact of poor sleep on arousal levels and attention was the primary driver of the relationship between self-reported sleep and cognitive deficits. It is important to acknowledge that the processes by which poor sleep and arousal levels impact on attention and vigilance are not well understood. Neuroimaging studies have suggested a role of the cortico-thalamic network during tasks of sustained attention after sleep deprivation (Drummond et al., 2005; Tomasi et al., 2009). However, regardless of the mechanism of action, the vigilance hypothesis postulates that sleep impacts on cognition via effects on arousal and attention levels, which stands in contrast to the frontal and medial temporal lobe hypotheses that
predict direct effects of sleep on cognition.

Sleep duration and sleep efficiency
Contrary to predictions, Sleep Efficiency did not predict memory or executive functioning in this study. Results do not support predictions based on cognitive findings that are explained in terms of neurobiological changes in the frontal and medial temporal lobes due to poor sleep (e.g., Beebe & Gozal, 2002). Previous studies show changes in prefrontal cortex and hippocampal functions following acute sleep deprivation (e.g., Killgore et al., 2008), and in individuals with sleep disorders (e.g., Beebe & Gozal, 2002). However, there are important differences between studies showing direct effects of sleep on cognition, and in this study. Firstly, this study did not assess cognition objectively as in the aforementioned studies. Secondly, the current study used community adults instead of clinical populations (e.g., OSA) or sleep-deprived individuals. The severity of sleep problems and profile of cognitive deficits of these populations may differ – for instance, daytime sleepiness and depression may play a larger role in community samples, whereas other factors may be equally, if not more important, in clinical samples. Neurobiological deficits or health problems are common in clinical/sleep-deprived individuals, and these may affect a range of behaviours and cognition, only some of which may be attributable to poor sleep. For example, in clinical studies of OSA, the impact of sleep on cognition may be due to hypoxic effects on the brain, rather than poor sleep quality (Beebe & Gozal, 2002). Therefore, community samples are ideally placed to explore the interplay between sleep problems and cognitive profiles, as there is a smaller percentage of comorbidities (e.g., stress, hypertension), and/or medication effects commonly associated with clinical or sleep-deprived populations (e.g., Ho & Brass, 2011). In community samples, at least, we can conclude that attention and vigilance play a significant role in mediating the relationship between self-reported sleep quality and self-rated cognition.

Findings from community studies cited in support of the direct impact of sleep on memory and executive functioning may, in fact, have been driven by attention. For example, one study cites that sleep latency/continuity is associated with memory abilities (Foley et al., 2004). Yet, this finding is not inconsistent with the vigilance model, which advocates for the important role of attention processes. Thus, the predicted outcomes are similar according to both models, although the proposed mechanisms of action are different. It is possible that some of the findings cited in
support of effects of sleep on the frontal lobes and medial temporal lobes, may be explained, at least partly, by variations in attention levels.

**Depression, age, gender, and SDB-risk**

Subclinical depression was a significant covariate in the relationship between sleep and attention and executive functioning, and explained the relationship between poorer PSQI Sleep Efficiency and poorer executive functioning. That is, Sleep Efficiency only impacted on executive functions because of its impact on depression. This is consistent with findings that depression impacts on cognition (e.g., Mowla et al., 2008), and highlights the need to control for depression in all investigations of sleep and cognition. However, this finding is not consistent with previous research by Nebes et al. (2009), which found that effects of sleep remained significant after controlling for depression. This discrepancy in findings could be due to our use of self-reports of cognition, which might be more closely related to depression than objective measures (e.g., Zlatar, Moore, Palmer, Thompson, & Jeste, 2014). Furthermore, effects of poor self-reported sleep on self-reported cognition may be due to the mood effect of sleep loss, rather than sleep loss *per se*.

The direction of these relationships, is not straightforward as low mood can be a consequence of the daytime effects of poor sleep, and/or low mood can lead to poor sleep and daytime sleepiness (Sbarra & Allen, 2009). For example, Mayers, Grabau, Campbell, and Baldwin (2009) report that depression is associated with poor sleep satisfaction. However, regardless of the direction of the effect, self-reported sleep and depression were important determinants of cognitive complaints across the lifespan.

Age was not associated with self-reported cognitive deficits, which stands in contrast to previous reports linking age to cognitive complaints (e.g., Mol, van Boxtel, Willems, & Jolles, 2006). Rather, and critically, results indicate that sleep had a greater impact on self-reported cognition than age. These results have important clinical implications if they apply to objective measures of sleep and cognition, since they suggest that sleep may explain many lifespan changes in cognitive performance, including age-related cognitive changes. Given that sleep interventions can produce significant improvements in cognition (Ferini-Strambi et al., 2003), sleep assessment and treatment should be given a high priority in clinical practice (see Waters & Bucks, 2011 for clinical and practical recommendations).
Finally, there was no effect of gender or SDB-risk on the relationship between sleep and cognitive complaints.

**Limitations**

First, the use of self-reports in the current study limits the inferences that can be drawn about the relationships between sleep and cognition. For instance, it is important to acknowledge that poor sleep may be associated with negative evaluation of cognition (Broman, Lundh, Aleman, & Hetta, 1992) rather than objective cognitive deficits. However, self-reports are an important dimension of sleep quality, and have been found to correlate well with objective measures of sleep in some studies (e.g., Girschik et al., 2012).

Secondly, while mediation analysis is commonly conducted using cross-sectional data, as in the current study, it must be acknowledged that mediation involves causal processes that evolve over time, and therefore, technically requires a prospective study design (Maxwell & Cole, 2007).

Thirdly, while the use of sleep medications was of interest, given that Use of Sleep Medication was a component of one of the PSQI factor scores, it was not included in the final analyses to avoid the risk of excluding the critical variance of interest linked to the presence of depression. However, future research should consider the impact of other medications that may impact on the influence of sleep and cognition.

Finally, given the significant correlations between the PSQI sleep variables, problems of multicollinearity may have influenced the accuracy of our findings.

**Conclusions**

This study applied theoretical models of sleep and cognition to self-report data, controlling for age, gender, depression and risk of SDB. Overall, the current findings suggest a prominent role for the attention system in explaining how poor sleep impacts on cognition. Given the current findings that self-reported attention deficits play a critical role in the relationship between self-reported sleep and cognitive complaints in healthy individuals, it would be useful for studies using chronic/acute sleep-deprived samples to assess the contribution of attention to sleep-dependent cognitive performance. The current findings call for replication using objective measures of sleep.
that allow for investigation of multiple aspects of night-time sleep quality (e.g.,
actigraphy) and cognition, and which control for depression and attention.
Foreword to Chapter 4

Results of the past two chapters highlight the role of subclinical levels of depression in self-reported sleep and cognition. Chapter 2 found that subclinical depression was a significant predictor of self-reported sleep quality. Similarly, in Chapter 3 subclinical depression was a significant predictor of cognitive complaints and even explained the relationship between self-reported sleep efficiency and self-rated executive functioning. The next two chapters report on data in individuals with and without a clinical diagnosis of MDD to examine further the relationship between depression and sleep (Chapter 4), and the impact of depression on the relationship between sleep and cognition (Chapter 5). Therefore, the focus of the current chapter is to characterise the sleep of older adults with a current versus past diagnosis of MDD, using objective (actigraphy, polysomnography) and subjective (questionnaires) measures of sleep. One particularly important research question, which is pertinent to address before Chapter 5, is whether sleep problems are linked to current symptoms of depression (i.e., state-related), or whether they persist after remission, representing vulnerability to future episodes of depression (i.e., trait-related). This research has not yet been systematically investigated in older adults, and is key in characterising the groups in Chapter 5.
Chapter 4

Sleep abnormalities in older adults with and without major depression: evidence from polysomnography, actigraphy and questionnaires

Mellor, A., Waters, F., Maul, J., Sanders, K. A., McGowan, H., & Bucks, R. S.

Author Contributions:
A. Mellor, F. Waters and R. S. Bucks developed the study concept, hypotheses, and design. A. Mellor recruited and assessed all participants. A. Mellor coded, analysed and interpreted the data, and wrote the manuscript under the supervision of R. S. Bucks and F. Waters. Kathy Sanders provided access to her laboratory and offered training in cortisol collection. She also analysed all samples. Jennifer Maul provided training in polysomnography and analysed all sleep studies. H. McGowan provided access to the clinical sample, and feedback on the manuscript. All authors approved the final version of the manuscript.
Abstract

The link between sleep and depression in older adults is complex and not fully understood. This study aimed to characterise the sleep of 43 older adults (50+ years) with and without a history of clinical depression, using several methods (questionnaires, actigraphy, and home-polysomnography; PSG). Participants were divided into three groups using clinical interviews and questionnaire data: current Major Depressive Disorder (MDD; ‘Current’, \( n = 10 \)), a history of clinical depression (‘Past’, \( n = 14 \)), and no history of clinical depression (‘Never’, \( n = 19 \)). Morning salivary cortisol measures confirmed elevated waking cortisol levels in those with current depression. Examination of sleep measures revealed significantly less Slow Wave Sleep (%SWS), as well as greater self-reported sleep problems in the current depression group compared to other groups. By contrast, those with a past history of depression were not significantly different to those with no history of depression. Across the whole sample, more severe depression was associated with more %REM, less %NREM and less %SWS, as well as poorer self-reported sleep, but less time spent awake after sleep onset (WASO). In addition, increased cortisol was linked to more %REM and less %NREM sleep, as well as shorter actigraphic total sleep time and poorer sleep efficiency. Overall, sleep abnormalities were a feature of current MDD, and increased across the whole sample in a dose-dependent fashion with the severity of depression, and cortisol levels. The current findings argue against the idea that poor sleep persists after remission from depression, and rather suggest that current depressive symptoms, and cortisol levels, are more important determinants of sleep problems than a past history of depression.
Sleep problems occur in up to 50–60% of adults over the age of 65, and include difficulties with sleep onset and duration, and changes in sleep architecture such as reduced slow wave sleep (SWS; or Stage N3) and increased Rapid Eye Movement (REM) sleep (Ancoli-Israel, 2005; McCrae, 2009; Vitiello, 2006). The presence of depression in this population can exacerbate sleep problems (e.g., Sbarra & Allen, 2009). Indeed, sleep problems are common complaints in patients with depression (Buysse et al., 1989; Sbarra & Allen, 2009) and also cause problems for their carers (HigHet, Thompson, & McNair, 2005). However, the association between sleep and depression in older adults is not well understood. A better understanding of the link between sleep and depression in older adults has important clinical implications, as treating sleep problems has the potential to improve depressive symptoms (Sbarra & Allen, 2009).

Evidence for the link between depression and sleep impairment in older adults derives from studies using a range of different methodologies. For example, questionnaire studies indicate poorer self-reported sleep in older adults with a lifetime history of depression (current and past diagnoses) compared to healthy controls (e.g., Naismith et al., 2011). Research using overnight polysomnography (PSG) in older adults is sparse, but tends to support findings from studies in middle-aged adults that show sleep architecture abnormalities such as decreased SWS, reduced REM sleep latency, and increased REM sleep (e.g., Armitage, 2007; Hatzinger, Hemmeter, Brand, Ising, & Holsboer-Trachslser, 2004; Palagini, Baglioni, Ciapparelli, Gemignani, & Riemann, 2013; Pillai et al., 2011; Reynolds III et al., 1985). Older adults with clinical depression largely show similar sleep architecture changes (e.g., reductions in REM latency and SWS, and increased REM) to those seen in adults, but with additional age-related decline (e.g., Knowles & MacLean, 1990). For example, Gillin et al. (1981) showed that age-related sleep changes are more pronounced in depression and occur earlier than in healthy controls. Similarly, findings from Knowles and MacLean (1990) support the idea of ‘accelerated ageing’ in depression, reporting that the difference between the sleep of depressed and healthy people increases with age.

While PSG findings of sleep abnormalities in older depressed adults are largely convergent, studies using actigraphy recordings are fewer and findings are mixed. Actigraphy has been recommended by the American Academy of Sleep Medicine to provide accurate assessment of sleep-wake patterns (Morgenthaler et al., 2007).
Actigraphy output is recorded with an ‘actiwatch’, which allows 24-hour recording while the individual goes about their normal routine. Using actigraphy, Naismith et al. (2011) reported that the presence of depression in middle- to old-aged adults (46–86 years) was linked to a greater duration of night-time awakenings and reduced sleep efficiency, compared to age-matched healthy controls. In contrast, negative findings were reported by Lieverse (2014), who found poorer self-reported sleep quality, as assessed with questionnaires, in older adults (60+ years; \( n = 93 \)) with Major Depressive Disorder (MDD), but no differences on any actigraphic sleep variables relative to a group of healthy aged-matched controls (\( n = 74 \)). The reason for the discrepancies between these two studies is not clear, although it is likely that differences in age and clinical characteristics played a role.

Overall, sleep problems in older adults appear to be more pronounced in the presence of depression, but evidence derives primarily from studies using questionnaires and PSG, with the few existing actigraphy studies yielding contradictory findings. A related domain of interest is in the presence of sleep problems in older adults who have had previous episodes of depression, but who no longer show clinical depressive symptoms. This group is of considerable interest, given the suggestion that persistent sleep abnormalities during remission from depression may act as vulnerability markers for experiencing further clinical episodes (Anderson & Bradley, 2013; Palagini et al., 2013). In middle-aged adults, evidence exists of SWS and REM changes that persist during remission from depression (Hatzinger et al., 2004). A recent review also suggests that shortened REM latency and increased REM sleep in adults have the potential to predict relapse and recurrence (Palagini et al., 2013).

Polysomnography studies in older adults are less common than in middle-aged adults. Lee et al. (1993) tested older adults (60+ years) with depression (\( N = 15 \)) before and after remission, and found persistent REM sleep changes (shortened REM latency) during remission, suggesting that sleep impairment may not be confined to individuals with current depressive symptoms. Using actigraphy, another longitudinal study supported the idea of enduring sleep problems after successful treatment of depression. Ten older depressed outpatients (60+ years) showed persistent sleep impairments on total sleep time, wake after sleep onset (WASO), sleep efficiency, and sleep latency, after 60 days of antidepressant treatment (Rothschild-Fuentes et al., 2013). While these
studies point to enduring sleep problems in individuals with a past history of depression, they were limited by small sample sizes and the lack of a healthy control group.

An additional limitation of the above studies is their reliance on clinical interviews and self-reports for assessing the presence and severity of depression. Interviews and self-reports depend on subjective experiences and the participant’s willingness and motivation to report symptoms. Increasingly, studies are including measures such as morning salivary cortisol as a means of detecting biological abnormalities in depression. While cortisol cannot be considered a ‘pure’ measure of depression as it is linked to other factors such as alcohol consumption (Badrick et al., 2008), research shows that depression is linked to hyperactivity of the hypothalamic pituitary adrenal axis (HPA), which releases the stress hormone, cortisol (Riemann et al., 2001). Cortisol, therefore, has been suggested to play an important role in the pathophysiology of depression (Herbert, 2013). Since cortisol levels remain elevated in adults (Bhagwagar, Hafizi, & Cowen, 2003; Cowen, 2010) and older adults (Beluche et al., 2009) who have recovered from depression, it offers advantages over a clinical diagnosis as it assesses HPA axis activity as a continuous variable. In this way, elevated cortisol is thought to index a biological vulnerability for depression or, at least, act as a marker of previous episodes (Bhagwagar et al., 2003; Bhagwagar, Hafizi, & Cowen, 2005; Herbert, 2013). Freely circulating cortisol levels also impact on other systems, including sleep architecture and circadian rhythms, as well as human cognition (e.g., Born & Fehm, 2000; Hinkelmann et al., 2009; Reynolds et al., 2010; Van Reeth et al., 2000). In this study, the effects of cortisol secretion (as a biological index of depression levels) on sleep were examined.

For the first time, this study used multiple methods of sleep (questionnaires, actigraphy and home-PSG) to investigate the sleep of 43 older adults (50+ years) with and without a history of MDD. We aimed to: (i) provide a comprehensive evaluation of the sleep of older adults with a clinical diagnosis of depression; and (ii) compare the sleep characteristics of individuals with current versus past depression with those of a healthy age-matched control group in order to determine whether sleep problems are confined to those with current depression (i.e., state-related), or whether they also occur in those with a past history of depression (i.e., trait-related). A strength of the current study is the use of multiple methods of sleep assessment, which aids in providing a comprehensive profile of sleep in older adults.
Finally, since age (Ancoli-Israel, 2005), gender (Voderholzer et al., 2003), duration of depression (Jindal et al., 2002), sleep-disordered breathing (SDB) and associated hypoxemia (Malhotra & White, 2002), have all been shown to impact on sleep and depression, we examined for the contribution of these variables in the analyses.

**Method**

**Participants**

Recruitment of the non-depressed individuals was conducted via advertisements in community centres and mail outs to volunteer groups. Participants with a diagnosis of depression (past/present) were either outpatients from the North Metropolitan Adult Mental Health Older Adults outpatient services at Osborne Park Hospital, Perth, Western Australia, or individuals recruited from the community. Ethics committee approval was granted by both the North Metropolitan Mental Health Service and the University of Western Australia Human Research Ethics Committees.

Inclusion criteria for the depressed groups included a clinical diagnosis of unipolar MDD provided by a mental health professional, and age 50–80 years. Exclusion criteria for all participants included: chronic infectious illness; neurological or neurodegenerative conditions; a history of moderate or severe traumatic brain injury; previous loss of consciousness > 30 minutes duration; treatment for substance abuse; or any other severe psychiatric disorder. Forty-six participants were recruited. After excluding three participants due to a history of traumatic brain injury and bipolar depression, data were analysed for 43 participants (12 male, 31 female) aged 50–78 years.

**Procedure**

Assessments were conducted in the participant’s home over two visits. The first visit included: consent forms, questionnaires, clinical and sleep interviews, and the first night of PSG. Participants were also given an actiwatch for seven days, as well as a sleep diary. The second overnight sleep study was one week later, in order to reduce the ‘first night effect’ due to unfamiliarity with PSG (Agnew et al., 1966). Saliva samples to assess cortisol levels were collected by the participant at the second visit, which was a weekday.
Materials
Clinical assessment and classification

A demographics questionnaire included questions about age, gender, occupation, and mental health.

The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) is a 9-item questionnaire, assessing levels of depression in the past 2 weeks on a scale of 0 (not at all), to 3 (nearly every day). The PHQ-9 has excellent reliability ($\alpha = .94$ in the current study). The PHQ-9 has diagnostic value as items map onto the DSM-IV criteria for diagnosis of depression (Kroenke et al., 2001). Scores range from 0–24, with higher scores indicating more severe depression. A score of $\geq 10$ (for moderate depression) is recommended to identify clinical levels of depression (Kroenke & Spitzer, 2002).

The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) is a short, diagnostic interview for psychiatric evaluation. This was used to exclude comorbid psychiatric disorders and to confirm diagnosis of current depression.

Clinical interviews and questionnaire results were used to class participants into groups: (i) those with a diagnosis of depression by a mental health professional confirmed by the MINI, and a PHQ-9 score of $\geq 10$ were categorised as currently depressed (‘Current’); (ii) participants who had a diagnosis of clinical depression by a mental health professional in the past, but who were asymptomatic on interview and in the questionnaire were categorised as ‘Past’; and (iii) those who had never seen a doctor for mental health issues and had a PHQ-9 score of $< 10$ were categorised as ‘Never’ (see Table 1).

Salivary cortisol assessment

Three samples were taken: immediately after waking, wake+15 minutes, and wake+30 minutes. Participants were asked to collect their saliva in small vials and to store them in the freezer until collection. For each sample, salivary cortisol was assessed in duplicates using a commercial enzyme immunoassay (Salimetrics, LCC). All samples from the same participant were assessed in the same batch. The inter-assay coefficient of variation was 1.63% and 9.61% for high and low quality control standards.
respectively, indicating consistency between runs. The Area Under the Curve with respect to ground (AUCG) was calculated from the three samples as an estimate of total cortisol secretion over the first half an hour after awakening (y = 0; cortisol levels at time 0). This method is often used in cortisol research where there are repeated measurements over time (e.g., Fekedulegn et al., 2007; Vreeburg et al., 2010).

Sleep assessment

Sleep electrophysiology: Home-polysomnography (PSG) was conducted using the Compumedics ‘Somté’ device. It includes electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), airflow, body position, thoracic and respiratory belts, and blood oxygen saturation. Data were analysed by a Senior Sleep Technologist (J Maul) and checked over by another Sleep Technologist (A Mellor) according to the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events (Iber, Ancoli-Israel, Chesson, & Quan, 2007a). Data from the second sleep study are reported. Outcome variables included REM latency (minutes), and the percentage of REM and NREM sleep, and SWS (Stage N3). In order to assess for sleep-disordered breathing (SDB) and associated hypoxia, analyses included the Respiratory Disturbance Index (RDI), and the Oxygen Desaturation Index (ODI-3; no. of times per hour that there is a decrease of ≥3% in blood oxygen saturation).

Actigraphy: Participants wore an ‘actiwatch’, a wrist-worn device that non-invasively monitors sleep-wake cycles, for the duration of two weeks to ensure seven nights of viable data were collected. Output measures were mean total sleep time (minutes), sleep efficiency (%), sleep latency (minutes), and minutes spent awake after sleep onset (WASO). Participants kept a sleep diary to enable interpretation of actiwatch data. Actigraphic data were checked against the sleep diary by two researchers to ensure concordance (AM, FW). The Actiwatch Spectrum (MiniMitter Philips) was used in this study. Seven days of actigraphy recording has been shown to be sufficient to obtain stable measures of domains of sleep quality (Knutson et al., 2007; Tworoger et al., 2005).

Participants also completed a daily sleep diary to record details such as sleep and wake times, whether they took any naps, and whether the watch was removed for any period.
of time. This information was used to cross-validate and edit the actigraphy data as needed. Each actigram was visually inspected and compared to the sleep diary in order to identify any major discrepancies. Time in bed was adjusted by the scorer in cases where there was a discrepancy of greater than one hour between the diary and actigraph, so it was consistent with the sleep diary.

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) assesses sleep quality in the previous month. Higher scores indicate poorer sleep. Global scores > 5 indicate significant sleep problems (Buysse et al., 1989). In this study, Cronbach’s alpha was .87 for the global score. The three factor model by Cole et al. (2006), was applied generating three scores: Sleep Efficiency (comprising PSQI components: sleep efficiency and sleep duration), Perceived Sleep Quality (comprising PSQI components: use of sleep medication, sleep latency, and subjective sleep quality), and Daily Disturbances (comprising PSQI components: daytime dysfunction and sleep disturbance), calculated by multiplying the component scores comprising each factor by the factor loadings published by Cole et al. (2006), and summing them.

**Statistical Analysis**

Data were analysed using SPSS Version 20 (IBM). Prior to analysis, data were ‘Log10 transformed’ where required (Field, 2009). Because the self-report data (PSQI) were missing completely at random (Little’s MCAR $p > .05$), Estimation Maximisation was used to replace missing values for self-report data (Field, 2009).

In order to investigate differences on demographic and sleep variables between the depression groups, and given that predictions were made a priori, separate one-way analyses of variance (ANOVA) were conducted using one-tailed tests. Estimates of effect sizes included partial $\eta^2$ for continuous data or Cramer’s Phi for categorical data. Alpha throughout was .05. Post-hoc pairwise comparisons were used to investigate the nature of any between-group effects. There was no adjustment for multiple comparisons, given that the relationships were predicted *a priori* (Rothman, 1990). Gabriel’s method was selected for post-hoc comparisons based on the unequal sample size of the groups (Field, 2009). Pearson’s correlations were conducted to investigate the relationship of depression severity and cortisol and sleep measures. One-tailed tests were used when the direction of the effect was predicted in advance. Variables that were
related to the dependent variable (as determined by correlations or t tests) were added as covariates in subsequent analyses of covariance (ANCOVA) and partial correlation analyses (Overall & Woodward, 1977).

While the use of sleep medications and antidepressants was of interest, these variables were not included as covariates in the final analyses to avoid the risk of excluding the critical variance of interest linked to the presence of depression. That is, taking antidepressant medication is somewhat synonymous with depression.

Results

Descriptive statistics for demographic and sleep variables for the three groups are reported in Table 1. Apart from the use of antidepressant medication, where the Current group took the most, there were no between-group differences in age, gender, time since first diagnosis of depression, or measures of sleep-disordered breathing (RDI, and ODI-3).

Group differences in sleep variables
On PSG measures, the Current group had significantly less %SWS compared to the Never group ($p = .022$), whereas the Past group did not differ from the other groups ($p > .050$). The Past group had significantly longer REM latency than the Never group ($p = .004$), whereas the Current group did not differ from either group ($p > .050$). These differences remained significant after controlling for age: $F(2, 39), = 8.91, p < .001$.

On self-report measures, the Current group had greater PSQI Global and Cole’s PSQI factor scores (Sleep Efficiency, Perceived Sleep Quality, Daily Disturbances), indicating poorer self-reported sleep quality, compared to the Past and Never groups, who did not differ (see Table 1). Poorer Sleep Efficiency was associated with age, and SDB (oxygen saturation desaturations below 3%; ODI-3), hence these variables were entered as covariates, but this did not affect the group difference for Cole’s Sleep Efficiency, $F(2, 33), = 11.74, p < .001$, partial $\eta^2 = .42$. Age and ODI-3 were not significant covariates of Perceived Sleep Quality or Daily Disturbances.

There were no significant group differences in any actigraphic sleep variables (see Table 1).
<table>
<thead>
<tr>
<th>Variable</th>
<th>Never</th>
<th>$^a$</th>
<th>Past</th>
<th>$^a$</th>
<th>Current</th>
<th>$^a$</th>
<th>$p$</th>
<th>Pairwise comparisons</th>
<th>Partial $\eta^2$ or Cramer’s Phi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>62.32(7.34), 50–79</td>
<td>19</td>
<td>59.07(4.60), 52–68</td>
<td>14</td>
<td>59.80(6.76), 51–74</td>
<td>10</td>
<td>.329</td>
<td>2&gt;.01, 1=0</td>
<td>.05</td>
</tr>
<tr>
<td>Gender: Males</td>
<td>5(26.3%)</td>
<td>19</td>
<td>2(14.3%)</td>
<td>14</td>
<td>4(40%)</td>
<td>10</td>
<td>.361</td>
<td>2&gt;.01, 1=0</td>
<td>.22</td>
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<tr>
<td>Currently employed</td>
<td>12(63.2%)</td>
<td>19</td>
<td>8(57.1%)</td>
<td>14</td>
<td>5(50%)</td>
<td>10</td>
<td>.789</td>
<td>2&gt;.01, 1=0</td>
<td>.11</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHQ-9 scores$^2$</td>
<td>2.21(1.5), 0–8</td>
<td>14</td>
<td>3.64(3.72), 0–9</td>
<td>11</td>
<td>16.60(4.62), 11–24</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.78</td>
</tr>
<tr>
<td>Time since diagnosis (years)</td>
<td>N/A</td>
<td>0</td>
<td>11.69(12.68), 0–40</td>
<td>13</td>
<td>15.90(16.79), 0–52</td>
<td>10</td>
<td>.500</td>
<td>2&gt;.01, 1=0</td>
<td>.02</td>
</tr>
<tr>
<td>Taking antidepressants</td>
<td>2(10.5%)</td>
<td>19</td>
<td>7(50%)</td>
<td>14</td>
<td>8(80%)</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.57</td>
</tr>
<tr>
<td>Cortisol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sample 1 (Wake)</td>
<td>9.28(2.06), 2.07–35.16</td>
<td>16</td>
<td>8.67(1.48), 2.30–18.06</td>
<td>11</td>
<td>14.98(4.45), 3.75–42.69</td>
<td>8</td>
<td>.157</td>
<td>2&gt;.01, 1=0</td>
<td>.07</td>
</tr>
<tr>
<td>Sample 2 (Wake+15)</td>
<td>10.62(1.66), 2.21–24.75</td>
<td>16</td>
<td>11.32(1.96), 1.78–25.06</td>
<td>11</td>
<td>20.72(6.36), 4.36–58.12</td>
<td>8</td>
<td>.139</td>
<td>2&gt;.01, 1=0</td>
<td>.08</td>
</tr>
<tr>
<td>Sample 3 (Wake+30)</td>
<td>13.80(1.95), 2.64–31.41</td>
<td>16</td>
<td>13.82(2.15), 3.19–28.14</td>
<td>11</td>
<td>23.44(5.75), 7.50–48.72</td>
<td>8</td>
<td>.122</td>
<td>2&gt;.01, 1=0</td>
<td>.09</td>
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<tr>
<td>AUC$_G$, cortisol</td>
<td>332.39(199.28), 82.47–817.36</td>
<td>16</td>
<td>338.54(174.90), 67.77–684.21</td>
<td>11</td>
<td>598.98(649.20), 168.35–1557.42</td>
<td>8</td>
<td>.109</td>
<td>2&gt;.01, 1=0</td>
<td>.09</td>
</tr>
<tr>
<td>ESS (daytime sleepiness)$^3$</td>
<td>6.5(4.1), 2–18</td>
<td>19</td>
<td>9.5(4.2), 4–16</td>
<td>13</td>
<td>7.5(4.4), 0–14</td>
<td>10</td>
<td>.073</td>
<td>2&gt;.01, 1=0</td>
<td>.09</td>
</tr>
<tr>
<td>PSQI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global score$^4$</td>
<td>6(3.6), 1–15</td>
<td>19</td>
<td>6(3.7), 3–15</td>
<td>14</td>
<td>15.8(3.5), 10–21</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.57</td>
</tr>
<tr>
<td>Global &gt; 5</td>
<td>8(42.1%)</td>
<td>19</td>
<td>9(46.3%)</td>
<td>14</td>
<td>10(100%)</td>
<td>10</td>
<td>.004</td>
<td>2&gt;.01, 1=0</td>
<td>.48</td>
</tr>
<tr>
<td>Cole’s Sleep Efficiency$^5$, $^6$, $^7$, $^11$</td>
<td>1.5(1.5), 0–5</td>
<td>19</td>
<td>1.1(1.4), 0–5</td>
<td>14</td>
<td>3.8(1.5), 1.7–5</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.35</td>
</tr>
<tr>
<td>Cole’s Perceived Sleep Quality$^8$, $^9$, $^11$</td>
<td>1.8(1.4), 0–5.1</td>
<td>19</td>
<td>2.2(1.4), 0.7–5.1</td>
<td>14</td>
<td>5(1), 3.8–6</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.52</td>
</tr>
<tr>
<td>Cole’s Daily Disturbances$^6$, $^7$, $^11$</td>
<td>1.2(0.5), 0–2</td>
<td>19</td>
<td>1.5(0.7), 0.7–2.9</td>
<td>14</td>
<td>2.4(0.5), 1.9–3.6</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.47</td>
</tr>
<tr>
<td>Use of Sleep Medication component</td>
<td>0.2(0.42), 0–1</td>
<td>19</td>
<td>0.46(0.88), 0–3</td>
<td>14</td>
<td>1.80(1.55), 0–3</td>
<td>10</td>
<td>&lt;.001</td>
<td>2&gt;.01, 1=0</td>
<td>.34</td>
</tr>
</tbody>
</table>
Table 1. Descriptive statistics for all study variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Never n 1</th>
<th>Past n 1</th>
<th>Current n 1</th>
<th>p</th>
<th>Pairwise comparisons</th>
<th>Partial η²</th>
<th>Cramer’s Phi</th>
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<tbody>
<tr>
<td><strong>Actigraphy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total sleep time</td>
<td>387.3(61.41), 217.21–458.50</td>
<td>19</td>
<td>431.07(49.74), 339.33–510.05</td>
<td>14</td>
<td>395.10(123.71), 144.19–553.60</td>
<td>10</td>
<td>.133</td>
</tr>
<tr>
<td>Sleep Latency</td>
<td>12.76(1.44), 1.40–48.14</td>
<td>19</td>
<td>15.07(12.50), 1.27–41.07</td>
<td>14</td>
<td>21.21(16.26), 4.54–60</td>
<td>10</td>
<td>.130</td>
</tr>
<tr>
<td>Sleep Efficiency (%)</td>
<td>82.25(0.86), 53.84–92.47</td>
<td>19</td>
<td>84.83(5.56), 80–91.45</td>
<td>14</td>
<td>77.12(19.80), 24.36–89.35</td>
<td>10</td>
<td>.155</td>
</tr>
<tr>
<td>WASO (%)</td>
<td>53.60(26.81), 21.53–118.80</td>
<td>19</td>
<td>52.27(20.05), 11.15–95.13</td>
<td>14</td>
<td>44.74(14.84), 23.77–70</td>
<td>10</td>
<td>.294</td>
</tr>
<tr>
<td><strong>Polysonmography</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM Latency (%)</td>
<td>79.13(38.45), 36.50–173.50</td>
<td>19</td>
<td>156.50(98.91), 41.50–366</td>
<td>14</td>
<td>114.55(101.34), 41.50–366</td>
<td>10</td>
<td>.005</td>
</tr>
<tr>
<td>%REM</td>
<td>23.31(8.06), 3.30–40.70</td>
<td>19</td>
<td>23.71(6.43), 13–33.50</td>
<td>14</td>
<td>27.71(9.73), 14.50–43.90</td>
<td>10</td>
<td>.174</td>
</tr>
<tr>
<td>%NREM</td>
<td>76.81(8.09), 59.30–96.70</td>
<td>19</td>
<td>76.29(6.46), 66.50–85.50</td>
<td>14</td>
<td>72.30(9.74), 56–85.50</td>
<td>10</td>
<td>.192</td>
</tr>
<tr>
<td>%SWS</td>
<td>14.83(7.34), 1.50–36.50</td>
<td>19</td>
<td>11.56(7.83), 1.40–34.60</td>
<td>14</td>
<td>7.68(3.56), 0–17.70</td>
<td>10</td>
<td>.025</td>
</tr>
<tr>
<td>RDI (%)</td>
<td>8.30(9.01), 0.20–31</td>
<td>18</td>
<td>4.76(5.63), 0–16.5</td>
<td>14</td>
<td>15.76(28.39), 0.10–94</td>
<td>10</td>
<td>.371</td>
</tr>
<tr>
<td>ODI (%)</td>
<td>4.15(6.80), 0.20–22.02</td>
<td>17</td>
<td>3.99(4.46), 0.41–13.63</td>
<td>11</td>
<td>6.80(8.16), 0.15–23.69</td>
<td>10</td>
<td>.932</td>
</tr>
</tbody>
</table>

Note: 1 Sample size varies due to missing data; PHQ-9 = Patient Health Questionnaire; MINI = Mini International Neuropsychiatric Interview; AUCG = Area Under the Curve with reference to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen Desaturation Index below 3%; 2 Higher scores indicate increased depression severity; 3 Higher scores indicate increased Respiratory Disturbance; 4 Higher scores indicate greater oxygen desaturations; ESS = Epworth Sleepiness Scale; MEQ = Morningness Eveningness Questionnaire; 5 Higher scores indicate more sleepiness; 6 Higher scores indicate longer sleep latency; 7 Higher scores indicate better sleep efficiency; WASO = Wake After Sleep Onset (mins); REM = Rapid Eye Movement sleep; NREM = Non Rapid Eye Movement sleep; SWS = Slow Wave Sleep; 8 Higher scores indicate greater minutes spent awake after sleep onset; 9 Higher scores indicate longer REM latency; PSQI = Pittsburgh Sleep Quality Index; 10 Higher scores indicate poorer sleep quality; (2>0, 1=0,2) The Current group had higher scores than the Never and Past groups, who did not differ; (2=0, 1=0,2) The Current group had higher values than the Never group, the Past group did not differ from the remaining groups; (1<0, 0,1=2) The Past group had lower values than the Never group, the current group did not differ from the remaining groups; (0>2, 1=0,2) The Never group had higher values than the Current group, the Past group did not differ from the remaining groups; 11 Cole et al. (2006); 12 two-tailed. All tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.
Table 2. Correlation matrix for all study variables

<table>
<thead>
<tr>
<th></th>
<th>Cortisol AUCG</th>
<th>PSQI Global</th>
<th>PSQI Cole’s Sleep Efficiency</th>
<th>PSQI Cole’s Perceived Sleep Quality</th>
<th>Acti TST</th>
<th>Acti Sleep Latency</th>
<th>Acti Sleep Efficiency</th>
<th>Acti WASO</th>
<th>PSG REM latency</th>
<th>PSG %REM</th>
<th>PSG %NREM</th>
<th>PSG %SWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol AUCG</td>
<td>-</td>
<td>.27</td>
<td>.25</td>
<td>.22</td>
<td>.18</td>
<td>-.19</td>
<td>-.26</td>
<td>-.15</td>
<td>.33†</td>
<td>-.33†</td>
<td>-.06</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.22†</td>
<td>-.23</td>
<td>-.29†</td>
<td>-.20</td>
<td>-.07</td>
<td>-.35†</td>
<td>-.28†</td>
<td>-.20</td>
<td>.30†</td>
<td>-.27†</td>
<td>.28†</td>
<td>-.12</td>
</tr>
<tr>
<td>PSQI Use of Sleep</td>
<td>.39†</td>
<td>.72†</td>
<td>.42†</td>
<td>.68†</td>
<td>.03</td>
<td>.15</td>
<td>-.01</td>
<td>-.02</td>
<td>.16</td>
<td>.38‡</td>
<td>-.38‡</td>
<td>-.19</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHQ-9</td>
<td>.22†</td>
<td>.88‡</td>
<td>.72‡</td>
<td>.87‡</td>
<td>.68‡</td>
<td>-.02</td>
<td>.27</td>
<td>-.08</td>
<td>-.32†</td>
<td>-.07</td>
<td>.46‡</td>
<td>-.34‡</td>
</tr>
<tr>
<td>RDI</td>
<td>-.02†</td>
<td>-.08</td>
<td>-.20</td>
<td>-.05</td>
<td>.05</td>
<td>-.36†</td>
<td>.18</td>
<td>-.33†</td>
<td>.13</td>
<td>.25</td>
<td>-.14</td>
<td>.14</td>
</tr>
<tr>
<td>ODI-3</td>
<td>-.20†</td>
<td>-.21</td>
<td>-.37†</td>
<td>-.14</td>
<td>-.05</td>
<td>-.36†</td>
<td>.14</td>
<td>-.32†</td>
<td>.17</td>
<td>.25</td>
<td>-.31†</td>
<td>.30†</td>
</tr>
<tr>
<td>Time since diagnosis (years)</td>
<td>.38†</td>
<td>.18†</td>
<td>.22†</td>
<td>.15†</td>
<td>-.12†</td>
<td>-.32†</td>
<td>.06†</td>
<td>-.16†</td>
<td>.22†</td>
<td>-.36†</td>
<td>.14†</td>
<td>-.13†</td>
</tr>
</tbody>
</table>

Note: AUCG = Area Under the curve with respect to ground; PHQ-9 = Patient Health Questionnaire; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen desaturation Index, 3%; PSQI = Pittsburgh Sleep Quality Index; Acti = Actigraphy; TST = Total sleep time; WASO = Wake after sleep onset; PSG = Polysomnography; REM = Rapid eye movement sleep; NREM = Non Rapid eye movement sleep; SWS = Slow wave sleep; † p < .05, ‡ p < .01; † two-tailed: all tests were one-tailed, based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.
Correlations with depression severity
In order to examine whether sleep problems increased as a function of depression severity regardless of group, correlations were conducted between PHQ-9 scores and sleep variables across the whole sample. This had the benefit of maximising sample size and accounting for the possible impact of any subclinical depression. After controlling for age and ODI-3, more severe depression was associated with greater %REM ($r = .42, p = .009$) and lower %NREM ($r = -.42, p = .010$). In addition, more severe depression was correlated with lower %SWS (see Table 2). Depression severity was associated with less WASO (see Table 2), which was an unexpected result. On self-report, results were consistent with the group comparisons and revealed that greater depression severity was associated with poorer sleep quality on PSQI global score (see Figure 1), Cole’s Sleep Efficiency, Cole’s Perceived Sleep Quality and Cole’s Daily Disturbances (see Table 2). After controlling for age and ODI-3, the correlation for PHQ-9 and Cole’s Sleep Efficiency remained significant, $r(29) = .71, p < .001$.

![Figure 1. Scatterplot of the relationship between PHQ-9 and PSQI Global score. (All plots were of a similar shape, but in the opposite direction for %REM, %SWS and WASO).](image)

Analyses of cortisol
When the three subgroups were compared, there were no significant differences for morning cortisol measures (AUC$_G$ or means; see Table 1). However, further analyses revealed that when the Past and Never groups were collapsed, individuals with Current depression showed significantly elevated cortisol readings: $t(33) = -1.81, p = .040$, partial $\eta^2 = .09$. Across the whole sample, cortisol AUC$_G$ levels increased in a dose-dependent manner with PHQ-9 scores (see Table 2, Figure 2).
Whole sample correlations also revealed a relationship between higher cortisol levels (AUCG) and greater %REM and less %NREM (see Table 2). These relationships remained significant after controlling for age and ODI-3. After controlling for age, RDI and ODI-3, a negative association of cortisol (AUCG) and actigraphy TST was revealed ($r = -.35, p = .039$); and a negative association of cortisol (AUCG) and actigraphy Sleep Efficiency was likewise revealed ($r = -.39, p = .021$). However, cortisol was not linked to self-reported sleep quality.

Figure 3. Scatterplot of the relationship between cortisol AUCG and %NREM. (All remaining plots were of similar shape, but in the opposite direction for %REM).
Discussion

This study aimed to characterise the sleep of older adults with current MDD compared to those with a past history, and no history of depression, using PSG, actigraphy recording, and questionnaires. In summary, results indicate that individuals with current symptoms of depression (Current group) had the lowest percentage of SWS sleep and poorer self-reported sleep quality. Depression symptom severity was also associated with greater sleep architecture abnormalities (more %REM, less %NREM and %SWS) and poorer self-reported sleep quality across the whole sample. Finally, increased cortisol levels were significantly associated with greater sleep abnormalities, including greater %REM, less %NREM, and shorter total sleep time and sleep efficiency. The results are discussed in turn.

Group comparisons

The group comparisons revealed that the currently depressed individuals exhibited the most sleep abnormalities, including lower %SWS and greater self-reported sleep problems. These findings are consistent with previous studies reporting reduced SWS in middle-aged and older adults with depression (e.g., Armitage, 2007), as well as increased subjective reports of poor sleep (e.g., Lieverse, 2014).

An examination of the profile of those individuals classed as having a past history of depression (‘Past’) revealed a puzzling finding – that of longer REM latency compared to the Never group. This contrasts with studies showing shorter REM latency in depression (e.g., Lee et al., 1993). One possibility for this unexpected result is that a significant proportion of the Past group was taking antidepressants. In support, studies show that the use of antidepressants is linked to increased REM latency (Wilson & Argyropoulos, 2005), and group comparisons in this sample using individuals taking antidepressants versus those not taking antidepressants (results not presented) confirm this suggestion (p < .001). Altogether, the Past group had longer REM latency relative to the Never group, which was linked to the use of antidepressants.

On the basis of these analyses, our findings show that sleep abnormalities were a particular feature of those with current MDD. Sleep stages and self-reported sleep

7 Of interest, two participants in the Never group were also taking antidepressant medication. This is not uncommon in community samples. The reasons these individuals gave for taking medication included low levels of anxiety and hormonal issues.
quality were the most sensitive to depression status, and sleep-wake patterns were the least sensitive. Group comparisons made on the basis of diagnosis, however, do not take into consideration the continuous nature of depressive symptoms, so our next step was to examine the performance of patients based on severity of symptoms.

**Symptom severity correlations across the whole sample**

Correlation analyses of sleep with depressive symptom severity (PHQ-9) revealed the expected pattern of findings for sleep architecture. Specifically, depression has been linked to sleep architecture abnormalities concerning REM and SWS sleep (Reynolds III et al., 1985). In the current study, higher depression levels across the whole sample were linked to significantly more %REM, and less %NREM and %SWS, as well as greater subjective reports of poor sleep. These findings underscore the findings in middle-aged and older adults (e.g., Maglione et al., 2012; Paudel et al., 2008; Reynolds III et al., 1985), and suggest a dose-dependent relationship, whereby more severe depression is linked to greater sleep problems. One reason why %REM and %NREM did not show group differences, but showed an association with symptom severity might be related to our sample size. Group comparisons contained relatively small groups of participants – due to the demanding nature of PSG studies – whereas whole sample correlation analyses comprised a larger sample size and, therefore, had greater statistical power.

With regard to actigraphy, our correlational analyses revealed an association between greater depression severity and less WASO. This finding is counterintuitive as we would expect poorer sleep quality (i.e., greater WASO) in depression. We explored the possibility that WASO might be linked to demographic or clinical characteristics (e.g., antidepressant use), but our analyses did not yield any significant associations. Other actigraphy studies in older adults with depression reported greater WASO (Naismith et al., 2011), so this contradictory finding remains unexplained.

**Cortisol readings**

Elevated morning cortisol was linked to more %REM, and less %NREM in the whole sample. These results are largely consistent with those demonstrated above using correlational analyses of symptom severity – both increased cortisol and depression severity were associated with more %REM and less %NREM. On actigraphy measures,
Overall, the current findings stand in contrast to studies that report enduring sleep problems and elevated cortisol during remission from depression. One reason for this discrepancy could be due to differences in the characteristics of the Past group. However, the most likely possibility is that definitions of ‘remission’ are not uniform across studies. For example, (Lee et al., 1993) tested participants after an average of 38 weeks since the last episode – whereas our study was reliant simply on a ‘previous’ diagnosis without exclusion criteria regarding the date of previous diagnosis. The possibility exists, therefore, that our participants had been in remission for too long. Although we collected information regarding the time of the first diagnosis of depression, the time since last depressive episode was not known. However, it is noteworthy that a significant percentage of the Past group was taking antidepressants, which argues against the idea that the Past group was in complete remission. Therefore, future research must clearly delineate the period of remission.

Another interesting observation was that depression status and severity (assessed with diagnoses, interviews, and self-reports) were largely only associated with sleep architecture indices and self-reports. By contrast, cortisol levels were associated with actigraphy monitoring and sleep staging. This suggests that different methods of assessing depression yield different profiles of associations with sleep assessment methods.

**Limitations**

Given that we do not know how much time had passed since the last depressive episode in the Past group, nor information about the use of different medications, we must be cautious in drawing firm conclusions about the Past group. We were also limited by power, as our sample was small. For example, an a priori sample size calculation revealed that an overall sample of 114 would be needed to detect a significant effect on key actigraphy sleep measures such as sleep latency (effect size = 0.26 at .05 significance and .80 power, one-tailed), and an overall sample size of 130 would be needed to detect an effect of sleep efficiency (effect size = 0.25 at .05 significance and .80 power, one-tailed; Faul, Erdfelder, Lang, & Buchner, 2007). However, because PSG is costly, onerous and time consuming, PSG studies are commonly limited by issues of
power, and our sample size is not greatly different from other studies in older adults. Despite being limited by power, it is worth noting the substantial magnitudes of the effect sizes (partial $\eta^2$ and $r$; see Tables 1 and 2), which range from medium to large (Cohen, 1988).

In summary, results indicate that current MDD was associated with a range of sleep abnormalities. This finding was confirmed across three different metrics of depression: group comparisons based on depression diagnosis; depression symptom severity; and cortisol measures. Our examination of different methods of sleep assessment is a notable strength of this study. This enabled a comprehensive evaluation of sleep, recognising it as a multifaceted process, and yielded valuable information about assessing sleep abnormalities in healthy and depressed populations. Results revealed that the association of depression and sleep differed depending on the method of sleep assessment. Firstly, depression was not reliably associated with sleep-wake pattern disturbance as assessed with actigraphy. Secondly, self-reported sleep measures, while associated with current depression, did not detect evidence of sleep disturbance in individuals with a past history of depression, nor were they correlated with depression severity across the whole sample. Finally, sleep architecture abnormalities were associated with current depression and depressive symptoms across the entire sample. Due to limitations of the current study (as discussed above), we must avoid making firm conclusions regarding the influence of a past history of depression on sleep. However, in view of these findings, clinicians may wish to consider screening for sleep architecture abnormalities (using PSG) as potential vulnerability markers of depression. This is of clinical importance given that persistent sleep abnormalities during remission from depression could represent vulnerability to experiencing further clinical episodes. Identification of sleep problems during remission allows for the possibility of early intervention. In fact, research has shown that treating sleep problems can reduce the risk of future clinical depression (Sbarra & Allen, 2009). For example, therapies aimed at treating SDB, such as Continuous Positive Airways Pressure (CPAP) have been shown to improve depressive symptoms (e.g., Schwartz & Karatinos, 2007), and Cognitive Behavioural Therapy (CBT) for insomnia has been associated with a higher rate of remission from depression (Manber et al., 2008).
Foreword to Chapter 5

Results from Chapter 3 suggest an association between self-reported sleep and cognition in community adults across the lifespan, and indicate that depression was a significant predictor of cognitive complaints. Limitations of Chapter 3 included the use of subjective measures of sleep and cognition, and subclinical levels of depression. Therefore, Chapter 5 presents an investigation of this association using objective measures of sleep (actigraphy, home-PSG) and cognition in a sample of older adults with and without MDD. Based on the results from the last chapter that revealed that sleep abnormalities were present only in those older adults with current depressive symptoms, comparisons in the current chapter comprise participants with a current diagnosis versus no current diagnosis of MDD (Past + Never). This chapter addresses two objectives of this thesis: i) to determine whether sleep problems are linked to cognitive deficits in ageing; and ii) to determine whether depression alters the relationship between sleep and cognitive performance in older adults.
Chapter 5
Sleep and cognition in older adults: does depression matter?

*An actigraphy and polysomnography study*

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**Author Contributions:**

A. Mellor, F. Waters and R. S. Bucks developed the study concept, hypotheses, and design. A. Mellor analysed and interpreted the data under the supervision of R. S. Bucks and F. Waters. Kathy Sanders provided access to her laboratory and offered training in cortisol collection. She also analysed all samples. Jennifer Maul provided training in polysomnography and analysed all sleep studies. H. McGowan provided access to the clinical sample, and feedback on the manuscript. All authors approved the final version of the manuscript.
Abstract

The impact of sleep on cognition is well recognised, although research in older adults is lacking, as are investigations into how depression might impact on this relationship. This study assessed the relationship between sleep (assessed with actigraphy and polysomnography) and cognitive performance in older adults (50–78 years) with \( n = 10 \), and without \( n = 33 \) a current diagnosis of Major Depressive Disorder (MDD). There were associations between sleep-wake patterns and cognition across the whole sample: time spent awake after sleep onset (WASO) was associated with speed of responding on vigilance tasks, and accuracy on delayed recall tasks; sleep efficiency and total sleep time (at trend-level) were linked to working memory accuracy.

Examination of the impact of depression on the relationship between sleep and cognition was explored using moderation analysis. Current depression was a significant moderator of the relationship between sleep-wake patterns (assessed with actigraphy), but not sleep architecture (assessed with PSG), and cognition. That is, there was a different pattern of association of sleep-wake patterns and cognitive performance depending on depression status. Specifically, in the MDD group, sleep-wake patterns were linked to speed of processing on cognitive tasks, whereas in the not currently depressed group, sleep-wake patterns were linked to performance accuracy. In conclusion, depression impacted on the relationship between sleep and cognition, but questions remain regarding the nature of the relationship between sleep and cognition in older adults. Given that sleep problems are potentially modifiable risk factors for cognitive impairment, these findings point to the importance of assessing sleep in both depressed and healthy older adults.
The link between sleep and cognition is now well recognised. However, fewer studies have been conducted in older adults (without sleep disorders) relative to younger adults. Understanding the importance of sleep in cognition in older adults is challenging because on average, ageing is linked to changes both in cognition – including a decline in attention, memory and executive functioning (Rabbitt & Lowe, 2000) – and in sleep (D’Ambrosio & Redline, 2014)⁸. Changes in the latter include sleep-wake cycle changes, such as increased sleep fragmentation, and sleep architecture changes, such as decreases in Slow Wave (SWS; Ohayon et al., 2004) and Rapid Eye Movement (REM) sleep (Stanley, 2005). This has led to questions regarding the nature of the relationship between sleep and cognition in the context of ageing.

One theory suggests that age-related cognitive deficits could be, at least in part, due to poor sleep (e.g., Altena et al., 2010). Evidence mostly derives from subjective (questionnaire) studies, and from studies using actigraphy to assess sleep-wake patterns, although there is a paucity of evidence using polysomnography (PSG). Using questionnaires, sleep disturbances in older adults have been linked to poorer cognitive performance on tasks of executive functioning (e.g., Nebes et al., 2009), memory (e.g., Schmutte et al., 2007), and attention (e.g., Amer et al., 2013). Actigraphy is a noninvasive method of sleep assessment, which involves wearing an ‘actiwatch’ that permits 24-hour recording of activity while the individual goes about their normal routine. It has been recommended by the American Academy of Sleep Medicine as an accurate assessment of sleep-wake patterns (Morgenthaler et al., 2007). Actigraphy studies in older adults are largely consistent in showing a link between sleep-wake patterns and executive functioning (Blackwell et al., 2006; Haimov et al., 2008), working memory (Miyata et al., 2013) and sustained attention (Haimov et al., 2008). Sleep duration (< 5 hours) has also been linked to simple attention and short-term memory (e.g., Miyata et al., 2013), although this is not the case in all studies (e.g., Blackwell et al., 2011; Blackwell et al., 2006), which might suggest that sleep quality, rather than quantity, is more important for cognition.

Studies using PSG provide evidence of a link between specific stages of sleep to cognition in older adults, but the evidence is largely contradictory. For example, some studies report a positive association between SWS and memory and executive function.

⁸ It is important to refer to age-related declines in sleep and cognition as ‘on average’, given that there is substantial inter-individual variability in age-related changes in functioning and that marked decline is not inevitable.
functioning performance (e.g., Anderson & Horne, 2003; Backhaus et al., 2007; Mander et al., 2013; Van der Werf et al., 2009; Van der Werf et al., 2011), although some studies report no such association (e.g., Scullin, 2013).

The reasons for the negative findings are not entirely clear. One view regarding how sleep relates to cognition is that of a neurodevelopmental account. This view observes that qualitative and quantitative aspects of sleep change substantially during development and that these changes parallel the learning and cognitive needs across the lifespan (Geiger et al., 2010). The assumption, therefore, is that of a close association between sleep stages (REM, SWS) and cognition and brain morphology. Evidence derives from early-life studies, where sleep duration and REM sleep is significantly increased in childhood, perhaps linked to children’s significant learning needs (e.g., Mirmiran & Van Someren, 1993). Studies in younger adults are also supportive of a continued relationship of sleep stages and cognition (e.g., Backhaus et al., 2007). However, studies in older adults are less consistent. We know that there are significant changes in sleep architecture associated with ageing, including reductions in SWS and REM sleep (Stanley, 2005), which perhaps reflect a decreased need for learning in older adults compared to children. But nevertheless, according to the neurodevelopmental account, the tight relationship between sleep architecture and cognition should remain if human cognitive needs are so entirely dependent on sleep physiology. One prediction, based on this view, therefore, is that age-related decreases in SWS and REM are associated with corresponding decreases in cognitive performance. A suggested mechanism of action is that decreases in these sleep stages reduce Sleep Dependent Memory Consolidation (SDMC) and/or compromise the integrity of the hippocampus or prefrontal cortex (PFC; Scullin, 2013).

There is indeed some support for this view. As mentioned above, some studies indicate a role of SWS in episodic memory and executive functioning in older adults. For example, Van der Werf et al. (2009) showed that experimental reduction of SWS affected activity in the hippocampus and associated declarative memory encoding the next day. While the current study investigated the impact of sleep on cognitive performance within one testing session (‘waking cognitive performance’), evidence also derives from overnight consolidation paradigms. Backhaus et al. (2007), for example, revealed that although SWS decreased with age, greater SWS was none-the-less significantly associated with better overnight episodic memory consolidation in the
older adult group. SWS has also been linked to executive functioning. Lafortune et al. (2014) implicated SWS in ‘waking’ performance on a verbal fluency task. Furthermore, another study linked experimental SWS reduction to more lapses on vigilance tasks in healthy older adults the next day (Van der Werf et al., 2011).

Rapid Eye Movement (REM) sleep has also been implicated in cognition, usually in the context of procedural or emotional memory (Maquet, 2001). Two recent studies, however, point to a possible role of REM sleep in non-emotional declarative memory in older adults. For example, a consolidation study implicated REM sleep in overnight improvement on a word recall task (Schredl et al., 2001), and another study linked longer REM sleep to better ‘waking cognitive performance’ on a verbal learning task (Lafortune et al., 2014).

A contrasting view suggests that the nature of the relationship between sleep and cognition may change with age. For example, the benefit of sleep on cognition has been shown to be reduced in older adults (e.g., Spencer et al., 2007), and older adults appear more resilient to sleep deprivation than younger adults (e.g., Stenuit & Kerkhofs, 2005). Some studies also suggest that sleep stages important in younger adults may no longer be important in older adults (e.g., Hornung, Regen, Danker-Hopfe, Schredl, & Heuser, 2007). Taken together, these findings suggest that sleep may no longer be closely related to cognition in ageing, which Spiegel et al. (1986) referred to as ‘functional-dissociation’. The mechanisms behind the weakening sleep-cognition relationship seen in ageing are not yet understood. Scullin (2013) describes an interesting study in younger adults, which found that exposure during SWS to an odour, which had been presented as context during prior learning, caused better retention of the associated memory (Rasch, Büchel, Gais, & Born, 2007). Scullin suggests that method might be useful in encouraging older adults to consolidate information during sleep, and goes on to suggest that if this intervention were successful in older adults, it would imply that the neural circuits and the ability to consolidate information remain intact in ageing. On the other hand, if the manipulation did not result in improved consolidation, this might indicate that the ability to consolidate memories in ageing has diminished, which could be the result of age-related changes in brain structure and function (e.g., Grady, 2006), neurochemical changes (e.g., Bäckman, Nyberg, Lindenberger, Li, & Farde, 2006), or other unknown age-related mechanisms. Hence, there is a need for more research into why some studies have found that the sleep–cognition association weakens in ageing.
Evidence cited in support of the ‘functional-dissociation’ account includes studies showing a different pattern of cognitive performance in older adults relative to younger adults. For example, Scullin (2013) found that sleep benefited word-pair learning in a group of younger adults, which was correlated with greater SWS. In contrast, the opposite pattern was found in the older adults, in whom greater SWS was linked to poorer memory. Hence, results indicate that the relationship between episodic memory and SWS changed with age. Furthermore, in contrast to research in younger adults indicating a close association between SWS and vigilance (Jurado et al., 1989), research in healthy older adults suggested an erosion of this relationship (Crenshaw & Edinger, 1999).

Tentative evidence for the ‘functional dissociation’ account also derives from studies showing no link of REM sleep and cognition in older adults. For example, a recent ‘waking’ study found that REM sleep was not related to executive functioning performance in older adults (Lafortune et al., 2014), which stands in contrast to consolidation studies in younger adults that have implicated REM in improvement on problem-solving tasks after a period of sleep (e.g., Cai et al., 2009; Walker et al., 2002). Thus, this may suggest a changing relationship of REM sleep and cognition in ageing.

In summary, while some studies, then, support the neurodevelopment approach suggesting a continued relationship of sleep architecture and cognition in older adults, others suggest that this relationship changes and even disappears with age (the ‘functional dissociation’ account).

The impact of depression on sleep and cognition

One factor that may influence the relationship between sleep and cognition differentially with age is depression. Surprisingly few studies in older adults have considered the role of depression in the association between sleep and cognition, despite the fact that depression is common in older adults (Garcia, 2008); poor sleep and depression are closely related (Sbarra & Allen, 2009); and that sleep problems and depression are independently associated with cognitive impairment (Koehler et al., 2010; Naismith et al., 2009; Riemann et al., 2001; Smagula et al., 2013; Snyder, 2013).

Existing studies in older adults, however, have either assessed for subclinical levels of depression (e.g., Blackwell et al., 2011; Blackwell et al., 2006; Nebes et al., 2009;
Schmutte et al., 2007), and/or have excluded older adults with clinical levels of depression (e.g., Bastien et al., 2003; Nebes et al., 2009; Sutter et al., 2012; Van der Werf et al., 2011; Vignola et al., 2000). A cross-sectional study using questionnaires in 107 older adults (61+ years) with varying levels of subclinical depressive symptoms on the Geriatric Depression Scale (GDS; Yesavage et al., 1983) found that poorer global sleep quality on the PSQI was linked to decreased performance on tasks of executive functioning (reasoning, semantic fluency and shifting), but only in individuals with higher levels of depressive symptoms (Sutter et al., 2012). This intriguing finding suggests that the profile of sleep-associated cognitive performance may be different depending on levels of depression, although results have yet to be replicated using objective measures.

To our knowledge, only one study has assessed sleep and cognition using objective measures of sleep in older adults with depression. Naismith et al. (2011) assessed sleep-wake patterns using actigraphy in older adults with a lifetime history of clinical depression (n = 44) compared to healthy controls (n = 22). In the depressed individuals (currently depressed and individuals in remission), greater WASO was linked to poorer memory (verbal learning on the Rey Auditory Verbal Learning Test; RAVLT; Spreen & Strauss, 1998) and poorer executive functioning (response inhibition on the Stroop test, Delis Kaplan Executive Functioning System; DKEFS; Delis, Kaplan, & Kramer, 2001); and poorer sleep efficiency was linked to poorer memory (verbal learning on the RAVLT, and visual learning on the Ray Complex Figure Test; RCFT; Spreen & Strauss, 1998) and poorer executive functioning (response inhibition on the Stroop test, and problem-solving on the Sorting test; DKEFS).

This suggests that sleep problems are related to cognitive impairment in depression. Although the causal mechanisms remain to be understood, depression and poor sleep have been found to impact on overlapping brain areas (e.g., frontal lobes) and their associated cognitive functions (Naismith et al., 2011). While this is an important study, Naismith et al. (2011) included low levels of depression; did not separate current from past depression; and did not compare the cognitive performance of the depressed group with the control group. Furthermore, the study did not specifically examine the potential moderating effect of depression on the relationship between sleep and cognition. In other words, the authors did not examine whether the association of sleep and cognition differed as a function of depression, an important aim of the current study.
This study conducted a comprehensive investigation into the association of sleep-wake patterns and sleep architecture and daytime cognitive performance in older adults. We were particularly interested in whether this pattern of association differed between currently depressed (MDD) versus not currently depressed older adults. Given that the effect of sleep on arousal levels and attention has been suggested as a mechanism of sleep-related deficits in higher order cognitive processes (Doran et al., 2001), we controlled for vigilance in all analyses of memory and executive functioning. We also controlled for sleep-disordered breathing (SDB; Bucks et al., 2013; Malhotra & White, 2002) and the associated effects of hypoxia (Beebe & Gozal, 2002), gender (Voderholzer et al., 2003; Weiss, Kemmler, Deisenhammer, Fleischhacker, & Delazer, 2003), age (Ancoli-Israel, 2005; Bruce & Aloia, 2006), and ‘morningness-eveningness’ preference (Ramirez et al., 2006), given that these variables can affect sleep and/or cognition.

**Method**

**Participants**

Forty-six participants took part in this study. After excluding three people due to a history of traumatic brain injury or bipolar depression, data were analysed for 43 participants (12 male, 31 female) aged 50–78 years.

Recruitment of the not currently depressed individuals \((n = 33)\) was conducted via advertisements in community centres and mail outs to volunteer groups. Participants with a present diagnosis of MDD \((n = 10)\) were either outpatients from the North Metropolitan Adult Mental Health Older Adults, Osborne Park Hospital, Perth, Western Australia, or individuals recruited via advertising from the community.

Inclusion criteria for the depressed group included age (50–80 years) and a current clinical diagnosis of unipolar depression provided by a mental health professional. Exclusion criteria for all participants included: chronic infectious illness; neurological or neurodegenerative conditions; history of moderate or severe traumatic brain injury; previous loss of consciousness > 30 minutes duration; treatment for substance abuse; or any other psychiatric disorder. Ethics committee approval was granted by both the North Metropolitan Mental Health Service and the University of Western Australia Human Research Ethics Committees.
All individuals with current MDD had received a clinical diagnosis by a mental health clinician, and had their diagnosis confirmed with a research interview with the MINI (Sheehan et al., 1998) and PHQ-9 score of ≥10 (Kroenke et al., 2001). Morning cortisol levels were also collected as a potential biological index of depression. While elevated morning cortisol cannot be considered a ‘pure’ measure of depression as it is linked to other factors such as alcohol consumption (Badrick et al., 2008), it is associated with hypothalamic pituitary adrenal axis activity, which is regarded as important in the pathophysiology of depression (Riemann et al., 2001).

Procedure
All assessments were conducted in the participant’s home. The first visit included completion of consent forms and questionnaires; clinical and sleep interviews; and the first overnight sleep study (PSG). Participants were also given an actiwatch and sleep diary. One week later, cognitive assessments were administered and the second night of PSG was conducted. The sleep studies were separated by one week in order to reduce first night effects due to unfamiliarity with equipment (Agnew et al., 1966). Morning cortisol samples were collected on the morning of the last visit.

Sleep assessments

Sleep electrophysiology: Home-polysomnography (PSG) was conducted using the Compumedics ‘Somté’ device. It includes electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), airflow, body position, thoracic and respiratory belts, and blood oxygen saturation. Data were analysed by a Senior Sleep Technologist (J Maul) and checked by a second Sleep Technologist (A Mellor). Only data from the second sleep study are reported. Outcome variables included the percentage of REM and SWS sleep given the implication of these major sleep stages in cognition. In order to assess for sleep-disordered breathing (SDB), analyses included the Respiratory Disturbance Index (RDI), and the Oxygen Desaturation Index (ODI-3; no. times per hour that there is a decrease of ≥ 3% in blood oxygen saturation). This was to ensure that any effects of sleep on cognition were not due to SDB-associated hypoxemia.

Actigraphy: Participants wore an actiwatch, a wrist-worn device that non-invasively monitors sleep-wake cycles, for two weeks to ensure seven nights of viable data were
collected. Output measures were total sleep time (minutes), sleep efficiency (time spent asleep as a proportion of time spent in bed, expressed as a percentage), sleep latency (minutes), and time spent awake after sleep onset (WASO; minutes). Participants kept a sleep diary to enable interpretation of actigraphy data. Actigraphic data were checked against the sleep diary by two researchers to ensure concordance (A Mellor and F Waters). The Actiwatch Spectrum (MiniMitter Philips) was used in this study. Seven days of actigraphy recording has been shown to be sufficient to obtain stable measures of sleep-wake patterns (Knutson et al., 2007; Tworoger et al., 2005).

Participants completed a daily sleep diary to record details such as sleep and wake times, whether the day was a work day, if they took any naps, and if the watch were removed for any period. This information was used to cross-validate and edit the actigraphy data as needed. Each actigram was visually inspected and compared to the sleep diary in order to identify any major discrepancies. Time in bed was adjusted by the scorer in cases where there was a discrepancy of greater than one hour between the diary and actigraph, so it was consistent with the sleep diary.

Salivary cortisol assessment: Three samples were taken: immediately after waking, wake+15 minutes, and wake+30 minutes. Participants were asked to collect their saliva in small vials and store in the freezer until collection. For each sample, salivary cortisol was assessed in duplicates using a commercial enzyme immunoassay (Salimetrics, LCC). All samples from the same participant were assessed in the same batch. The inter-assay coefficient of variation was 1.63% and 9.61% for high and low quality control standards respectively, indicating consistency between runs.

Materials
A demographics questionnaire asked about age, occupation, medical history (including respiratory problems, chronic pain, sleep disorders), and medication use.

The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) is a 9-item questionnaire, which assesses levels of depression in the past 2 weeks on a scale of 0 (not at all), to 3 (nearly every day). The PHQ-9 has excellent reliability ($\alpha = .94$ in the current study). The PHQ-9 has diagnostic value as items map onto the DSM-IV criteria for diagnosis of depression (Kroenke et al., 2001). Scores range from 0–24, with higher scores indicating more severe levels of depression. A score of $\geq 10$ (for moderate
depression) is commonly used to identify clinical levels of depression (Kroenke et al., 2001).

The Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) is a short, diagnostic interview for psychiatric evaluation. This was used by the investigator (A Mellor) to exclude co-morbid psychiatric disorders and to confirm diagnosis of depression.

The Morningness-Eveningness Questionnaire (MEQ; Horne & Ostberg, 1976) assesses individual differences in the time of day a person prefers to be active. It consists of 19 questions. Scores from each item are summed and produce a total score that ranges from 16–89. Scores of ≥ 59 are classified as ‘morning-types’, ≤ 41 as ‘evening-types, and scores between 42–58 as ‘intermediate-types’. Studies using the MEQ report good internal consistency (Adan & Natale, 2002; α = .82 in this study).

Cognitive Assessments:
The National Adult Reading Test-Revised (NART-R; Nelson & Willison, 1991) is a valid index of premorbid intellectual ability that measures the ability to read aloud a list of 50 irregular English nouns. Performance on the NART correlates highly with measures of general intelligence (Crawford, Stewart, Cochrane, Parker, & Besson, 1989). We estimated IQ for each participant using a regression equation (Nelson & Willison, 1991).

The CogState is a computerised test battery used to assess cognitive functioning (http://www.cogstate.com.au) and has been described in detail elsewhere (Maruff et al., 2009). It has well-established validity (e.g., Weaver Cargin, Collie, Masters, & Maruff, 2008), is suitable in older populations (Collie et al., 2003) and is sensitive to subtle cognitive impairment (Falleti et al., 2003). It assesses psychomotor speed, long-term memory (immediate and delayed), working memory and other components of executive functioning. The CogState battery was administered using a laptop computer. Tasks were selected in order to measure a range of cognitive functions. The primary outcome measures recommended by the CogState manual were used for analyses. Additional variables were also analysed to derive measures of accuracy and speed of responding measures for each task. In this study, participants completed the following tasks:
Detection Task – A measure of simple reaction time, speed of processing and psychomotor function (Cockayne et al., 2011; Davidson et al., 2011; Hammers et al., 2012; Lim, Ellis, Pietrzak, Ames, Darby, Harrington, Martins, Masters, Rowe, Savage, Szoeeke, Villemagne, Maruff, et al., 2012; Maruff et al., 2009; Olver, Ignatiadis, Maruff, Burrows, & Norman, 2008; Pietrzak, Snyder, & Maruff, 2010). Participants are instructed to respond as quickly as possible when a playing card presented on screen turns face up (Primary outcome measure = speed of responding: mean of the log10 transformed reaction time in ms for correct responses; log10, ms. Additional outcome variable = accuracy).

Identification Task – A measure of choice reaction time, attention and vigilance (Davidson et al., 2011; Hammers et al., 2012; Lim, Ellis, Pietrzak, Ames, Darby, Harrington, Martins, Masters, Rowe, Savage, Szoeeke, Villemagne, & Maruff, 2012; Maruff et al., 2009; Olver et al., 2008). Participants must decide whether the playing card is red or black. (Primary outcome measure = speed of responding; log10, ms. Additional outcome variable = accuracy).

One Back Memory Task – Widely agreed as a working memory task (Biggs et al., 2011; Davidson et al., 2011; Lim, Ellis, Pietrzak, Ames, Darby, Harrington, Martins, Masters, Rowe, Savage, Szoeeke, Villemagne, & Maruff, 2012; Maruff et al., 2009; Yoshida et al., 2011). Participants are required to identify whether the current card was the same as the previously presented card (Primary outcome variable = accuracy. Additional outcome variable = speed of responding; log10, ms).

Groton Maze Learning Task – This task requires participants to navigate their way through a 10x10 grid of tiles on a computer screen using the mouse to find a hidden pathway using trial and error-based feedback. While this task taps a diverse range of functions including spatial learning and error monitoring (Pietrzak et al., 2008), rule use and spatial memory (Pietrzak et al., 2010), which rely on attention, processing speed and decision-making (Darby & Walsh, 2005), it is widely used as a measure of spatial problem-solving and therefore, executive functioning (Cockayne et al., 2011; Collie, Maruff, Snyder, Darekar, & Huggins, 2006; Davidson et al., 2011; Olver et al., 2008; Yoshida et al., 2011).
There are a total of five trials. (Primary outcome measure = total number of errors. Additional outcome variable = speed of responding; moves per second). There is also a delayed recall version of this task (recall is tested approximately 20 minutes later, during which participants complete the remaining cognitive tasks).

*International Shopping List* – A verbal learning task (Davidson et al., 2011; Thompson et al., 2011; Yoshida et al., 2011). Participants listen to a list of items (n = 12) on a shopping list and are asked to recall them aloud. There are three learning rounds. (Primary outcome measure = total number of correct responses across three consecutive trials. No speed of responding measure). There is also a delayed recall version of this task (recall is tested approximately 30 minutes later, during which participants complete the remaining cognitive tasks).

*Continuous Paired Learning* – This task assesses paired-associate learning, a form of visual learning and memory (Davidson et al., 2011). In the learning phase, participants learn the location of objects on screen. In the test phase, they must find the correct location of the object across five rounds. (Primary outcome measure = total number of errors. Additional outcome measure = speed of responding; log₁₀, ms).

*The Hayling Sentence Completion Test* (HSCT; Burgess & Shallice, 1997) – This is a widely used measure of executive functioning, comprising two sets of 15 sentences, each missing the last word. In section 1, participants are asked to complete the sentence with a logical word as fast as possible. In section 2, they are asked to complete the sentence with an incongruent word as fast as possible. There are three possible outcome measures: the sum of the response latencies in section 1, the number of intrusions (errors) in section 2, and the time taken to respond in section 2. In this study, we analysed the total number of intrusion errors in section 2 (Burgess & Shallice, 1997).

**Statistical Analysis**

Salivary Cortisol: The Area Under the Curve with respect to ground (AUC₇₀) was calculated from the three morning cortisol saliva samples as an estimate of the total cortisol secretion over the first half an hour after awakening (y = 0; cortisol levels at time 0). This method is often used in cortisol research where there are repeated measurements over time (e.g., Fekedulegn et al., 2007; Vreeburg et al., 2010).
Cognitive variables: to reduce the number of variables in the analyses, cognitive variables were clustered into cognitive domains. We separated working memory tasks from other tasks of executive functioning based on the division of tasks in the CogState manual (also see Baddeley, 1992). Separate speed and accuracy composites were calculated by standardising the scores for each relevant task and averaging them. See Table 1 for a complete list of composites and their components. For composites that included tasks with primary outcome measures in different directions (e.g., total number of errors vs total number of correct responses), z-scores were reflected prior to averaging, such that higher scores indicate better performance for accuracy measures, and longer response times for speed measures. These were then re-reflected to make all scores positive for ease of interpretation of the moderation graphs.

Table 1. Cognitive Composites derived from CogState tasks and paper and pencil measures

<table>
<thead>
<tr>
<th>Composites</th>
<th>Tasks</th>
<th>Primary Outcome Measure</th>
<th>Additional Outcome Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vigilance</td>
<td>Detection Task</td>
<td>Speed of responding (lmin: log10 ms)</td>
<td>Accuracy</td>
</tr>
<tr>
<td></td>
<td>Identification Task</td>
<td>Speed of responding (lmin: log10 ms)</td>
<td>Accuracy</td>
</tr>
<tr>
<td>Long-term Memory</td>
<td>Continuous Paired Learning Task</td>
<td>Total no. errors across 5 rounds</td>
<td>Speed of responding (lmin: log10 ms)</td>
</tr>
<tr>
<td></td>
<td>International Shopping List (Immediate recall)</td>
<td>No. correct responses across the 3 rounds</td>
<td>N/A</td>
</tr>
<tr>
<td>Delayed-Recall</td>
<td>Groton Maze Learning Test (Immediate recall)</td>
<td>Total no. errors</td>
<td>Moves per second</td>
</tr>
<tr>
<td></td>
<td>International Shopping List (Delayed recall; delay of approx. 30 minutes)</td>
<td>No. correct responses across the 3 rounds</td>
<td>N/A</td>
</tr>
<tr>
<td>Executive Functioning</td>
<td>Groton Maze Learning Test (Immediate recall)</td>
<td>Total no. errors</td>
<td>Moves per second</td>
</tr>
<tr>
<td></td>
<td>Hayling Sentence Completion Test</td>
<td>Total no. of intrusion errors</td>
<td></td>
</tr>
<tr>
<td>Working Memory</td>
<td>One Back Card Task</td>
<td>Accuracy</td>
<td>Speed of responding (lmin: log10 ms)</td>
</tr>
</tbody>
</table>

Note: The immediate recall version of the International Shopping List is a measure of long-term memory. Any measure which requires the recall of information after >2 minutes is, by definition, a measure of long-term memory (Cardwell & Flanagan, 2005).

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9 This component is referred to as ‘attention-psychomotor functioning’ in the original CogState manual.
Data were analysed using SPSS Version 20 (IBM, Inc) and were screened for normality according to the criteria: skewness < 2, kurtosis < 7 (Curran, West, & Finch, 1996). Prior to analysis, data were transformed using ‘Log_{10} transformations’ where appropriate (Field, 2009). Cronbach’s alpha was used as a measure of internal consistency. Estimates of effect sizes were partial $\eta^2$ for continuous data or Cramer’s Phi for categorical data (Tables 2 and 3).

There were two stages of analysis. Firstly, to investigate the association of sleep and cognition in the whole sample, standard regression analyses were conducted entering relevant covariates (demographic variables that were significantly associated with the dependent variable) in the first step and then predictors (sleep) in the second step. In all analyses of memory and executive functioning, vigilance (also examined as an independent variable) was added as a covariate (at Step 1). Sleep variables assessed with actigraphy included: total sleep time (TST), sleep latency, sleep efficiency, and time spent awake after sleep onset (WASO). Sleep variables assessed with PSG included: %REM and %SWS. All correlations were one-tailed based on theory, except those where no prediction of the direction of the expected effect could be made, in which case two-tailed tests were reported. Secondly, a series of moderation analyses was conducted to investigate the effect of depression (dummy-coded for currently depressed versus not currently depressed) on the relationship between sleep and cognition using Hayes’ moderation method (Hayes, Glynn, & Huge, 2012). Bootstrapping was used to calculate 95% bias-corrected confidence intervals (CI) using 5000 bootstrapped samples (Hayes et al., 2012). All analyses were corrected for heteroscedasticity. Means were not centered (Echambadi & Hess, 2007) – (for critical discussion see Hayes et al., 2012; included in the appendix, p 115). According to Cohen (1988) effect sizes for values of $R^2$ can be defined as follows: small effect = .02; medium effect = .13; large effect = .26. Predictions regarding the nature of the effects were made a priori, hence no adjustments for multiple comparisons were made (Rothman, 1990). For the sake of parsimony, only significant results ($p < .050$) or trend-level findings ($p < .100$) are reported.
Results

Descriptive statistics for demographic and sleep variables are reported in Table 2, and cognitive data are provided in Table 3. Simple group comparisons are provided, however, they did not take into account the effect of covariates, hence the regression analyses below.

Bivariate associations for all study variables can be seen in Table 4.
Table 2. Descriptive statistics for demographic and sleep variables for currently depressed versus not currently depressed participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Currently Depressed</th>
<th>n</th>
<th>Not Currently Depressed</th>
<th>n</th>
<th>p</th>
<th>Partial $\eta^2$ or Cramer's Phi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>59.80(6.76), 51-74</td>
<td>10</td>
<td>60.94(6.45), 50-79</td>
<td>33</td>
<td>.631</td>
<td>.01</td>
</tr>
<tr>
<td>Gender</td>
<td>4:40%</td>
<td>10</td>
<td>7:21%</td>
<td>33</td>
<td>.233</td>
<td>.18</td>
</tr>
<tr>
<td>Taking antidepressants</td>
<td>8: 80%</td>
<td>10</td>
<td>9: 27.27%</td>
<td>33</td>
<td>&lt;.001</td>
<td>.46</td>
</tr>
<tr>
<td>PSQI Use of Sleep Medication $^2$</td>
<td>1.80(1.55), 0-3</td>
<td>10</td>
<td>0.31(0.64), 0-3</td>
<td>33</td>
<td>&lt;.001</td>
<td>.33</td>
</tr>
<tr>
<td>Currently employed</td>
<td>5: 50%</td>
<td>10</td>
<td>20: 60.61%</td>
<td>33</td>
<td>.551</td>
<td>.09</td>
</tr>
<tr>
<td>PHQ-9 $^3$</td>
<td>16.60 (4.23), 11-24</td>
<td>10</td>
<td>2.84 (2.97), 0-9</td>
<td>33</td>
<td>&lt;.001</td>
<td>.77</td>
</tr>
<tr>
<td>Cortisol (AUC$_G$)</td>
<td>598.98(469.20), 168.36-1557.42</td>
<td>8</td>
<td>334.89(186.24), 67.77-817.36</td>
<td>27</td>
<td>.040</td>
<td>.09</td>
</tr>
<tr>
<td>RDI $^4$</td>
<td>4.75[16.20], 0.10-94</td>
<td>10</td>
<td>3.45[10.20], 0-31</td>
<td>32</td>
<td>.443</td>
<td>.02</td>
</tr>
<tr>
<td>ODI-3 $^5$</td>
<td>2.83[13.62], 0.15-23.69</td>
<td>10</td>
<td>1.83[4.96], 0-22.02</td>
<td>28</td>
<td>.907</td>
<td>0</td>
</tr>
<tr>
<td>MEQ total $^6$</td>
<td>52.33(5.98), 42-61</td>
<td>9</td>
<td>57.90(9.26), 37-75</td>
<td>33</td>
<td>.099</td>
<td>.07</td>
</tr>
<tr>
<td>Actigraphy TST $^7$</td>
<td>395.10(123.71), 144.19-553.60</td>
<td>10</td>
<td>405.89(60.67), 217.21-510.05</td>
<td>33</td>
<td>.177</td>
<td>0</td>
</tr>
<tr>
<td>Actigraphy Sleep Efficiency $^8$</td>
<td>77.12(19.80), 24.36-89.35</td>
<td>10</td>
<td>83.35(8.55), 53.84-92.47</td>
<td>33</td>
<td>.058</td>
<td>.05</td>
</tr>
<tr>
<td>Actigraphy Sleep Latency $^9$</td>
<td>21.21(16.26), 4.54-60</td>
<td>10</td>
<td>13.74(11.77), 1.27-48.14</td>
<td>33</td>
<td>.079</td>
<td>.06</td>
</tr>
<tr>
<td>Actigraphy WASO $^{10}$</td>
<td>44.74(14.84), 23.77-70</td>
<td>10</td>
<td>53.03(28.83), 11.15-118.80</td>
<td>33</td>
<td>.153</td>
<td>.03</td>
</tr>
<tr>
<td>%REM</td>
<td>27.71(9.73), 14.50-43.90</td>
<td>10</td>
<td>23.48(7.31), 3.30-40.70</td>
<td>33</td>
<td>.073</td>
<td>.05</td>
</tr>
<tr>
<td>%SWS</td>
<td>7.68(6.35), 0-17.70</td>
<td>10</td>
<td>13.45(7.61), 1.40-36.50</td>
<td>33</td>
<td>.018</td>
<td>.10</td>
</tr>
</tbody>
</table>

Note. 1 Sample size varies due to missing data; PSQI = Pittsburgh Sleep Quality Index; PHQ-9 = Patient Health Questionnaire; AUC$_G$ = Area Under the Curve with reference to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen Desaturation Index, below 3%; MEQ = Morningness Eveningness Questionnaire; TST = Total Sleep Time (mins); WASO = Wake After Sleep Onset (mins); REM = Rapid Eye Movement sleep; SWS = Slow Wave Sleep; $^2$ Higher scores indicate greater use of sleep medication; $^3$ Higher scores indicate greater depressive symptoms; $^4$ Higher scores indicate greater respiratory disturbance; $^5$ Higher scores indicate greater oxygen desaturation; $^6$ Higher scores indicate morning preference; $^7$ Higher scores indicate longer sleep time; $^8$ Higher scores indicate better sleep efficiency (%); $^9$ Higher scores indicate more time spent awake after sleep onset; $^{10}$ Two-tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of the expected effect could be made, in which case two-tailed tests are reported.
Table 3. Descriptive statistics for cognitive variables (z scores) for currently depressed and not currently depressed participants

<table>
<thead>
<tr>
<th></th>
<th>Currently Depressed</th>
<th></th>
<th>Not Currently Depressed</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD), range</td>
<td>n1</td>
<td></td>
<td>n1</td>
<td>p</td>
<td>Partial η² or Cramer’s Phi</td>
<td></td>
</tr>
<tr>
<td>NART converted WAIS-R IQ ³</td>
<td>108.78(10.01), 95.88 – 119.44</td>
<td>10</td>
<td>113.58(8.72), 89.68-128.12</td>
<td>33</td>
<td>.148 ⁶</td>
<td>.05</td>
<td></td>
</tr>
<tr>
<td>Vigilance (speed)⁴</td>
<td>2.66(1.21), 1.15-4.69</td>
<td>10</td>
<td>2.45 (0.82), 0.10-3.90</td>
<td>33</td>
<td>.268</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>Vigilance (accuracy)⁵</td>
<td>2.26(0.76), 1.27-3.21</td>
<td>10</td>
<td>2.33(0.70), 1.3-3.21</td>
<td>33</td>
<td>.395</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Long-term memory (speed)⁴</td>
<td>3.57(0.10), 3.37 – 3.73</td>
<td>10</td>
<td>3.48(0.14), 3.20-3.76</td>
<td>33</td>
<td>.037</td>
<td>.08</td>
<td></td>
</tr>
<tr>
<td>Long-term memory (accuracy)⁵</td>
<td>2.54(0.94), 1.3-7.8</td>
<td>10</td>
<td>3.06(0.76), 1.39-4.41</td>
<td>33</td>
<td>.042</td>
<td>.07</td>
<td></td>
</tr>
<tr>
<td>Delayed recall (speed)⁴</td>
<td>0.17(0.07), 0.02 – 0.26</td>
<td>10</td>
<td>0.15(0.07), 0-0.3</td>
<td>33</td>
<td>.197</td>
<td>.02</td>
<td></td>
</tr>
<tr>
<td>Delayed recall (accuracy)⁵</td>
<td>3.08(1.02), 1-4.41</td>
<td>10</td>
<td>3.30(0.67), 2.10-4.67</td>
<td>33</td>
<td>.215</td>
<td>.02</td>
<td></td>
</tr>
<tr>
<td>Working memory (speed)⁴</td>
<td>2.87(0.11), 2.66 – 3</td>
<td>10</td>
<td>2.91(0.08), 2.75–3.05</td>
<td>33</td>
<td>.115</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>Working memory (accuracy)⁵</td>
<td>1.27(0.09), 1.16 – 1.40</td>
<td>10</td>
<td>1.34(0.16), 0.96-1.57</td>
<td>33</td>
<td>.083</td>
<td>.05</td>
<td></td>
</tr>
<tr>
<td>Executive functioning (speed)⁴</td>
<td>1.42(0.15), 1.27-1.69</td>
<td>10</td>
<td>1.34(0.15), 1-1.69</td>
<td>33</td>
<td>.096</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>Executive functioning (accuracy)⁵</td>
<td>2.22(0.81), 1.09-3.54</td>
<td>9</td>
<td>2.73(0.53), 1-3.62</td>
<td>32</td>
<td>.015</td>
<td>.12</td>
<td></td>
</tr>
</tbody>
</table>

Note. ¹Sample size varies due to missing data; NART = National Adult Reading Test; WAIS-R = Wechsler Adult Intelligence Scale Revised; ²Higher scores indicate a greater number of errors; ³Higher scores indicate higher IQ; ⁴Higher scores indicate slower performance; ⁵Higher scores indicate better performance; ⁶two-tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported.
Table 4. Correlation matrix (Pearson correlation coefficients) for all study variables

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>IQ</th>
<th>PSQI Use of sleep medication</th>
<th>Vigilance (speed)</th>
<th>Vigilance (accuracy)</th>
<th>RDI</th>
<th>ODI-3</th>
<th>MEQ</th>
<th>Cortisol AUCG</th>
<th>TST (mins)</th>
<th>Sleep latency (mins)</th>
<th>Sleep efficiency (%)</th>
<th>WASO (mins)</th>
<th>% REM</th>
<th>% NREM</th>
<th>% SWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-</td>
<td>.05†</td>
<td>-0.09</td>
<td>-0.09</td>
<td>-0.52†</td>
<td>.47†</td>
<td>-0.13</td>
<td>-0.22</td>
<td>-0.35†</td>
<td>0.17</td>
<td>-0.28†</td>
<td>0.20</td>
<td>-0.27†</td>
<td>0.28†</td>
<td>-0.12</td>
<td></td>
</tr>
<tr>
<td>Cortisol AUCG</td>
<td>-0.22</td>
<td>.21†</td>
<td>0.39†</td>
<td>-0.19</td>
<td>0.01</td>
<td>-0.02</td>
<td>-0.20</td>
<td>-0.25†</td>
<td>-0.19</td>
<td>0.13</td>
<td>-0.26</td>
<td>0.10</td>
<td>0.33†</td>
<td>0.33†</td>
<td>-0.06</td>
<td></td>
</tr>
<tr>
<td>Vigilance (speed)</td>
<td>0.31†</td>
<td>-0.21</td>
<td>-0.12†</td>
<td>-0.09</td>
<td>0.07</td>
<td>0.04</td>
<td>0.22†</td>
<td>-0.19</td>
<td>-0.10</td>
<td>0.04</td>
<td>-0.14</td>
<td>0.32†</td>
<td>-0.12</td>
<td>0.12</td>
<td>0.11</td>
<td></td>
</tr>
<tr>
<td>Vigilance (accuracy)</td>
<td>0.06</td>
<td>.17</td>
<td>0.06†</td>
<td>-0.07</td>
<td>0.16</td>
<td>0.03</td>
<td>0.01</td>
<td>-0.09</td>
<td>0.04</td>
<td>0.04</td>
<td>-0.04</td>
<td>0.03</td>
<td>-0.04</td>
<td>0.03</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Long-term memory (speed)</td>
<td>-0.38†</td>
<td>-0.06</td>
<td>0.07†</td>
<td>0.33†</td>
<td>0.04</td>
<td>0.25</td>
<td>0.26</td>
<td>0.08†</td>
<td>-0.02</td>
<td>0.09</td>
<td>0.04</td>
<td>-0.04</td>
<td>-0.04</td>
<td>0.03</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Long-term memory (accuracy)</td>
<td>0.12</td>
<td>-0.05</td>
<td>-0.10†</td>
<td>-0.33†</td>
<td>-0.01</td>
<td>0.19</td>
<td>-0.19</td>
<td>0.13†</td>
<td>0.12</td>
<td>0.08</td>
<td>0.16</td>
<td>0.05</td>
<td>0.01</td>
<td>-0.13</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Delayed recall (speed)</td>
<td>0.16</td>
<td>-0.49†</td>
<td>0.12†</td>
<td>0.33†</td>
<td>-0.09</td>
<td>0.06</td>
<td>0.10</td>
<td>0.19†</td>
<td>-0.27</td>
<td>-0.02</td>
<td>0.25</td>
<td>-0.14</td>
<td>0.09</td>
<td>-0.23</td>
<td>0.22</td>
<td>0.19</td>
</tr>
<tr>
<td>Delayed recall (accuracy)</td>
<td>-0.11</td>
<td>0.36†</td>
<td>-0.27†</td>
<td>-0.38†</td>
<td>-0.02</td>
<td>-0.06</td>
<td>-0.02</td>
<td>-0.08†</td>
<td>-0.03</td>
<td>-0.12</td>
<td>-0.12</td>
<td>0.02</td>
<td>-0.26†</td>
<td>-0.01</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Working memory (speed)</td>
<td>0.42†</td>
<td>-0.19</td>
<td>-0.26†</td>
<td>0.61†</td>
<td>-0.13</td>
<td>-0.03</td>
<td>-0.15</td>
<td>0.08†</td>
<td>-0.35†</td>
<td>-0.17</td>
<td>0.25</td>
<td>-0.21</td>
<td>0.15</td>
<td>-0.14</td>
<td>0.14</td>
<td>0.03</td>
</tr>
<tr>
<td>Working memory (accuracy)</td>
<td>0.13</td>
<td>-0.02</td>
<td>0.04†</td>
<td>-0.11</td>
<td>0.20</td>
<td>0.01</td>
<td>0.10</td>
<td>0.04†</td>
<td>-0.13</td>
<td>-0.30†</td>
<td>-0.03</td>
<td>0.11</td>
<td>-0.09</td>
<td>0.08</td>
<td>-0.17</td>
<td>0.16</td>
</tr>
<tr>
<td>Executive functioning (speed)</td>
<td>0.34†</td>
<td>-0.29†</td>
<td>0.15†</td>
<td>-0.38†</td>
<td>0.03</td>
<td>0.16</td>
<td>0.21</td>
<td>0.04†</td>
<td>-0.30†</td>
<td>-0.03</td>
<td>0.11</td>
<td>-0.09</td>
<td>0.08</td>
<td>-0.19</td>
<td>0.09</td>
<td>0.09</td>
</tr>
<tr>
<td>Executive functioning (accuracy)</td>
<td>-0.09</td>
<td>0.54†</td>
<td>-0.39†</td>
<td>-0.18</td>
<td>-0.10</td>
<td>-0.02</td>
<td>0.09</td>
<td>0.02†</td>
<td>0.00</td>
<td>0.05</td>
<td>-0.22</td>
<td>0.01</td>
<td>0.11</td>
<td>0.01</td>
<td>0.0</td>
<td>0.14</td>
</tr>
</tbody>
</table>

Note. PSQI = Pittsburgh Sleep Quality Index; Cortisol AUCG = Area under the curve with respect to ground; RDI = Respiratory Disturbance Index; ODI-3 = Oxygen desaturation Index, below 3%; MEQ = Morningness-Eveningness Questionnaire; TST = Total sleep time (mins); WASO = Wake after sleep onset (mins); REM = Rapid eye movement sleep; NREM = Non Rapid eye movement sleep; SWS = Slow wave sleep; Higher scores indicate greater use of sleep medication; Higher scores indicate slower speed; Higher scores indicate better performance; Higher scores indicate greater respiratory disturbance; Higher scores indicate greater oxygen desaturation; Higher scores indicate morning preference; Higher scores indicate longer sleep time; Higher scores indicate longer sleep latency; Higher scores indicate lower sleep efficiency; Two tailed: all tests were one-tailed based on theory, except those where no prediction of the direction of expected effect could be made, in which case two-tailed tests are reported; † p < .05, ‡ p < .01.
Cortisol
Morning salivary cortisol differed between currently depressed and not currently depressed groups $t(33) = 1.80$, $p = .041$ (one-tailed), $d = .66$, indicating a moderate effect. This confirms elevated cortisol in the depressed sample, providing a biological index of depression.

Vigilance (speed)
Standard regression analysis revealed that age ($\beta = .04$, $p = .005$) was a significant covariate of vigilance ($R^2 = .09$, $p = .005$), indicating 9% of variance explained. After controlling for age, WASO ($\beta = .01$, $p = .043$) was a significant predictor of vigilance speed ($R^2 = .17$, $p = .007$), explaining an additional 8% of variance.

There was no effect of depression and no evidence of moderation effects.

Vigilance (accuracy)
Standard regression analysis revealed that there were no significant covariates and none of the sleep variables predicted vigilance accuracy.

There was no effect of depression and no evidence of moderation effects.

Long-term memory (speed)
Standard regression analysis revealed that age ($\beta = .01$, $p = .008$) was a significant covariate of long-term memory speed ($R^2 = .15$, $p = .008$), however, none of the sleep variables significantly predicted additional variance.

When depression was added as a moderator of the relationship between %SWS and long-term memory speed, it explained an additional 18% of variance in long-term memory speed ($\beta = .16$, $p = .004$, overall model $R^2 = .33$, $p < .001$). However, depression did not moderate the relationship between sleep and long-term memory speed.

Long-term memory (accuracy)
Standard regression analysis revealed that gender ($\beta = 1.20$, $p < .001$) and vigilance speed ($\beta = -.36$, $p = .022$) were significant covariates of long-term memory accuracy ($R^2$
=.45, p < .001), indicating 45% of variance explained, however, no additional variance was explained by sleep variables.

There were no effects of depression or any interactions between depression and sleep.

Delayed recall (speed)
Standard regression analysis revealed that IQ (β = 0, p = .004) was a significant predictor of delayed recall speed \((R^2 = .24, p = .004)\). After controlling for IQ, %SWS (β = 0, p = .064) was a trend-level predictor of delayed recall speed \((R^2 = .31, p = .002)\), explaining an additional 7% of the variance. No other sleep variables were significant predictors of delayed recall (speed).

There were no direct effects of depression or interaction effects of depression and sleep.

Delayed recall (accuracy)
Standard regression analysis revealed that IQ (β = .03, p = .029) was a significant covariate of delayed recall accuracy \((R^2 = .13, p = .029)\). After controlling for IQ, WASO was a significant predictor of delayed recall accuracy (β = -.01, p = .023, \(R^2 = .20, p = .016\)), explaining an additional 7% of variance.

Although no other sleep variables were significant, there was a significant interaction effect. When depression was added as a moderator of the relationship between sleep latency and delayed recall accuracy, depression was not a significant predictor, but the interaction of sleep latency and depression reached trend-level (β = .05, p = .052), explaining an additional 14% of variance (overall model was significant, \(R^2 = .27, p = .011\)). Examination of conditional effects of sleep latency (X) on delayed recall accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group, also at trend-level (p = .065). There were no effects within the depressed group. That is, longer sleep latency predicted poorer delayed recall in the not currently depressed group only, but only at trend levels.
Figure 1. Interaction of depression and sleep latency predicting delayed recall accuracy (z scores)

Working memory (speed)

Standard regression analysis revealed that age ($\beta = 0.10, p = .033$) and vigilance speed ($\beta = .05, p < .001$) were significant covariates of working memory speed ($R^2 = .44, p < .001$), explaining 44% of the variance. Sleep variables did not help to explain working memory speed.

Depression was a significant independent predictor of working memory speed ($\beta = .25, p = .016$), and there was a significant interaction of total sleep time and depression ($\beta = 0, p = .007$), explaining an additional 14% of variance (overall model was significant, $R^2 = .58, p < .001$). Inspection of conditional effects of total sleep time (X) on working memory speed (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction effect was being driven by the depressed group ($p = .019$). This was not significant within the not currently depressed group. That is, shorter total sleep time predicted slower working memory speed in the depressed group only.

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10 Non-standardised Beta weight = 0.0003. All Beta weights were rounded to 2 decimal places, therefore reported as 0.
Figure 2. Interaction of depression and total sleep time predicting working memory speed (z scores)

Working Memory (accuracy)
Standard regression analysis revealed that there were no significant covariates of working memory accuracy. Total sleep time ($\beta = 0, p = .054$) was associated with working memory accuracy at trend-level ($R^2 = .09, p = .054$), indicating 9% of variance explained. Sleep efficiency ($\beta = 0, p = .015$) was a significant predictor of working memory accuracy ($R^2 = .09, p = .015$), indicating 9% of variance explained.

Depression was not a significant predictor of working memory accuracy and there were no interaction effects.

Executive Functioning (speed)
Standard regression analysis revealed that age ($\beta = .01, p = .014$) and gender ($\beta = .14, p = .009$) were significant covariates of executive functioning speed ($R^2 = .27, p = .012$), indicating 27% of variance explained. No sleep variables were independent predictors of executive functioning speed.

Depression was not a significant independent predictor of executive functioning (speed), however, there was a significant interaction effect of WASO and depression ($\beta = .01, p = .037$), explaining an additional 17% of variance (overall model was significant, $R^2 = .44, p < .001$). Inspection of conditional effects of WASO (X) on executive functioning speed (Y) at values of the moderator (depression: 0 or 1), revealed that the interaction effect was being driven by the depressed group at trend-
level ($p = .084$). This was not significant within the not currently depressed group. That is, greater WASO was linked to slower executive functioning speed in the depressed group only, but at trend level.

![Figure 3. Interaction of depression and WASO predicting executive functioning speed (z scores)](image)

**Executive Functioning (accuracy)**

Standard regression analysis revealed that IQ ($\beta = .03, p = .014$) was a significant predictor of executive functioning accuracy ($R^2 = .18, p = .014$), indicating 18% of variance explained. No sleep variables were independent predictors of executive functioning accuracy.

Subsequent moderation analysis revealed that depression was a significant predictor of executive functioning accuracy at trend-level ($\beta = 2.72, p = .077$) and there was a significant interaction effect of total sleep time and depression ($\beta = -.01, p = .029$), explaining an additional 30% of variance in executive functioning accuracy (overall model was significant, $R^2 = .48, p = .002$). Inspection of conditional effects of total sleep time (X) on executive functioning accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group ($p = .019$). The association of total sleep time and depression was not significant within the depressed group. That is, longer total sleep time was linked to better executive functioning in the not currently depressed group only.
Further moderation analyses revealed that depression was a significant independent predictor of executive functioning accuracy in the context of sleep efficiency ($\beta = 3.89$, $p = .004$), and there was a significant interaction effect of sleep efficiency and depression ($\beta = -.05$, $p < .001$), explaining an additional 25% of variance (overall model was significant, $R^2 = .52$, $p < .001$). Inspection of conditional effects of sleep efficiency (X) on executive functioning accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the association of sleep efficiency and executive functioning accuracy was significant in both the depressed group ($p < .001$) and the not currently depressed group at trend-level ($p = .053$). That is, higher sleep efficiency was linked to better executive functioning within the not currently depressed group and poorer sleep efficiency was linked to better executive functioning within the depressed group. However, inspection of scatterplots revealed that the association within the depressed group was being driven by an outlier (a participant with very low sleep efficiency and high executive functioning). Once this score was removed, the interaction effect disappeared.

When depression was added as a moderator of sleep latency and executive functioning accuracy, it was a significant independent predictor of executive functioning ($\beta = -1.32$, $p = .007$) at trend-level, and there was a significant interaction effect of sleep latency and depression ($\beta = .05$, $p = .048$), explaining an additional 26% of variance (overall model was significant, $R^2 = .44$, $p = .019$). Inspection of conditional effects of sleep latency...
latency (X) on executive functioning accuracy (Y) at values of the moderator (depression: 0 or 1) revealed that the interaction was being driven by the not currently depressed group at trend-level ($p = .076$). The association of sleep latency and depression was not significant within the depressed group. That is longer sleep latency was linked to poorer executive functioning in the not currently depressed group only.

![Graph showing interaction of depression and sleep latency predicting executive functioning accuracy (z scores)](image)

**Figure 5.** Interaction of depression and sleep latency predicting executive functioning accuracy (z scores)

Therefore, overall results suggest that there was evidence of a different pattern of association of sleep and cognition in currently depressed versus not currently depressed participants. That is, specific aspects of actigraphic sleep, were linked to performance accuracy decrements in the not currently depressed group, and slower speed of processing in the currently depressed group.

**Discussion**

This study investigated the effect of sleep on cognition in older adults, and whether depression impacted on this relationship. Results indicate associations between sleep-wake patterns and cognition in the whole sample. By contrast, sleep architecture largely did not impact on cognition. Finally, depression altered the relationship between sleep and cognition, but only for aspects of sleep-wake patterns and not sleep stages. The results are discussed in turn.
Sleep and cognition in the whole sample

Sleep-wake patterns, as indexed by actigraphy, were linked to vigilance, memory and working memory in the entire sample. Specifically, greater WASO was linked to slower vigilance (speed) and poorer memory (delayed recall accuracy). Lower sleep efficiency and shorter sleep duration (at trend-level) were linked to poorer working memory accuracy.

Findings are consistent with an actigraphic study that found older insomniacs with greater WASO had slower reaction times on a sustained attention task and poorer working memory on a memory span task (Haimov et al., 2008). Results also replicate findings from self-report studies in older adults demonstrating the impact of poor sleep quality on memory (Schmutte et al., 2007) and working memory accuracy (Nebes et al., 2009). While some studies suggest that sleep quality is more important than sleep quantity (e.g., Blackwell et al., 2011), our results are in line with other studies showing a link between shorter self-reported sleep duration and poorer cognition (e.g., Ohayon & Vecchierini, 2005; Tworoger et al., 2006). Therefore, the current results suggest that sleep-wake patterns are important predictors of cognitive performance in older adults.

With regard to sleep stages, more SWS predicted slower memory performance (delayed recall tasks), albeit only at trend-level, but did not predict delayed recall accuracy. This is not in line with a previous SDMC study showing a positive relationship between SWS and declarative memory in older adults (Backhaus et al., 2007), and might indicate a discontinuation of the SWS-memory link. Results, thus, indicate that the relationship between sleep architecture and cognition might change with age (‘functional-dissociation’ account).

Overall, the lack of a positive correlation between SWS and REM and cognitive performance, lends support for the ‘functional-dissociation’ account of sleep stages and cognition, rather than the neurodevelopmental theories.

The role of depression

This study revealed medium to large interaction effects of depression and actigraphic sleep variables in predicting cognition. That is, depression had a significant impact on the relationship between sleep-wake patterns (but not sleep architecture) and cognition.
Sleep and cognition in the depressed group: Shorter total sleep time and greater WASO (at trend-level) were linked to slower performance on tasks of working memory and executive functioning, respectively. These findings are in line with a questionnaire study that linked poorer overall sleep quality (Global PSQI) to executive functioning problems in participants with high levels of depressive symptoms (Sutter et al., 2012), and with an actigraphy study that linked greater WASO to impaired executive functioning in older adults with depression (Naismith et al., 2011) – albeit these studies point to impaired accuracy rather than speed, which could be due to the different tasks used in the studies.

The impact of total sleep time on speed of working memory in the depressed group suggests that sleep duration, in addition to sleep quality, is an important factor in cognition in older adults with depression. Thus, although confined to measures of speed, these results suggest sleep-related deficits in working memory and executive functioning in depressed older adults, which is consistent with studies implicating fronto-subcortical networks in depression, cognition and sleep (Naismith et al., 2011).

Sleep and cognition in not currently depressed older adults: Shorter total sleep time and longer sleep latency (at trend-level) were associated with poorer executive functioning performance. This is consistent with actigraphy research in older adults, which shows that longer sleep latency is linked to poorer executive functioning (Blackwell et al., 2006), and with research in adults demonstrating the impact of sleep loss on executive functioning (e.g., Harrison & Horne, 2000).

Longer sleep latency affected memory (delayed recall accuracy) at trend-level. Results are consistent with one of the few published actigraphy studies that demonstrated a significant association of longer sleep latency and poorer overall cognition (which included measures of delayed recall) in older women (Blackwell et al., 2006). Findings are also consistent with the notion that poor sleep impacts on the medial temporal lobes and hippocampus, causing memory impairment (Drummond et al., 2000). However, the majority of evidence for this association in older adults derives from self-report studies (e.g., Schmutte et al., 2007). Therefore, the current findings provide further evidence of sleep-related memory deficits in older adults using actigraphy.
Taken together, the current findings offer novel evidence of a distinct pattern of sleep-related cognitive impairment in currently depressed versus not currently depressed older adults. In general terms, sleep disturbance was linked to poorer accuracy in the not currently depressed group, but slower processing speed in the depressed group. The finding in the depressed group is consistent with research indicating decreased processing speed in older adults with depression (McDermott & Ebmeier, 2009; Nebes et al., 2000; Sheline et al., 2006). However, it is unclear why we found accuracy deficits in the not currently depressed group, but only deficits in speed in the depressed group.

Overall, our results suggest that the relationship between sleep and cognition is qualitatively different in currently depressed versus not currently depressed older adults. Older adults with current MDD have a specific profile of sleep-related cognitive performance compared to those without MDD, suggesting that depression exerts a specific role on the relationship between sleep and cognition. An attractive explanation, therefore, suggests that the reason for the lack of positive associations found between sleep stages and cognition (‘functional-dissociation’ account) could be due to the presence of depression in older age, which alters the relationship between sleep and cognitive performance. However, there are problems with this explanation because the relationship between sleep-wake patterns and cognitive performance is in line with findings in the literature. In addition, we found no association of sleep architecture and cognition, in either depressed or non-depressed groups. We are limited in the conclusions we can make, but we can say that depression does impact on the relationship between sleep and cognition, but does not appear to be the reason for the changing relationship of sleep architecture and cognition observed in this study, and other studies. However, future studies would need to directly test a mediating effect of depression on the relationship between sleep architecture and cognition in order to derive firm conclusions.

Future directions
While this study demonstrates exciting and novel relationships between sleep, depression and cognition in ageing, further research is essential. Current findings provide tentative support for the ‘functional dissociation’ account, but the mechanistic processes cannot be identified using the current methodologies.
There were a number of limitations to this study. First, the moderating effects of depression must be interpreted with caution due to the substantially higher proportion of not currently depressed participants compared to currently depressed participants. This was due to difficulty recruiting depressed older adults who typically suffer from decreased motivation and loss of interest (Fiske et al., 2009).

Second, Hayes’ moderation method (Hayes et al., 2012) is useful in exploring the nature of any significant interaction effects found. However, while the exploration at the group level provides important information on how to interpret any interactions found, which is not available from conventional moderation analyses, this reduces the power of the analyses. Although limited by sample size and power, a strength of the current study was the size of the interaction effects of sleep and depression, which ranged from medium to large (Cohen, 1988) explaining a significant proportion of variance in cognitive functioning. Nevertheless, results warrant further investigation in a larger sample to see if these findings are replicated.

Third, the neuropsychological tests used in this study as part of the CogState battery, while well-validated (e.g., Falleti et al., 2003; Weaver Cargin et al., 2008), were broad. For example, the Groton Maze Learning task is said to tap into various different cognitive domains, such as problem-solving, decision making, attention and spatial memory (Pietrzak et al., 2008). Therefore, future studies may wish to investigate the possibility of using more precise cognitive tasks which tap into different types of memory and which assess hippocampal function directly.

Fourth, recent research has highlighted the important role of neurophysiological events associated with specific sleep stages in cognition, such as REM sleep theta and sleep spindles (e.g., Cox, Hofman, & Talamini, 2012; Fogel & Smith, 2006). While it was beyond the scope of this study, future research may wish to consider spectral and spindle analysis of sleep in addition to traditional sleep staging.

Future studies may also wish to consider an exploration of the impact of daytime sleepiness on cognition. Sleepiness was not assessed in this study given the focus on objective measures of sleep and the fact that the objective alternatives to self-reports of sleepiness (e.g., The Epworth Sleepiness Scale) are demanding in terms of time and effort.
Finally, given that this was a cross-sectional study, the direction of the relationship of sleep and cognition must be interpreted with caution. However, the majority of research in this area supports a uni-directional effect of sleep impacting on cognition, with few exceptions (e.g., see Haimov & Shatil, 2013, for an example of effects of cognition on sleep). In order to derive firm conclusions about the direction of the effects, intervention or sleep-deprivation studies would be needed.

**Conclusions**

This study investigated the association of sleep-wake patterns and sleep architecture and cognition in older adults, and whether the presence of current MDD impacted on this relationship. Sleep-wake patterns, but not sleep stages, were associated with cognitive performance in our sample of older adults. Furthermore, sleep-wake patterns had dissociable effects on cognition in currently depressed versus not currently depressed older adults, such that poor sleep was associated with working memory and executive functioning *speed* in the currently depressed group, and with delayed recall and executive functioning *accuracy* in the not currently depressed group. Because sleep problems were associated with cognitive deficits in both the clinical and not currently depressed group, results highlight the need to assess sleep in all older adults. Early detection of sleep problems is important given that research has shown that treatments for sleep problems, whether behavioural or pharmacological, can improve cognition in older adults (Pace-Schott & Spencer, 2011).
Chapter 6
General discussion

Overview of aims and hypotheses

The objectives of this thesis were to conduct a thorough investigation into the association of sleep and cognition in the context of ageing and to determine how depression impacted on this relationship.

Specifically, this thesis aimed to:

1. investigate how self-reported sleep quality changed across the lifespan in a community sample
2. examine the association between self-reported sleep and cognition in a community cohort varying in severity on subclinical symptoms of depression
3. characterise the sleep of older adults with Major Depressive Disorder (MDD)
4. assess the relationship between sleep and cognition in older adults with and without current MDD.

There were four specific hypotheses. It was predicted that:

i) Self-reported sleep quality would decline across the lifespan.
ii) Sleep would be associated with cognition, but this relationship would depend on the method of sleep assessment. Specifically, self-reported sleep quality would be associated with cognitive complaints across the lifespan; sleep-wake patterns (assessed using actigraphy) would impact on cognitive performance in older adults; and finally, there were contrasting hypotheses regarding sleep architecture: (a) there would be a positive relationship between %SWS and %REM and cognitive performance in older adults (consistent with neurodevelopmental accounts); (b) there would be no association between sleep stages and cognition as a result of age-related changes in this relationship (‘the functional-dissociation’ account).

iii) There would be specific sleep impairments in individuals with MDD.
iv) Depression would have a significant impact on the relationship between sleep and cognition in older adults.
Brief overview of findings

Chapter 2 investigated whether self-reported sleep (measured using the Pittsburgh Sleep Quality Index total score and factor scores; PSQI) changed with age. Participants were 582 community adults (18–89 years). Psychological symptoms (depression, anxiety and stress) and risk of sleep-disordered breathing (SDB) were controlled for, since they are common in ageing and can affect sleep quality. Overall, results revealed that depression and SDB-risk had an impact on sleep quality. However, there were modest age-related differences in subjective sleep quality, not due to mood or SDB-risk.

Chapter 3 investigated associations between self-reported sleep (PSQI factor scores, and sleepiness on the Epworth Sleepiness Scale) and cognitive complaints in 205 community adults (19–89 years), controlling for subclinical depression and risk of SDB. Neurobiological models suggest that poor sleep has direct biological effects on the brain, which cause cognitive deficits. In contrast, the vigilance model suggests that poor sleep affects cognition via decreased arousal levels which cause lapses in attention. For the first time, these theoretical models were contrasted using self-reports chosen as proxies for these two hypotheses. Results provided support for the vigilance model in that self-reported sleep largely affected cognition via effects on attention.

Chapter 4 aimed to characterise the sleep of older adults (50–79 years) with and without MDD. Particularly, it aimed to investigate whether sleep abnormalities would be present in participants with a history of depression but who were no longer symptomatic. The sleep of older adults with a current \( n = 10 \) and past diagnosis of MDD \( n = 14 \), and no history of clinical depression \( n = 19 \), was compared using a combination of subjective (questionnaires) and objective (PSG, actigraphy) measures of sleep. Overall, sleep problems were a feature of current MDD, and increased across the sample with increasing severity of depression.

Chapter 5 sought to investigate the impact of multiple aspects of sleep (using PSG and actigraphy) on cognitive performance (using a battery of cognitive tasks) in older adults, who were currently depressed \( n = 10 \) and not currently depressed \( n = 33 \). An important focus of this chapter was to investigate how the presence of MDD would impact on the relationship between sleep and cognition. Findings revealed that sleep-wake disturbances were linked to poorer cognitive performance across the whole
sample. In contrast, sleep architecture (%REM and %SWS) did not impact significantly on cognition. Depression moderated the relationship between sleep and cognition, such that there was a distinct association of sleep-wake pattern disturbances and cognitive deficits depending on the presence of depression. Overall, the presence of current MDD had a significant impact on the relationship between sleep-wake patterns and cognitive performance.

**Discussion of findings**

Each of the main hypotheses was wholly or partially supported by the findings of the studies in this thesis. Findings relating to each hypothesis are discussed:

**Hypothesis 1 – Self-reported sleep quality would decline across the lifespan**

Self-reports of sleep were selected to allow for recruitment of a large sample of adults across the lifespan in order to examine how subjective sleep changes with age (and whether subclinical depression and risk of SDB affects this relationship, see below). This study attended to an area of inconsistency in previous studies. That is, while age-related changes in objective sleep are widely reported (e.g., McCrae, 2009), there is some disagreement over whether self-reported sleep changes with age. Some studies suggest that subjective sleep problems increase with age (e.g., Stein et al., 2008), whereas others suggest that the sleep of older adults does not differ from that of middle-aged adults (e.g., Middelkoop et al., 1996).

This study found changes in some, but not all, dimensions of self-reported sleep, and that age was modestly linked to poorer overall sleep quality (PSQI global scores). In contrast to previous studies, the current examination of PSQI factor scores enabled investigation of distinct dimensions of subjective sleep quality, which other studies did not address. This revealed that age was linked to global sleep quality, which was largely driven by effects on Sleep Efficiency, a measure of night-time sleep quality. In contrast, no relationship was found between age and Perceived Sleep Quality and Daily Disturbances. A possible explanation for these findings is that older individuals adjust their expectations about their sleep quality (Buysse et al., 1991). Furthermore, they may perceive fewer effects of poor sleep on their daily functioning due to reduced daytime responsibilities, such as full-time employment (Zilli et al., 2009).
In order to control for the possibility that sleep problems in older adults are linked to psychological symptoms or SDB, rather than ageing, this study examined for the contribution of depression, anxiety and stress (using the Depression Anxiety Stress Scale; DASS-21), and risk of SDB (using the Berlin Questionnaire). Results revealed that although related to sleep quality, depression and SDB-risk did not explain the relationship between sleep and age. Nevertheless, given links to sleep quality and age, measures of psychological symptoms and SDB-risk, should remain as important covariates in similar studies.

Overall, findings provide partial support for Hypothesis 1 in that there was a subtle, but significant, decline in self-reported sleep quality with age. Thus, this study provided novel information concerning how self-rated sleep quality changes across the lifespan. Subjective reports of sleep are particularly important components of sleep quality given that they are used in clinical settings to determine the need for and type of treatment.

**Hypothesis 2 – Sleep would be associated with cognition depending on the method of sleep assessment**

Results pertaining to each method of sleep assessment are presented here:

*Self-reports of sleep and cognition*

In line with Hypothesis 2, results from Chapter 3 showed that self-reported sleep was linked to cognitive complaints in community adults. Results were consistent with predictions based on the vigilance model only. That is, sleep factors related to low arousal predicted self-rated attention deficits, which were also largely responsible for subjective memory and executive functioning deficits.

The wide age range of this sample allowed investigation of the impact of age on the relationship between subjective sleep and cognition. Results indicate that sleep problems predicted cognitive complaints over and above the effect of age. If these findings apply to objective measures of cognition, results suggest that sleep problems might account for age-related cognitive deficits. This research question is addressed in a sample of older adults in Chapter 5.

Depression was an important factor in the relationship between self-reported sleep and cognition, and even explained the relationship between PSQI Sleep Efficiency and
executive functioning. It is possible that this is because low mood is linked to poor self-rated cognition, and not necessarily objective cognition (e.g., Zlatar et al., 2014), however this finding highlights the importance of controlling for depression in all investigations of sleep and cognition. Furthermore, results are clinically relevant as cognitive complaints are clinical indicators of functioning, which are often used as markers of objective cognitive problems (Hohman, Beason-Held, & Rednick, 2011).

**Objective measures of sleep and cognition**

Chapter 5 assessed the impact of sleep-wake patterns, measured using actigraphy, and sleep architecture abnormalities (%REM and %SWS), measured using home-PSG, on cognitive performance within the same sample of older adults with and without depression. This allowed for a novel examination of how sleep-wake patterns, as well as sleep stages, affect cognition in older adults.

**Sleep-wake patterns**

Chapter 5 supported the hypothesis of sleep-wake disturbances being linked to poorer cognitive performance in older adults. Across the whole sample (irrespective of depression status), results revealed that disturbed sleep-wake patterns (assessed with actigraphy) were associated with poorer cognition. Specifically, measures of disturbed sleep-wake patterns, such as greater time spent awake after sleep onset (WASO) and lower sleep efficiency were linked to slower performance on tasks of vigilance, as well as poorer delayed recall and working memory accuracy. Consistent with the existing literature in older adults (Blackwell et al., 2011; Miyata et al., 2013), these results also importantly extend previous findings by linking sleep-wake disturbances to long-term memory deficits in older adults, which have previously been linked to self-reports of poor sleep (Schmutte et al., 2007), but not to actigraphic measures of poor sleep.

An interesting finding was that shorter total sleep time was associated with poorer working memory accuracy, albeit at trend-level. This finding stands in contrast to previous actigraphy studies in older adults showing no association of short sleep duration and poor cognitive functioning (e.g., Blackwell et al., 2006), but is in line with previous self-report studies in older adults linking shorter sleep duration to poorer cognition (Ohayon & Vecchierini, 2005; Tworoger et al., 2006). Hence, results of this study appear to oppose suggestions that sleep quality is more important in cognition than sleep quantity (e.g., Blackwell et al., 2006). However, given the lack of actigraphy
studies in older adults, there is a need for replication of these results.

Sleep architecture
There were no significant associations of sleep architecture (%REM and %SWS) and cognitive performance in this sample of older adults. Findings are in direct contrast to neurodevelopmental accounts of sleep and cognition which suggest a close relationship of sleep architecture and cognition, and rather suggest that the relationship of sleep architecture and cognition changes with age (the ‘functional-dissociation’ account). Results revealed one trend-level negative relationship between SWS and delayed recall speed. This was in the opposite direction to predictions based on the neurodevelopmental account that SWS should be linked to better, or faster, cognitive performance. However, results are in line with a previous study showing a negative association of SWS and memory, suggesting that SWS may be associated with poorer cognition in older adults (Scullin, 2013). Thus, this finding is important as it supports a changing relationship of sleep architecture and cognition in ageing.

Hypothesis 3 – There would be specific sleep impairments in individuals diagnosed with depression
Chapter 4 is the first study to include multiple measures of subjective (questionnaires) and objective sleep (actigraphy, home-PSG) within the same sample of older adults with and without clinical depression. Consistent with Hypothesis 3, results revealed significant sleep abnormalities associated with MDD across all sleep measures. Findings underscore studies in older adults with depression, who commonly report poor sleep (Foley et al., 2004). In contrast to the suggestion that sleep problems persist after remission (e.g., Cho et al., 2008), this study found that sleep problems were pronounced in individuals with current depression only (state-related).

A significant strength of this study was the comprehensive assessment of depression, using clinician diagnosis, questionnaires and clinical interviews, and morning salivary cortisol measures as a biological index of depression (Herbert, 2013). Results revealed that depression status and severity (assessed with diagnoses, interviews, and self-reports) were largely only associated with sleep architecture and self-reported sleep quality. In contrast, higher cortisol levels were associated with actigraphy measures (less total sleep time and poorer sleep efficiency), as well as sleep architecture (greater %REM and less %NREM). This suggests that different methods of assessing depression
yield different profiles of associations with sleep assessment methods, which is an important consideration for future studies.

These findings argue against the suggestion that sleep problems continue after remission from depression (trait-related). However, it is possible that the sample of individuals with a history of depression in this study had been in remission too long, or that continued antidepressant treatment impacted on sleep. Future studies, therefore, should clearly state how remission was defined and implement inclusion criteria regarding time since last depressive episode.

Hypothesis 4 – Depression would have a significant impact on the relationship between sleep and cognition in older adults

This is the first study to pursue this line of enquiry in a sample of older adults using objective measures of sleep. Chapter 5 sought to examine the impact of sleep on cognition across the entire sample of older adults (reviewed above), and then to address the question of whether depression impacted on this relationship.

An interesting finding was that when depression was investigated as a moderator, results revealed a different pattern of association between sleep-wake patterns and cognition in currently depressed versus not currently depressed older adults. In the depressed group, sleep-wake disturbances were associated with response time on tasks of executive functioning (at trend-level) and working memory. This is consistent with research reporting decreased processing speed in older adults with depression (e.g., McDermott & Ebmeier, 2009). Although confined to measures of speed, these results point to sleep-related deficits in executive functioning in depressed older adults, which is a consistent finding in depression (e.g., Naismith et al., 2011). In contrast, in the not currently depressed group, sleep-wake disturbances were associated with accuracy of performance on tasks of delayed recall (at trend-level) and executive functioning, which is consistent with findings in healthy older adults (e.g., Blackwell et al., 2011).

Overall, the presence of depression in older age was linked to a specific profile of association between sleep-wake patterns and tasks subserved by the frontal lobes, and was different from the performance of individuals without depression. The ‘functional-dissociation’ account proposes that the nature of the relationship between sleep and cognition changes as people age. The lack of an association between sleep architecture
and cognition is consistent with this account, and is at odds with the neurodevelopmental view, which would suggest there should be a close association between sleep architecture and cognition at all life stages. The conclusions that can be drawn from this thesis are that: (i) ageing is associated with a changing relationship between sleep architecture and cognition, although the mechanistic processes are complex and not easily understood, at least using the current methodologies; and (ii) depression does impact on the relationship between sleep and cognition, but does not appear to be the primary reason for the changing relationship of sleep architecture and cognition observed in this, and other studies, given the lack of association of sleep architecture in both the depressed and not currently depressed groups. However, it will important for subsequent research to directly investigate a mediating role of depression in the relationship between sleep stages and cognition.

**Theoretical and clinical implications**

The results of this thesis have important implications for theory and practice. First, in contrast to existing objective studies, dramatic changes in self-reported sleep quality were not a prominent feature of older age. This suggests that those older adults who do complain of poor sleep merit further clinical investigation, since poor sleep can impact on physical and psychological health.

Second, self-reports of sleep affected subjective cognition in adults via effects on attention (in support of the vigilance model). This has important implications for the community as it suggests that daytime effects of poor sleep, such as sleepiness, may impact on a range of cognitively mediated activities and behaviours, limiting functional independence in older people.

Third, sleep-wake disturbance was an important predictor of cognitive deficits in older adults, which supports the suggestion that age-related changes in cognition might be due to poor sleep (Altena et al., 2010). This is a most important finding because it suggests that age-related cognitive decline may not simply be a byproduct of ageing. Instead, the quality and quantity of sleep explained a significant proportion of the variance in age-related cognitive deficits. Mild cognitive impairment is often seen as an early indicator of dementia (Petersen et al., 1999) and is a most important stage for early interventions to delay the onset of dementia and cognitive decline. Unlike age and
genetics, sleep difficulties are potentially modifiable vulnerability factors, and can be used in interventions aimed at maximising sleep and cognition (Ferini-Strambi et al., 2003). This is important information for neuropsychologists, who should routinely screen for potential sleep problems in older adults.

Fourth, the lack of a significant association of sleep stages and cognition has empirical implications as it suggests that there is no longer a close relationship of sleep architecture and cognition in older age. Hence, current findings do not support the notion that age-related decreases in specific sleep stages might explain age-related deficits in cognition, and suggest that neurodevelopmental theories of sleep and cognition may need to be reappraised in view of the absence of a relationship in older adults. It is possible that age-related reductions in REM sleep and SWS reflect a decreased need for learning in older adults, which results in a weakened association with cognition. On the other hand, REM and SWS may not be critical for learning and cognition, and rather may be involved in broader neurodevelopmental processes, which are relevant in infants, but not in older adults. Therefore, the current findings suggest that sleep-wake patterns were more important than sleep architecture in the cognitive performance of older adults. Sleep-wake patterns are the result of biological, psychological and lifestyle influences, and are targets for intervention. These findings are promising as early detection and treatment of sleep-wake disturbances can improve cognition in older adults (Pace-Schott & Spencer, 2011). Regarding treatment options, while sleep-wake pattern disturbance and insomnia are often treated pharmacologically, non-pharmacological treatments are available, which have the benefit of avoiding problems of dependence and side-effects. Non-pharmacological treatments include stimulus control, sleep restriction, sleep hygiene education and Cognitive Behavioural Therapy (CBT), which aims to alter the psychological aspects of sleep disturbance. Previous research has shown that CBT is superior in treating insomnia compared to the other aforementioned treatments (Wang, Wang, & Tsai, 2005). This is important information for clinicians who should consider educating patients about CBT as validated treatment option for sleep disturbance.

Fifth, depression was linked to sleep disturbance, and impacted on the relationship between sleep-wake patterns and cognition, such that there was a distinct profile of sleep-related cognitive deficits in older adults with, versus without, current MDD. Depression is one of the leading causes of disability worldwide in all age groups, and its
impact on physical and cognitive health has long been demonstrated (Reddy, 2010). Here, this thesis was able to add to the existing literature by showing that depression also impacts on sleep and on its relationship with cognitive functioning. Thus, the management of depression and sleep is key for maintaining good health in ageing and findings highlight the necessity of considering depression in empirical and clinical studies of sleep and cognition.

**Methodological implications**

The use of multiple measures to assess sleep and depression was a significant strength of this thesis, providing valuable information for researchers and clinicians.

*Sleep:* A combination of self-reports (questionnaires), actigraphy, and PSG was used to assess sleep quality, sleep-wake patterns and sleep staging in this thesis. Advantages of **self-reports** include the fact that they are easy to use, inexpensive, convenient and non-obtrusive, and are ideal for longitudinal data collection (Cohen, 1992). However, they are reliant on the respondent’s perceptions, which can differ from objective measures of sleep and may be influenced by factors such as depression. In addition to possible reporting bias, self-reports commonly yield missing data and may not provide a complete picture of sleep quality given that they typically assess sleep in previous weeks (Cohen, 1992). In this thesis, the PSQI was used to assess sleep quality. Results highlight the benefit of using factor scores over the global PSQI sleep quality score, which allows for investigation of distinct dimensions of subjective sleep quality (Cole et al., 2006). Overall, the use of self-reports provided valuable information about specific components of subjective sleep quality and their association with age and cognition. **Actigraphy monitoring** was used to assess sleep-wake patterns in this thesis and has several benefits – it is easy to use, less expensive and time-consuming than PSG, good for large population groups, and the actiwatch can be worn for extended periods of time, providing more reliable data about sleep-wake patterns than PSG, which is usually performed for only one or two nights (Blackwell et al., 2008). However, limitations include the fact that sleep-wake patterns are inferred from movement, which may not be fully accurate (Sadeh, 2011). This supports the use of sleep diaries as an adjunct to actigraphy, as in the current thesis. In addition, data management and interpretation are time-intensive and expensive (Beck et al., 2004). Finally, while actigraphy has reasonable validity and reliability in assessing sleep-wake
patterns in normal individuals, the validity of actigraphy in older adults needs further exploration (Sadeh, 2011). Therefore, given the limitations of actigraphy, I recommend that researchers and clinicians use complementary measures of sleep assessment (e.g., self-reports, sleep diaries). **Polysomnography** is the ‘gold standard’ of sleep assessment and is considered the most accurate measure of sleep, providing information about specific sleep stages (Beck et al., 2004). This was of particular use in the current thesis, which examined the relationship of specific sleep stages to cognitive performance. Limitations of PSG include the fact that it is demanding in terms of cost and time, is invasive and can be disruptive to sleep (Blackwell et al., 2008). Overall, the combination of the three sleep methods (self-reports, actigraphy, PSG) allowed me to assess specific dimensions of sleep and how they impacted on cognition, thus addressing a primary aim of this thesis. Results showed that specific aspects of sleep, assessed with different methods, impact on cognition in quite diverse ways. For example, while self-reported sleep was linked to cognition largely as a result of effects of poor sleep on attention (Chapter 3), objective measures of sleep were linked to cognition over and above effects on attention (Chapter 5). Furthermore, sleep-wake disturbances (assessed with actigraphy) were linked to vigilance, delayed recall and working memory, whereas sleep architecture (assessed with PSG) largely did not affect cognition (Chapter 5). Finally, while depression affected the relationship between actigraphic sleep quality and cognition, this was not the case for sleep architecture. Overall, results highlight the multifaceted nature of sleep, which all researchers and clinicians should recognise when selecting methods of sleep assessment.

**Depression**: A combination of self-reports (DASS-21, PHQ-9), clinical interviews (the MINI), and morning cortisol levels were used to assess depression in this thesis. **Self-reports** have the benefit of being easy to administer. For example, the DASS-21 takes no time to complete, has good validity and provides a severity rating scale (Crawford & Henry, 2005). Similarly, the PHQ-9 provides a severity rating, is well validated and maps onto the DSM-IV criteria for depression (Kroenke et al., 2001). However, self-reports of depression rely on patient insight and are not sensitive to fluctuations in depressive symptoms over time. The MINI – the **clinical interview** used in this thesis – is easy to administer and is fully validated (Sheehan et al., 1998). However, the restriction to yes/no answers may miss symptoms and provides no information about severity level. Hence, the combination of clinical interviews and self-reports is recommended to provide a more complete assessment of depressive symptoms.
assessed morning cortisol levels as a potential biological index of depression. Benefits include the fact that they are based on physiological factors, which are not reliant on the participant’s perceptions, and that saliva samples are relatively easy to collect. Cortisol measures were of benefit in this thesis, which investigated sleep in older adults with current and past depression, because, unlike self-reports, cortisol has been shown to remain elevated in remitted individuals (e.g., Beluche et al., 2009). In addition, cortisol assessment offers advantages over a clinical diagnosis because it assesses HPA axis activity as a continuous variable. However, it must be acknowledged that cortisol is not an untainted measure of depression and has been linked to external factors such as stress and alcohol use (e.g., Badrick et al., 2008). Current results suggest that the relationship of sleep and depression is dependent on how both are assessed. For example, while depression assessed using self-reports (PHQ-9) and cortisol measures were both linked to greater sleep architecture abnormalities (more %REM and less %NREM sleep) in older adults, cortisol was also linked to sleep-wake pattern disturbance (shorter actigraphic total sleep time and poorer sleep efficiency), which was not true of self-reports (Chapter 4).

Overall, results of this thesis highlight the importance of selecting measures based on current theoretical understanding of the links between sleep, depression and cognition. Given the multifaceted nature of sleep, researchers and clinicians should consider using a combination of sleep assessments where possible. Current results also draw attention to the fact that different tools to assess depression may yield different results. The use of cortisol measures in this thesis adds to a body of literature attempting to find a biological measure of depression.

**Limitations and suggestions for future research**

One limitation of this thesis, common to all cross-sectional research, is that studies of this nature do not allow explicit testing of the direction of relationships. In order to discuss the direction of effects with any certainty, intervention or sleep deprivation and restriction paradigms would be needed. Throughout this thesis there are references to the impact of sleep on depression, despite evidence of a bidirectional relationship (e.g., Alvaro et al., 2013). Similarly, while the majority of the literature is in support of sleep impacting on cognition, some research suggests that cognitive activity can affect sleep. For example, one study found that cognitive training improved sleep quality (sleep
latency and sleep efficiency) in older adults (Haimov & Shatil, 2013), and another found increases in SWS after learning (Fogel & Smith, 2006). However, cross-sectional research has significant benefits in that it is less demanding than longitudinal studies and provides valuable information about associations between variables. Arguably, it would also be unethical to conduct an intervention study without first establishing the potential for such intervention to produce significant benefit either to participants or to society more generally.

A pertinent issue to part two of this thesis (Chapters 4 and 5) was sample size constraints, due to difficulty with recruitment of older adults with and without depression. First, participants took part in demanding methodologies, which included two overnight sleep studies, seven days of actigraphy monitoring and completing sleep diaries, and extensive cognitive testing, over the course of a week. Given the demands on both the researcher and participants, small samples in PSG research are not uncommon (e.g., Backhaus et al., 2007). Second, recruitment of the depressed sample was also difficult due to common comorbidities, which needed to be excluded, such as bipolar depression and dementia. Furthermore, individuals with depression commonly experience decreased motivation (Fiske et al., 2009), and are therefore less likely to take part in research.

A significant strength of this study was the use of portable-PSG, which allowed participants to sleep in their own homes rather than a sleep clinic. This likely increased the chance of interested participants taking part in the study as many older adults are unable or unwilling to leave their homes for laboratory-based studies. Nevertheless, there were issues of power, which may have affected the validity of results. There is, therefore, a need for future studies using larger samples of older adults with and without depression or the meta-analysis of multiple, smaller studies such as this one.

Recent studies suggest that neurophysiological events associated with specific sleep stages (e.g., REM sleep theta and sleep spindles) may play an important role in cognitive functioning (e.g., Cox et al., 2012; Fogel & Smith, 2006). This was beyond the scope of the current study, however future studies should consider the possibility of spectral and spindle analysis of sleep in addition to traditional sleep staging.
A further issue common to investigations of this nature is the confounding effect of medications. Analyses were careful to examine the data and compare the performance of individuals who were taking, versus not taking, medications (e.g., antidepressants), but this is a common and unavoidable limitation of human clinical studies. It is also unethical to ask participants to refrain from using medications, which have been prescribed to maintain good health.

While this thesis had significant strengths regarding the assessment of depression by using several metrics (clinician diagnosis, self-reports, clinical interviews and cortisol levels), future studies may wish to distinguish between subtypes of depression, including endogenous versus exogenous depression; early versus late-life depression; and recurrent versus single episode depression. However, due to difficulty in recruiting older depressed samples, this would be challenging. Future studies investigating the impact of a past history of depression on sleep should also implement criteria relating to the time since last depressive episode.

While it is important to acknowledge that cortisol is not a ‘pure’ measure of depression as it is linked to other factors such as alcohol consumption (Badrick et al., 2008), the inclusion of cortisol as a potential biological index of depression was a strength of this study. An avenue for future research, which was beyond the scope of this study, is to explore cortisol as a potential mechanism of the relationship between sleep problems and cognitive deficits. Changes in the hypothalamic-pituitary adrenal axis (HPA) have been implicated in age-related declines in sleep quality, which in turn, may be responsible for cognitive decline (Buckley & Schatzberg, 2005).

**Overarching conclusions**

In summary, this thesis offers valuable and novel contributions to the study of sleep and cognition in older adults, and how depression affects this relationship. Results indicate that sleep affects cognitive performance in older adults, and that depression plays an important role in this relationship. These are exciting findings as they imply that some of what we regard as age-related cognitive impairment might, at least in part, be due to poor sleep. Bearing in mind that longitudinal data was not collected, nor was there a younger sample for comparison, these findings provide tentative evidence that sleep has the potential to explain the cognitive changes widely reported in older adults. This is
important information for clinicians given that sleep problems are potentially modifiable risk factors of cognitive impairment (Ferini-Strambi et al., 2003). It is hoped that future research can expand on the results of this thesis by examining the longitudinal association of multiple aspects of sleep and cognition, and by exploring the impact of sleep interventions on cognition, in older adults with and without depression.
References


sustained wakefulness and a blood alcohol concentration of 0.05%. *Journal of Sleep Research, 12*(4), 265-274.


Ohayon, M. M., Zulley, J., Guilleminault, C., Smirne, S., & Priest, R. G. (2001). How age and daytime activities are related to insomnia in the general population:


Schwartz, D. J., & Karatinos, G. (2007). For individuals with obstructive sleep apnea, institution of CPAP therapy is associated with an amelioration of symptoms of depression which is sustained long term. *Journal of clinical sleep medicine : JCSM : official publication of the American Academy of Sleep Medicine, 3*(6), 631-635.


neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep*, 26(2), 117-126.


### Appendix Table 1. *Chapter 1.4.2 Sleep and depression in older adults – study details*

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size</th>
<th>Methods – Sleep</th>
<th>Methods – Depression</th>
<th>(Age, % female)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cross-sectional self reports</strong></td>
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<tr>
<td>Almeida et al., 2005</td>
<td>N = 1029 General Practice patients</td>
<td>CAMDEX-R: difficulty falling asleep; restlessness/wakefulness during the night; waking early &amp; being unable to fall back asleep</td>
<td>CES-D; ≥ 16 indicates clinical levels of depressive symptoms</td>
<td>60+ years 57.2% female</td>
</tr>
<tr>
<td>Foley et al., 1995</td>
<td>N = 9282 older adults from 3 communities</td>
<td>Insomnia interview: difficulty falling asleep, waking during the night, waking too early &amp; not being able to fall asleep again, sleepiness, feeling unrested when waking (summary sleep disturbance score)</td>
<td>CES-D</td>
<td>65+ years 61.2% female</td>
</tr>
<tr>
<td>Foley et al., 2004</td>
<td>N = 1506 community older adults as part of National Sleep Foundation survey</td>
<td>Insomnia interview – bed/wake times, sleep duration (&lt; 6h = short sleepers), difficulty falling asleep, waking during the night, waking too early, falling back to sleep, waking feeling un-refreshed Other – snoring, pauses in breathing, unpleasant feelings in legs, sleepiness, quality of sleep, presence of sleep disorder</td>
<td>Physician diagnosis of depression</td>
<td>55-84 years 57.9% female</td>
</tr>
<tr>
<td>Ito et al., 2000</td>
<td>N = 518 community older adults</td>
<td>Questionnaire: sleep duration, frequency of waking during the night, feeling unrefreshed in the morning (any item)</td>
<td>GDS (short-form) ≥6 indicates clinical levels of depressive symptoms</td>
<td>65 years 50.8% female</td>
</tr>
<tr>
<td>Maglione et al., 2012</td>
<td>N = 3045 community older women</td>
<td>PSQI ESS</td>
<td>GDS</td>
<td>70+ years</td>
</tr>
<tr>
<td>Newman et al., 1997</td>
<td>N = 5201 community older adults</td>
<td>Questionnaire: symptoms of difficulty initiating &amp; maintaining sleep, including difficulty falling asleep, frequent awakenings, waking too early, sleepiness</td>
<td>CES-D</td>
<td>65+ years 57% female</td>
</tr>
<tr>
<td>Paudel et al., 2008</td>
<td>N = 3051 community older men</td>
<td>PSQI ESS</td>
<td>GDS</td>
<td>67+ years</td>
</tr>
<tr>
<td>Study</td>
<td>Sample size</td>
<td>Methods – Sleep</td>
<td>Methods – Depression</td>
<td>(Age, % female)</td>
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<tr>
<td>Schechtman et al., 1997</td>
<td>N = 485 community older adults</td>
<td>Questionnaire including items about trouble falling asleep, waking during the night, feeling rested in the morning (sleep disturbance summary score)</td>
<td>CES-D</td>
<td>68+ years 61.9% female</td>
</tr>
<tr>
<td>van den Berg et al., 2009</td>
<td>N = 5019 community older adults</td>
<td>PSQI: Time in bed, total sleep time, sleep latency, Global score</td>
<td>Psychiatric interview (the Schedules for Clinical Assessment in Neuropsychiatry for depressive disorders) based on DSM-IV for major, &amp;minor depressive disorder, dysthymic disorder</td>
<td>58-100 years 56.7% female</td>
</tr>
<tr>
<td>Wu et al., 2012</td>
<td>N = 100 older community adults</td>
<td>PSQI Global score</td>
<td>TDQ based on DSM-III-R</td>
<td>65+ years 55% female</td>
</tr>
<tr>
<td>Zimmerman et al., 2013</td>
<td>N = 702 community older adults</td>
<td>Global sleep problem index &amp; difficulties with sleep onset/maintenance assessed using 2 questions from the MOS-SS questionnaire</td>
<td>GDS; ≥5 indicates clinically significant depressive symptoms</td>
<td>70+ years 60.8% female</td>
</tr>
<tr>
<td>Study</td>
<td>Sample size</td>
<td>Methods – Sleep</td>
<td>Methods – Depression</td>
<td>(Age, % female)</td>
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<tr>
<td>Maglione et al., 2012</td>
<td>N = 3045 community older women</td>
<td>Actigraphy (M = 4.1 ± 0.8 days): TST, sleep efficiency, sleep latency, WASO, no. long-wake episodes, no. nap episodes (no. inactive episodes between wake time &amp; sleep onset &gt; 5 minutes)</td>
<td>GDS</td>
<td>70+ years 100% female</td>
</tr>
<tr>
<td>Naismith et al., 2011</td>
<td>N = 44 adults with a lifetime history of MDD, 22 controls</td>
<td>Actigraphy (2 weeks): sleep latency, no. &amp; duration of nocturnal awakenings, sleep efficiency</td>
<td>Affective component of the Structured Clinical Interview for DSM-IV-R Disorders GDS</td>
<td>46-86 years % female: patients (50%); controls (36.4%)</td>
</tr>
<tr>
<td>Paudel et al., 2008</td>
<td>N = 3051 community older men</td>
<td>Actigraphy (M = 5.2 nights): Sleep efficiency, TST, sleep latency, WASO, long wake episodes</td>
<td>GDS</td>
<td>67+ years 0% female</td>
</tr>
<tr>
<td>Robillard et al., 2014</td>
<td>N = 238 individuals with a history of a diagnosed mood disorder</td>
<td>Actigraphy: (5-22 days): sleep onset/offset, time in bed, TST, WASO, sleep efficiency, circadian profile</td>
<td>HDRS</td>
<td>12-90 years. Age groups: 12-19, 20-39, 40-59, 60+ % Females: 12-19: 78.7% 20-39: 58.9% 40-59: 63.3% 60+: 55.8%</td>
</tr>
<tr>
<td>Study</td>
<td>Sample size</td>
<td>Methods – Sleep</td>
<td>Methods – Depression</td>
<td>(Age, % female)</td>
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<tr>
<td><strong>PSG studies</strong></td>
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<tr>
<td>Gillin et al., 1981</td>
<td>N = 69 unmedicated inpatients with primary affective illness, 36 controls</td>
<td>3-6 nights of PSG</td>
<td>Diagnosis of MDD according to Research Diagnostic Criteria</td>
<td>15-64 years, % female: patients(72.5%); controls (58.3)%</td>
</tr>
<tr>
<td>Kupfer et al., 1989</td>
<td>N = 18 older adults (6 delusional depressive inpatients, 6 non-delusional depressive inpatients, 6 controls)</td>
<td>2 nights of PSG (second night analysed)</td>
<td>SADS-L HRSD &gt; 30 Diagnosis of delusional &amp; non-delusional depression according to Research Diagnostic Criteria</td>
<td>50+ years, 50% females in each group</td>
</tr>
<tr>
<td>Lauer et al., 1991</td>
<td>N = 125 adults (74 MDD, 51 healthy controls)</td>
<td>≥ 3 nights of PSG</td>
<td>Diagnosis of MDD according to Research Diagnostic Criteria</td>
<td>18-65 years, % female: patient (67.6%); controls (49%)</td>
</tr>
<tr>
<td>Reynolds III et al., 1985</td>
<td>N = 75 older adults (25 depression, 25 dementia, 25 controls)</td>
<td>3 consecutive nights of PSG</td>
<td>SADS-L for recurrent MDD; HDRS &gt; 15</td>
<td>49-85 years, % female: depressed (84%); dementia (72%); controls (68%)</td>
</tr>
<tr>
<td>Reynolds III et al., 1991</td>
<td>N = 60 older adults (30 recurrent MDD, 30 controls)</td>
<td>3 consecutive nights of PSG (at baseline and at follow-up)</td>
<td>Recurrent MDD according to SADS-L; HDRS ≥ 17</td>
<td>60-80 years, % female: 70% in each group</td>
</tr>
<tr>
<td>Smagula et al., 2013</td>
<td>N = 2853 older men</td>
<td>1 night of home-PSG</td>
<td>GDS; &gt; 6 indicates clinically significant depression</td>
<td>65+ years</td>
</tr>
</tbody>
</table>
# Study Sample size Methods – Sleep Methods – Depression (Age, % female)

## Longitudinal studies

### Depression predicting sleep problems

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size</th>
<th>Methods – Sleep</th>
<th>Methods – Depression</th>
<th>(Age, % female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foley et al., 1999</td>
<td>N = 9289 community adults</td>
<td>Interview for insomnia</td>
<td>CES-D</td>
<td>65+ years, 69.8% female at follow-up</td>
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<td></td>
<td>N = 6899 at follow-up 3 years later</td>
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<tr>
<td>Kim et al., 2009</td>
<td>N = 1204 community older adults at baseline</td>
<td>Insomnia = difficulty initiating or maintaining sleep ≥3 nights per week in the last month</td>
<td>GMS B3</td>
<td>65+ years, 58% female at follow-up</td>
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<td></td>
<td>N = 909 at follow-up 2 years later</td>
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</table>

### Sleep problems predicting depression

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size</th>
<th>Methods – Sleep</th>
<th>Methods – Depression</th>
<th>(Age, % female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almeida et al., 2011</td>
<td>N = 5127 community older men at follow-up (Mean follow-up time = 6 years)</td>
<td>Health questionnaire assessing difficulty falling asleep, remaining awake, early morning awakening</td>
<td>Recorded diagnosis of depressive episode (single or recurrent) &amp; dysthymia (ICD-10)</td>
<td>70-90 years</td>
</tr>
<tr>
<td>Cho et al., 2008</td>
<td>N = 351 community older adults (145 = a history of depression in remission, 206 = no history of depression) N = 329 at 2 year follow-up</td>
<td>PSQI</td>
<td>BDI</td>
<td>60+ years, % female: history of depression (59.3%); no history of depression (51%)</td>
</tr>
<tr>
<td>Livingston et al., 1993</td>
<td>N = 705 community older adults at baseline N = 524 at follow-up 2 years later</td>
<td>The short-care instrument for assessment of depression, dementia &amp; disability – Sleep items</td>
<td>The short-care instrument for assessment of depression, dementia &amp; disability</td>
<td>65+ years 66% female at follow-up</td>
</tr>
<tr>
<td>Study</td>
<td>Sample size</td>
<td>Methods – Sleep</td>
<td>Methods – Depression</td>
<td>(Age, % female)</td>
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<tr>
<td>Martin et al., 2010</td>
<td>N = 121 older adults in assisted living facilities N = 115 at follow-up 6 months later</td>
<td>PSQI &gt; 5 Actigraphy confirmed poor sleep quality for 3 consecutive nights</td>
<td>GDS-5 (5 items) &gt; 2</td>
<td>65+ years, 86% female</td>
</tr>
<tr>
<td>Perlis et al., 2006</td>
<td>N = 247 older adults (189 with no prior history of depression) N = 147 at follow-up 1 year later</td>
<td>HRSD sleep items (Q 4–6): type &amp; severity of insomnia in the past week</td>
<td>Structured Clinical Interview for the DSM–III–R HRSD</td>
<td>60+ years, 57% female at follow-up</td>
</tr>
<tr>
<td>Roberts et al., 2000</td>
<td>N = 2370 community older adults (at baseline &amp; follow-up 1 year later)</td>
<td>2 sleep-related items of DSM-12D (insomnia, hypersomnia)</td>
<td>DSM-12D</td>
<td>50-95 years, 56.4% female at follow-up</td>
</tr>
</tbody>
</table>

**Abbreviations**: BDI: Beck Depression Inventory; CAMDEX-R = Cambridge Examination for Mental Health Disorders of the Elderly Revised; CES-D: Centre for Epidemiologic Studies Depression; DSM-III-R Diagnostic and Statistical Manual of Mental Disorders, third edition revised, DSM-IV: fourth edition, DSM-IV-R: fourth edition revised, DSM-12D: 12-item scale for depression; ESS: Epworth Sleepiness Scale; GDS: Geriatric Depression Scale; GMS B3: Geriatric Mental State Schedule; HRSD: Hamilton Rating Scale for Depression (also known as HAM-D and HDRS); ICD-10: International Classification of Diseases; MDD: Major Depressive Disorder; MOS-SS: Medical Outcomes Study Sleep Scale; PSG: Polysomnography; PSQI: Pittsburgh Sleep Quality Index; SADS-L: Schedule for Affective Disorders and Schizophrenia, Lifetime version; TDQ: Taiwanese Depression Questionna.
Appendix Table 2. List of CogState primary outcome measures

**COGSTATE COMPOSITE COGNITIVE SCORES**

Composite scores as a measure of overall cognitive performance, and they are calculated from all tasks in the battery.

In order to combine the task scores, each score at each assessment must be standardised (z-scores). This is done by standardising data against the baseline values from that sample (which acts as the control data for the study group) or from normative data.

Primary outcome measures for the CogState battery and the Completion criteria

<table>
<thead>
<tr>
<th>Task name (Abbreviation in Data Extract)</th>
<th>Variable code</th>
<th>Unit of measurement</th>
<th>Completion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>International Shopping List Task (ISLT)</td>
<td>cor</td>
<td>Number of correct responses</td>
<td>All 12 words were presented on all three trials, that is: sti =&gt; 36</td>
</tr>
<tr>
<td>Detection Task (DET)</td>
<td>lmn</td>
<td>Log_{10} milliseconds</td>
<td>At least 27 responses made (presnt=&gt;27)</td>
</tr>
<tr>
<td>Identification Task (IDN)</td>
<td>lmn</td>
<td>Log_{10} milliseconds</td>
<td>At least 23 responses made (presnt=&gt;23)</td>
</tr>
<tr>
<td>One Back Memory Task (OBN)</td>
<td>lmn</td>
<td>Log_{10} milliseconds</td>
<td>At least 23 responses made (presnt=&gt;24)</td>
</tr>
<tr>
<td>Set Shifting (SETS)</td>
<td>err</td>
<td>Total number of errors</td>
<td>At least 90 responses made (sti=&gt;90)</td>
</tr>
<tr>
<td>Social-Emotional Cognition Task (SECT)</td>
<td>acc</td>
<td>Arcsine proportion correct</td>
<td>At least 36 responses made (presnt=&gt;36)</td>
</tr>
<tr>
<td>Continuous Paired Associate Learning (CPAL)</td>
<td>err</td>
<td>Total number of errors</td>
<td>All 8 targets correctly identified over the 7 learning trials (sti=&gt;56)</td>
</tr>
<tr>
<td>International Shopping List Task – Recall (ISLR)</td>
<td>cor</td>
<td>Number of correct responses</td>
<td>Task was attempted sti =12</td>
</tr>
</tbody>
</table>
Journal article removed due to copyright restrictions. The article is available to subscribers via this link: http://dx.doi.org/10.1080/15402002.2013.801343

Sleep and Aging: Examining the Effect of Psychological Symptoms and Risk of Sleep-Disordered Breathing
Alix Mellor, Flavie Waters, Michelle Olaithe, Helen McGowan, Romola S. Bucks
Behavioral Sleep Medicine
Vol. 12, Iss. 3, 2014
Appendix: Advertisement for volunteers (all ages)

Volunteers required for sleep and thinking skills questionnaire study!

Research tells us that sleep difficulties can cause distress and reduced quality of life, and affect thinking skills.

Researchers from the University of Western Australia are conducting a questionnaire study to explore the effects of sleep on memory and thinking skills. Our study aims to increase understanding about how sleep can affect adults of different ages (from 18 to 100+!).

Participation is entirely voluntary. Questionnaires are available online and in hardcopy form and should take approximately 45 minutes to complete.

You do not have to have sleep difficulties to participate in this study. In fact, we would like to compare good and poor sleepers. If you are aged over 18 and would like to participate in this study, please either:

1. Follow this link to the questionnaires online.

   http://tinyurl.com/2cmuqub

   OR

2. Contact for further questions or to request a questionnaire pack by post:

Alix Mellor (mellora@meddent.uwa.edu.au, 08 9347 6404 – answering machine)

All information given and data collected will be kept completely confidential.

Thank you for considering this request!

Alix
Appendix: Advertisement for volunteers (older adult group)

This was the initial phase of the recruitment process. Once the questionnaire was completed, respondents had the option to give their details in order to be contacted for the subsequent, objective study.

Volunteers required for sleep and thinking skills questionnaire study!

Research tells us that sleep difficulties can cause distress and reduced quality of life, and affect thinking skills.

Researchers from the University of Western Australia are conducting a questionnaire study to explore the effects of sleep on memory and thinking skills. Our study aims to increase understanding about how sleep can affect older adults (50+ years).

Participation is entirely voluntary. Questionnaires are available online and in hardcopy form and should take approximately 45 minutes to complete.

You do not have to have sleep difficulties to participate in this study. In fact, we would like to compare good and poor sleepers. If you are aged over 50 and would like to participate in this study, please either:

3. Follow this link to the questionnaires online.

   http://tinyurl.com/2cmuqub

   OR

4. Contact for further questions or to request a questionnaire pack by post:

   Alix Mellor (mellora@meddent.uwa.edu.au, 08 9347 6404 – answering machine)

All information given and data collected will be kept completely confidential.

Thank you for considering this request!

Alix
Appendix: Additional analyses (referring to Chapter 4, page 94)

$t$ test for REM latency – people taking antidepressants versus not taking antidepressants in the Past group

$t(12) = -2.01, p = .034$ (one-tailed).

Descriptive statistics

Not taking antidepressants: $N = 7$, $M = 2.01$
Taking antidepressants: $N = 7$, $M = 2.24$
Another simple transformation is to the mean center predictors that are being multiplied together prior to constructing the product. Such mean centering is often advocated when estimating models such as Equation 2, although the advantages of doing so are generally overstated (see, e.g., Cronbach, 1987; Kromrey & Foster-Johnson, 1998). To mean center a variable, the sample mean is subtracted from each measurement. Had this been done prior to calculating the product of ideology and perceived reality, the resulting model would be as presented in the model 5 column of Table 1. The coefficient for perceived reality is positive and statistically significant, $b_1 = 0.099$, $p = .034$ (see Figure 2 for a visual depiction). This represents the effect of perceived realism estimated at the sample mean political ideology (as a value of zero on political ideology corresponds to the sample mean after mean centering). If $M$ is a discrete variable, the regression coefficient for $X$ after mean centering can be interpreted as the weighted average conditional effect of $X$ across the values of $M$, with the weighting based on the relative frequency of the values of $M$. This is not the same thing as a main effect in ANOVA, which is an unweighted average conditional effect.

Another approach to estimating models with interactions is to standardize the variables being used to produce the interaction term prior to multiplying them together. This is similar to mean centering, in that a score of zero on variables that are standardized corresponds to the sample mean when unstandardized. However, the unit of measurement changes from the original metric to standard deviations. That is, a score of 1 corresponds to a response one standard deviation above the mean, and two cases that differ by 1 differ by a standard deviation rather than one point on the original metric. The final column in Table 1 (model 6) represents the coefficients from the model after first standardizing ideology and perceived reality prior to computing their product. Notice that the $t$ statistic and $p$-value for perceived reality have not changed compared to mean centering. This is because the hypothesis test still tests, as it did in the model in column 5, the null hypothesis that the conditional association between perceived reality and support for social spending is zero at the sample mean ideology. But the size of the coefficient for perceived reality has changed, reflecting the fact that two cases that differ by one unit on the original metric of measurement will differ by some different amount when the metric is changed to standard deviations. In this case, $b_1$ is smaller compared to when mean centering is used because the standard deviation for perceived reality is smaller than one.

Two additional points need to be made. First, one might argue that if researchers followed the practice of reporting standardized coefficients or expressing effects in terms of explained variance rather than reporting unstandardized regression coefficients, the differences reported above would vanish. That is, given that
researchers’ decisions about measurement are always somewhat arbitrary, would standardizing variables prior to analysis not make the effect of such arbitrary decisions on statistical results go away? Unfortunately, this is simply not true, as is illustrated in Table 1. The standardized regression coefficients and variance explained for perceived reality also change when one of the linear transformations described here is applied to the data. The unit of measurement does not change the basic fact that any measure of a variable’s effect, in the presence of an interaction involving that variable as in Equation 2, is conditional on the other variable in the interaction equaling zero. Furthermore, the outcome of a test of the null hypothesis that a variable’s effect is equal to zero is mathematically equivalent regardless of whether the effect is expressed as an unstandardized regression coefficient, a standardized coefficient, a semipartial or partial correlation, or an increase in $R^2$ when a variable is added to the model.

Second, Table 1 shows that the arbitrary choices of scaling described above have no effect on the test for the interaction. Whether one mean centers, standardizes, or adds or subtracts a constant from one or both of the variables involved in the interaction, the result of the null hypothesis test of no interaction (i.e., its $t$ statistic and $p$-value) in a model such as Equation 2 is unchanged, as are measures of effect size for the interaction. This might come as a surprise to those who advocate centering or standardizing in regression models on the grounds that it reduces multicollinearity and the problems multicollinearity produces. Although the regression coefficients, $t$ statistics, $p$-values, and measures of effect size are changed for those variables involved in the interaction, this has nothing to do with reducing multicollinearity. These differences are attributable to the effects of rescaling a variable in such a way that the “zero” point is changed vis-à-vis the original scale of measurement. The need to center (or standardize) $X$ and $M$ in a regression model including $XM$ as a predictor is a myth that doggedly persists in spite of having been repeatedly debunked (Cronbach, 1987; Echambi & Hess, 2007; Friedrich, 1982; Hayes, 2005, pp. 465–467; Kromrey & Foster-Johnson, 1998; Shieh, 2011).

That said, there are some advantages to mean centering or standardizing. First, mean centering $X$ and $M$ prior to computation of their product produces an estimate of the effect of $X$ (or $M$) on $Y$ that is conditioned on a value of $M$ (or $X$) that will always be within the range of the data (i.e., at the sample mean), unlike when mean centering is not done. Thus, by mean centering, the consequences of the kind of misinterpretations we describe here will be much less severe when they occur. Second, in complex models with several interactions involving the same variable, multicollinearity can in rare circumstances produce computational difficulties in some statistical programs. Mean centering $X$ and $M$ prior to computing their product does indeed reduce this “nonessential” multicollinearity and thereby eases the computation problems that sometimes though rarely arise. In the majority of applications of moderated multiple regression in the field of communication, however, mean centering or standardization is a choice one can make, to do or not to do, rather than a requirement.
CONCLUSION

In this editorial primer, we have attempted to clarify the interpretation of parameter estimates and hypothesis tests from a regression model with interactions. When $X$, $M$, and $XM$ all coexist in a regression model, the coefficients and tests of significance for $X$ and $M$ are estimated and tests of *conditional* effects or *simple* effects, not main effects as they are in ANOVA and sometimes interpreted as in the communication literature. They estimate the effect of one variable conditioned on the other equaling zero. They cannot be interpreted as partial regression coefficients in a model without an interaction, nor are they equivalent to main effects in ANOVA. A greater understanding of these subtle but very important principles will reduce the number of claims in the communication literature that are not substantiated by statistical evidence or that are based on statistical information from a linear model that is not pertinent to the claim being made.

We recommend that dichotomous variables not be standardized under any circumstances, for the regression coefficient for a standardized dichotomy has no sensible interpretation and is determined by the distribution of the sample across the two groups coded with the dichotomous variable (see e.g., Cohen et al., 2003, p. 316). This applies to regression models without interactions, and to the interpretation of standardized regression coefficients for dichotomous predictors as well.
Appendix: List of standardised sleep measures used in the questionnaire booklet (see next page)

Your sleep habits: Pittsburgh Sleep Quality Index (PSQI) p19
Your sleep and breathing: Berlin Questionnaire (BQ) p12
Sleepiness: Epworth Sleepiness Scale (ESS) p29

Your views about your mood:

  Depression Anxiety Stress Scale (DASS-21) p38
  Patient Health Questionnaire (PHQ-9) p43

Your behaviour: Frontal Systems Behavior Scale (FrSBe) p47

How you feel about your memory:
Multifactorial Memory Questionnaire (MMQ) p54

Difficulties in everyday life: Cognitive Difficulties Scale (CDS) p60
Appendix: Questionnaire given to participants

A STUDY ON HOW SLEEP AFFECTS MEMORY AND THINKING SKILLS

WE NEED YOUR HELP!

Research tells us that sleep difficulties can cause distress and reduced quality of life, and can affect thinking skills.

This questionnaire is part of a research study being conducted which aims to increase understanding of the ways in which sleep affects memory and thinking skills.

Following, is a series of questions that ask about:
- your background;
- your health;
- your sleep;
- your memory and thinking skills.

The information collected from this questionnaire will be used for research purposes only. You do not need to give your name anywhere in this questionnaire, unless you wish to at the end.

All information provided will be kept completely confidential.

This questionnaire is voluntary. If for any reason you do not wish to complete it you do not have to do so. The questionnaire will take approximately 45 minutes to complete.

We realise that there are quite a few questions and that some are rather personal. However, each question is important and your answers will help us better to understand how sleep affects everyday functioning and mood.

Please answer all questions. If you are uncertain, please write down your best guess. Try not to spend too long thinking about your answers. Your first response is usually the best one. Please only make one selection per question.

Please pay particular attention to the rating scales as they change from question to question. Doing the survey straight through in one go is probably best.

At the end of the questionnaire we would be grateful if you would indicate whether or not we can contact you again about further research in this area. Agreeing to be contacted does not commit you to taking part in further study. Your contact details will be stored separately to your answers to this survey.

Thank you very much for taking the time to help us with this important research.

Chief Investigator: Alix Mellor Phone number: (08) 9347 6404 email: mellora@meddent.uwa.edu.au

Supervisors:
A/Prof Flavie Waters Phone number: (08) 9347 6429/6650
Dr Romola Bucks Phone number: (08) 6488 3232

What is your country of residence? ____________________________________________

Which town do you live in? __________________________________________________

Please include your postcode here:
(UK respondents need not give their postcode unless they wish to)

A STUDY ON HOW SLEEP AFFECTS MEMORY AND THINKING SKILLS (MMP13/15)
YOUR BACKGROUND

We would like to know some details about your personal history and background.

1. Age (years) ________________

2. Date of birth: DD/MM/YYYY (day/month/year). ___ / ___ / ______

3. Were you born in Australia? Yes [ ] No [ ]
   If no, in which country were you born? __________________________

4. Where were your parents born? If you do not know, please write ‘unknown’.
   In which country was your mother born? _________________________
   In which country was your father born? _________________________

5. What language do you speak at home? __________________________

6. How familiar are you with the English language? (Tick one box)
   [ ] I speak English very well
   [ ] I speak English, but occasionally have some difficulty
   [ ] I don’t speak English well, but manage to communicate
   [ ] I understand very little or no English and require an interpreter

7. Gender: Male [ ] Female [ ]

8. Marital status: (Tick one box)
   [ ] Single
   [ ] Married
   [ ] De facto/co-habiting
   [ ] Divorced/separated
   [ ] Widowed
   [ ] Decline to say

9. Your measurements - please complete the following:
   Your Height: __________________________
   Your Weight: __________________________
10. **EDUCATION:**

   What is the *highest* level of education you have achieved?
   - [ ] Primary school
   - [ ] Secondary school
   - [ ] TAFE or apprenticeship
   - [ ] Undergraduate university degree
   - [ ] Postgraduate university degree

11. **OCCUPATION:**

   What is your occupation?
   (If retired, please state occupation before retirement. If housewife/househusband, please state your partner’s occupation)

12. What is your current employment status?
   - [ ] Full-time work
   - [ ] Part-time work
   - [ ] Unemployed
   - [ ] Retired
   - [ ] On a pension

13. Do you work early or late shifts? (NOT night shift)
   - [ ] Yes
   - [ ] No

   If you regularly work early or late shifts, please describe the work patterns and hours:
   

14. Do you work nights? (i.e., night shifts or shift work overnight)
   - [ ] Yes
   - [ ] No

   If you regularly work night shifts, please describe the work patterns and hours:
   

15. **HEALTH:**

Are you currently pregnant? ☐ Yes ☐ No

16. Do you have?

- Hearing problems? ☐ Yes ☐ No
- Visual impairment? ☐ Yes ☐ No
- Mobility problems? ☐ Yes ☐ No
- Chronic pain? ☐ Yes ☐ No
- Back problems? ☐ Yes ☐ No
- Diabetes? ☐ Yes ☐ No

If you answered yes to any of these questions, please describe:

17. Have you ever had any of the following?

- A chronic infectious disease ☐ Yes ☐ No
- Neurodegenerative condition ☐ Yes ☐ No
- Epilepsy ☐ Yes ☐ No
- Multiple Sclerosis ☐ Yes ☐ No
- Cancer affecting the brain ☐ Yes ☐ No
- Moderate or severe traumatic brain injury ☐ Yes ☐ No

If you answered yes to any of these questions, please describe:

18. Do you have any breathing/respiratory problems?
(e.g., asthma, chronic obstructive pulmonary disease) ☐ Yes ☐ No

If yes, please describe what they are:

19. Have you ever had a collapsed lung?
(pneumothorax) ☐ Yes ☐ No
20. Have you ever had major surgery on your head, neck or lungs? □ Yes □ No
If yes, what have you done and when?

21. MEDICATION:
Do you take medication? □ Yes □ No
If you answered yes to the previous question and you are happy to give us the details, please give the details below

<table>
<thead>
<tr>
<th>Medication name</th>
<th>Dose</th>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

22. MENTAL HEALTH:
Have you ever seen a doctor for mental health issues (e.g., depression, anxiety)? □ Yes □ No
If yes, please state the diagnosis and year of diagnosis:
_________________________________________ Year ________
_________________________________________ Year ________

23. Were you hospitalised as a result of this diagnosis? □ Yes □ No
24. YOUR BREATHING AND SLEEPING:

Are you able to breathe through your nose?

☐ Easily
☐ Some obstruction
☐ Not at all

25. Can you climb a flight of stairs without stopping?  ☐ Yes ☐ No
If no, what stops you?

Breathlessness  ☐ Yes ☐ No
Angina (heart pains)  ☐ Yes ☐ No
Arthritis  ☐ Yes ☐ No

Other (please describe below)

________________________________________________________________________

26. SLEEP:

Is your sleep ever disturbed by other people / children / pets in your household?  ☐ Yes ☐ No
If yes, please describe:

________________________________________________________________________

27. Have you ever been diagnosed with a sleep disorder?  (e.g., sleep apnoea, insomnia)  ☐ Yes ☐ No
If yes, what was the diagnosis?

________________________________________________________________________

When were you diagnosed?  (Year) __________
If you are undergoing treatment, please describe:

________________________________________________________________________
28. Do you take naps during the day?
   □ Nearly every day
   □ 3-4 times a week
   □ 1-2 times a week
   □ 1-2 times a month
   □ Never or nearly never

29. Do you suffer from nightmares or night terrors? □ Yes □ No
   If yes, does this disturb your sleep? □ Yes □ No

YOUR LIFE HABITS

30. SMOKING
   Do you currently smoke? □ Yes □ No

   How many cigarettes per day do you smoke? *(Tick one box)*
   □ 10 or less
   □ 11-20
   □ 21-30
   □ 31 or more

31. Have you ever been a regular smoker? □ Yes □ No

   If you have stopped smoking, but were once a regular smoker how many years ago
did you stop smoking? Years _________________________

   How many cigarettes per day did you smoke?
   □ 10 or less
   □ 11-20
   □ 21-30
   □ 31 or more
32. **ALCOHOL**

How often do you have a drink that contains alcohol?

☐ Never
☐ Monthly or less
☐ 2-4 times per month
☐ 2-3 times per week
☐ 4 times or more per week

How many standard alcoholic drinks do you have on a typical day when you are drinking?

☐ 1-2
☐ 3-4
☐ 5-6
☐ 7-8
☐ 9 +

How often do you have 6 or more standard drinks on one occasion?

☐ Never
☐ Less than monthly
☐ Monthly
☐ Weekly
☐ Daily or almost daily

33. **DRUGS:**

Do you use illicit drugs (e.g., cannabis, speed, meth)?

☐ Yes
☐ No
☐ Decline to say

If you answered yes to the previous question, what types of drugs?

________________________________________________________________________

________________________________________________________________________

How often do you use?

________________________________________________________________________

Do you wish to add any comments?

________________________________________________________________________
YOUR SLEEP HABITS
The following questions relate to your usual sleep habits during the **past month ONLY**.
Your answers should indicate the most accurate reply for the majority of days and nights in the **past month**.

34. During the past month, what time have you usually gone to bed at night? (Bed time)

Please write time using 15 minute divisions and include am/pm

e.g., 10:45pm  ____ : ____ am/pm

35. During the past month, how long has it usually taken you to fall asleep each night?
(Number of minutes)  _______ mins

36. During the past month, what time have you usually got up in the morning? (Getting up time)

Please write time using 15 minute divisions and include am/pm

e.g., 06:30am  ____ : ____ am/pm

37. During the past month, how many hours of actual sleep did you get at night? This may be different than the number of hours you spent in bed. (Hours of sleep per night)

_______ hours

38. During the past month, how often have you had trouble sleeping because you...

(Please tick once on each line)

<table>
<thead>
<tr>
<th>Reason</th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td>cannot get to sleep within 30 minutes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>wake up in the middle of the night or early morning</td>
<td></td>
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<td></td>
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<tr>
<td>have to get up to use the bathroom</td>
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<tr>
<td>cannot breathe comfortably</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>cough or snore loudly</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>feel too cold</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>feel too hot</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>had bad dreams</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>have pain</td>
<td></td>
<td></td>
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</tbody>
</table>

Other reason(s); also please describe below

Other; please describe:

__________________________________________________________________________

__________________________________________________________________________
<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>39. During the past month, how would you rate your sleep quality overall?</td>
<td>- Very good&lt;br&gt;- Fairly good&lt;br&gt;- Fairly bad&lt;br&gt;- Very bad</td>
</tr>
<tr>
<td>40. During the past month, how often have you taken medicine to help you sleep</td>
<td>- Not during the past month&lt;br&gt;- Less than once a week&lt;br&gt;- Once or twice a week&lt;br&gt;- Three or more times a week</td>
</tr>
<tr>
<td>41. During the past month, how much of a problem has it been for you to keep up</td>
<td>- No problem at all&lt;br&gt;- Only a very slight problem&lt;br&gt;- Somewhat of a problem&lt;br&gt;- A very big problem</td>
</tr>
<tr>
<td>42. Do you have a bed partner or roommate?</td>
<td>- No bed partner or roommate&lt;br&gt;- Partner / roommate in other room&lt;br&gt;- Partner in same room, but not same bed&lt;br&gt;- Partner in same bed</td>
</tr>
</tbody>
</table>
If you answered in the last question that you have a roommate or bed partner, ask him/her how often in the **past month** you have had...

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
<th>Unsure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loud snoring</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Long pauses between breaths while asleep</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Legs twitching or jerking while you sleep</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Episodes of disorientation or confusion during sleep</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other restlessness while you sleep; please describe below</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Other restlessness; please describe:

______________________________________________________________

43. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

- [ ] Not during the past month
- [ ] Less than once a week
- [ ] Once or twice a week
- [ ] Three or more times a week

Do you wish to add any comments?

______________________________________________________________
YOUR SLEEPING AND BREATHING

44. Do you snore?

☐ Yes  ☐ No  ☐ Don’t know

(If you answered no or don’t know to the last question please go to Question 46)

Your snoring is:

☐ Slightly louder than breathing
☐ As loud as talking
☐ Louder than talking
☐ Very loud - can be heard in adjacent rooms
☐ Don’t know

How often do you snore?

☐ Nearly every sleep
☐ 3-4 times a week
☐ 1-2 times a week
☐ 1-2 times a month
☐ Never or nearly never
☐ Don’t know

45. Has your snoring ever bothered other people?

☐ Yes  ☐ No

46. Has anyone ever noticed that you stop breathing during your sleep?

☐ Nearly every day
☐ 3-4 times a week
☐ 1-2 times a week
☐ 1-2 times a month
☐ Never or nearly never

47. How often do you feel tired or fatigued after your sleep?

☐ Nearly every day
☐ 3-4 times a week
☐ 1-2 times a week
☐ 1-2 times a month
☐ Never or nearly never
48. During your wake time, do you feel tired, fatigued or not up to par?

☐ Nearly every day
☐ 3-4 times a week
☐ 1-2 times a week
☐ 1-2 times a month
☐ Never or nearly never

49. Have you ever nodded off or fallen asleep while driving a vehicle?

☐ Yes   ☐ No

If you answered yes to the previous question, how often does it occur?

☐ Nearly every day
☐ 3-4 times a week
☐ 1-2 times a week
☐ 1-2 times a month
☐ Never or nearly never

50. Do you have high blood pressure?

☐ Yes   ☐ No   ☐ Don’t know

Do you wish to add any comments?

__________________________________________________________________

__________________________________________________________________
**SLEEPINESS**

How likely are you to fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in the *last few weeks*.

Even if you have not done some of the things recently, try to work out how they would have affected you.

51. How likely are you to ‘doze’ in the following situations, in contrast to just feeling tired?

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Would never doze</th>
<th>Slight chance of dozing</th>
<th>Moderate chance of dozing</th>
<th>High chance of dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Watching TV</td>
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<tr>
<td>Sitting inactive in a public place (e.g., a theatre or meeting)</td>
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<tr>
<td>As a passenger in a car for an hour without a break</td>
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<tr>
<td>Lying down in the afternoon, when circumstances allow</td>
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<tr>
<td>Sitting and talking to someone</td>
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<tr>
<td>Sitting quietly after lunch without alcohol</td>
<td></td>
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<tr>
<td>In a car, while stopped for a few minutes in traffic</td>
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</tbody>
</table>

Do you wish to add any comments?  
__________________________________________________________________________
__________________________________________________________________________
__________________________________________________________________________
EFFECTS ON YOUR EVERYDAY LIFE

The following questions ask about how your sleep affects your everyday life.

You will be asked whether you **generally** experience difficulties in carrying out activities of daily living because you feel tired or sleepy. It is important to note that the words tired or sleepy refer to the feeling of being unable to keep your eyes open or your head up due to sleepiness, the need to doze, or an urgent need to sleep.

Tiredness does not refer to the feeling you might experience after exercise.

52.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Yes extreme</th>
<th>Yes moderate</th>
<th>Yes a little</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you generally have difficulty concentrating on the things you do because you are sleepy or tired?</td>
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</tr>
<tr>
<td>Do you generally have difficulty remembering things because you are sleepy or tired?</td>
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<tr>
<td>Do you have difficulty finishing a meal because you become sleepy or tired?</td>
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</tr>
</tbody>
</table>

53.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Don't do this activity</th>
<th>Yes extreme</th>
<th>Yes moderate</th>
<th>Yes a little</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have difficulty working on a hobby (for example, collecting, gardening) because you are sleepy or tired?</td>
<td></td>
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<tr>
<td>Do you have difficulty doing work around the house (for example, cleaning house, doing laundry, taking out rubbish, repair work) because you are sleepy or tired?</td>
<td></td>
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<tr>
<td>Do you have difficulty operating a motor vehicle for short distances (less than 160 km) because you become sleepy or tired?</td>
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<tr>
<td>Do you have difficulty operating a motor vehicle for long distances (greater than 160 km) because you become sleepy or tired?</td>
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<td></td>
</tr>
<tr>
<td>(Please tick once on each line)</td>
<td>Don't do this activity</td>
<td>Yes extreme</td>
<td>Yes moderate</td>
<td>Yes a little</td>
<td>No</td>
</tr>
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</tr>
<tr>
<td>Do you have difficulty getting things done because you are too sleepy or tired to drive or take public transportation?</td>
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<tr>
<td>Do you have difficulty taking care of financial affairs and doing paperwork (for example, writing cheques, paying bills, keeping financial records, filling out tax forms etc.) because you are sleepy or tired?</td>
<td></td>
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<tr>
<td>Do you have difficulty performing employed or volunteer work because you are sleepy or tired?</td>
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<tr>
<td>Do you have difficulty maintaining a telephone conversation because you become sleepy or tired?</td>
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<tr>
<td>Do you have difficulty visiting with your family or friends in your home because you become sleepy or tired?</td>
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<td></td>
</tr>
<tr>
<td>Do you have difficulty visiting your family or friends in their home because you become sleepy or tired?</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Do you have difficulty doing things for your family or friends because you are too sleepy or tired?</td>
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<td></td>
</tr>
</tbody>
</table>

54. Has your relationship with family, friends, or work colleagues been affected because you are sleepy or tired?

- [ ] Yes extreme
- [ ] Yes moderate
- [ ] Yes a little
- [ ] No
55. (Please tick once on each line)

<table>
<thead>
<tr>
<th>Activity</th>
<th>Don't do this activity</th>
<th>Yes extreme</th>
<th>Yes moderate</th>
<th>Yes a little</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have difficulty exercising or participating in a sporting activity because you are too sleepy or tired?</td>
<td></td>
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</tr>
<tr>
<td>Do you have difficulty watching a movie because you become sleepy or tired?</td>
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<tr>
<td>Do you have difficulty enjoying the theatre or a lecture because you become sleepy or tired?</td>
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<tr>
<td>Do you have difficulty enjoying a concert because you become sleepy or tired?</td>
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<td></td>
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<td></td>
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<tr>
<td>Do you have difficulty watching television because you are sleepy or tired?</td>
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<tr>
<td>Do you have difficulty participating in religious services, meetings or a group or club because you are sleepy or tired?</td>
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</tr>
</tbody>
</table>

56. (Please tick once on each line)

<table>
<thead>
<tr>
<th>Activity</th>
<th>Yes extreme</th>
<th>Yes moderate</th>
<th>Yes a little</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you have difficulty being as active as you want to be in the evening because you are sleepy or tired?</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Do you have difficulty being as active as you want to be in the morning because you are sleepy or tired?</td>
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<tr>
<td>Do you have difficulty being as active as you want to be in the afternoon because you are sleepy or tired?</td>
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<tr>
<td>Do you have difficulty keeping pace with others your own age because you are sleepy or tired?</td>
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</tr>
</tbody>
</table>

57. How would you rate your general level of activity?

- [ ] Very low
- [ ] Low
- [ ] Medium / moderate
- [ ] High
### 58.

**Has your intimate or sexual relationship been affected because you are sleepy or tired?**

<table>
<thead>
<tr>
<th>Don't do this activity for other reasons</th>
<th>Yes, extreme</th>
<th>Yes, a little</th>
<th>No</th>
<th>Decline to say</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

**Has your desire for intimacy or sex been affected because you are sleepy or tired?**

<p>| | | | | |</p>
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</tbody>
</table>

**Has your ability to become sexually aroused been affected because you are sleepy or tired?**

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</tbody>
</table>

**Has your ability to have an orgasm been affected because you are sleepy or tired?**

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<table>
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</table>

**Do you wish to add any comments?**

________________________________________________________________________________________

________________________________________________________________________________________

________________________________________________________________________________________
YOUR VIEWS ABOUT YOUR MOOD

We realise that some of the questions seem very similar, but there are subtle differences, so answering all of them really is helpful.

For each statement, please select the box that best describes how often you felt or behaved this way during the past week.

Don’t take too long over your responses; your first reaction to each statement will probably be more accurate than a long thought-out response.

59. Over the past week...

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Did not apply to me at all</th>
<th>Applied to me to some degree, or some of the time</th>
<th>Applied to me to a considerable degree, or a good deal of the time</th>
<th>Applied to me very much, or most of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I found it hard to wind down</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>I was aware of dryness of mouth</td>
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<tr>
<td>I couldn’t seem to experience any positive feeling at all</td>
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<tr>
<td>I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)</td>
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<tr>
<td>I found it difficult to work up the initiative to do things</td>
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<tr>
<td>I tended to over-react to situations</td>
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<tr>
<td>I experienced trembling (e.g., in the hands)</td>
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<tr>
<td>I felt that I was using a lot of nervous energy</td>
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<tr>
<td>I was worried about situations in which I might panic and make a fool of myself</td>
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<tr>
<td>I felt that I had nothing to look forward to</td>
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<tr>
<td>I found myself getting agitated</td>
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<tr>
<td>I found it difficult to relax</td>
<td></td>
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<tr>
<td>I felt down-hearted and blue</td>
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<tr>
<td>I was intolerant of anything that kept me from getting on with what I was doing</td>
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<tr>
<td>I felt I was close to panic</td>
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<tr>
<td>I was unable to become enthusiastic about anything</td>
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<tr>
<td>I felt I wasn’t worth much as a person</td>
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<tr>
<td>I felt that I was rather touchy</td>
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<tr>
<td>(Please tick once on each line)</td>
<td>Did not apply to me at all</td>
<td>Applied to me by some degree, or some of the time</td>
<td>Applied to me to a considerable degree, or a great deal of the time</td>
<td>Applied to me very much, or most of the time</td>
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<tr>
<td>---------------------------------------------------------------</td>
<td>----------------------------</td>
<td>--------------------------------------------------</td>
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<tr>
<td>I was aware of the action of my heart in the absence of</td>
<td></td>
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<tr>
<td>physical exertion (e.g., sense of heart rate increase, heart</td>
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<tr>
<td>missing a beat)</td>
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<tr>
<td>I felt scared without any good reason</td>
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<tr>
<td>I felt that life was meaningless</td>
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</tbody>
</table>

60. Over the **last 2 weeks**, how often have you been bothered by any of the following problems?

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Little interest or pleasure in doing things</td>
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<tr>
<td>Feeling down, depressed, or hopeless</td>
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<tr>
<td>Trouble falling or staying asleep, or sleeping too much</td>
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<tr>
<td>Feeling tired or having little energy</td>
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<tr>
<td>Poor appetite or overeating</td>
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<tr>
<td>Feeling bad about yourself, or that you are a failure, or have let yourself or your family down</td>
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<tr>
<td>Trouble concentrating on things, such as reading the newspaper or watching television</td>
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<tr>
<td>Moving or speaking so slowly that other people could have noticed. Or the opposite - being so fidgety or restless that you have been moving around a lot more than usual</td>
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<tr>
<td>Thoughts that you would be better off dead, or of hurting yourself in some way</td>
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</tr>
</tbody>
</table>
YOUR PERSONAL THOUGHTS AND BEHAVIOUR

61. Please select one of the five boxes on each line according to the extent to which you consider the statements apply to you.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Strongly disagree</th>
<th>Disagree</th>
<th>Neither disagree or agree</th>
<th>Agree</th>
<th>Strongly agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>There are things I prefer not to think about</td>
<td></td>
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<tr>
<td>Sometimes I wonder why I have the thoughts I do</td>
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<tr>
<td>I have thoughts that I cannot stop</td>
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<tr>
<td>There are images that come to mind that I cannot erase</td>
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<tr>
<td>My thoughts frequently return to one idea</td>
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<tr>
<td>I wish I could stop thinking of certain things</td>
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<tr>
<td>Sometimes my mind races so fast I wish I could stop it</td>
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<tr>
<td>I always try to put problems out of mind</td>
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<tr>
<td>There are thoughts that keep jumping into my head</td>
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<tr>
<td>Sometimes I stay busy just to keep thoughts from intruding on my mind</td>
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<td></td>
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<tr>
<td>There are things that I try not to think about</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Sometimes I really wish I could stop thinking</td>
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<tr>
<td>I often do things to distract myself from my thoughts</td>
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<tr>
<td>I often have thoughts that I try to avoid</td>
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<tr>
<td>There are many thoughts that I have that I don't tell anyone</td>
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</tbody>
</table>
YOUR BEHAVIOUR

Below is a list of phrases that can be used to describe a person’s behaviour.

62. Please read each phrase carefully. Using the rating scale provided, indicate how often you have engaged in the behaviour described. Please provide a rating for all of the statements.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Almost never</th>
<th>Seldom</th>
<th>Sometimes</th>
<th>Frequently</th>
<th>Almost always</th>
<th>Don’t know to say</th>
</tr>
</thead>
<tbody>
<tr>
<td>I speak only when spoken to</td>
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<tr>
<td>I am easily angered or irritated; I have emotional outbursts without good reason</td>
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<td></td>
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<tr>
<td>I repeat certain actions or get stuck on certain ideas</td>
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<tr>
<td>I do things impulsively</td>
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<tr>
<td>I mix up a sentence, get confused when doing several things in a row</td>
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<tr>
<td>I laugh or cry too easily</td>
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<tr>
<td>I make the same mistakes over and over, do not learn from past experience</td>
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<tr>
<td>I have difficulty starting an activity, lack initiative, motivation</td>
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<tr>
<td>I make inappropriate sexual comments and advances, am too flirtatious</td>
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<tr>
<td>I do or say embarrassing things</td>
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<tr>
<td>I neglect my personal hygiene</td>
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<td>I can’t sit still, am hyperactive</td>
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<tr>
<td>I am unaware of my problems when I make mistakes</td>
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<tr>
<td>I sit around doing nothing</td>
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<tr>
<td>I am disorganised</td>
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<td>I lose control of my urine or bowels and it doesn’t seem to bother me</td>
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<tr>
<td>I cannot do two things at once (for example, talk and prepare a meal)</td>
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<tr>
<td>I talk out of turn, interrupt others in conversations</td>
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<tr>
<td>I show poor judgment, poor problem solving</td>
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<tr>
<td>I make up fantastic stories when unable to remember something</td>
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<tr>
<td>I have lost interest in things that used to be fun or important to me</td>
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<tr>
<td>I say one thing, then do another thing</td>
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<tr>
<td>(Please tick once on each line)</td>
<td>Almost never</td>
<td>Seldom</td>
<td>Sometimes</td>
<td>Frequently</td>
<td>Almost always</td>
<td>Decline to say</td>
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<tr>
<td>I start things but fail to finish them, “peter out”</td>
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<tr>
<td>I show little emotion, am unconcerned and unresponsive</td>
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<tr>
<td>I forget to do things but then remember when prompted or when it is too late</td>
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<tr>
<td>I am inflexible, unable to change routines</td>
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<tr>
<td>I get in trouble with the law or authorities</td>
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<tr>
<td>I do risky things just for the heck of it</td>
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<tr>
<td>I am slow moving, lack energy, inactive</td>
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<tr>
<td>I am overly silly, have a childish sense of humour</td>
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<tr>
<td>I find that food has no taste or smell</td>
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<tr>
<td>I swear</td>
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<tr>
<td>I apologise for misbehaviour (for example, apologise for swearing)</td>
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<tr>
<td>I pay attention, concentrate even when there are distractions</td>
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<tr>
<td>I think things through before acting (for example, consider finances before spending money)</td>
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<tr>
<td>I use strategies to remember important things (for example, write notes to myself)</td>
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<tr>
<td>I am able to plan ahead</td>
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<tr>
<td>I am interested in sex</td>
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<tr>
<td>I care about my appearance (for example, daily grooming)</td>
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<td>I benefit from feedback, accept constructive criticism from others</td>
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<tr>
<td>I get involved with activities spontaneously (such as hobbies)</td>
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<tr>
<td>I do things without being requested to do so</td>
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<tr>
<td>I am sensitive to the needs of other people</td>
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<tr>
<td>I get along well with others</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>I act appropriately for my age</td>
<td></td>
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<tr>
<td>I can start conversations easily</td>
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</tbody>
</table>

63. Do you wish to add any comments?

__________________________________________________________

__________________________________________________________

A STUDY ON HOW SLEEP AFFECTS MEMORY AND THINKING SKILLS (JH/10/11)

PAGE 24
EXPERIENCES YOU MAY HAVE HAD

Some of these questions may seem a little strange, but these experiences are actually much more common than previously thought, and many people experience them during the course of their everyday lives.

64. Please indicate if you have had any of these experiences during the last month.

Have there been times when you felt that something strange was going on?
☐ Yes ☐ No

Have you ever felt that your thoughts or actions were directly interfered with or controlled by some outside force or person?
☐ Yes ☐ No

Have there been times when you felt that people were against you?
☐ Yes ☐ No

Have there been times when you felt that people were deliberately acting to harm you, or plotting against you?
☐ Yes ☐ No

Have there been times when you heard or saw things that other people couldn’t?
☐ Yes ☐ No

Did you at any time hear voices saying quite a few words or sentences when there was no one around who might account for it?
☐ Yes ☐ No

Have you been told that you may have schizophrenia, schizoaffective disorder, bipolar disorder, or manic depression?
☐ Yes ☐ No

Do you wish to add any comments?

____________________________________________________________________

____________________________________________________________________

____________________________________________________________________

____________________________________________________________________
**HOW YOU FEEL ABOUT YOUR MEMORY**

Below are statements about feelings that people may have about their memory.

65. Please read each statement and decide whether you agree.

Think about how you have been feeling over the **past two weeks**, then select the appropriate box.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Strongly disagree</th>
<th>Disagree</th>
<th>Undecided</th>
<th>Agree</th>
<th>Strongly agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>I am generally pleased with my memory ability</td>
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<tr>
<td>There is something seriously wrong with my memory</td>
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<tr>
<td>If something is important, I will probably remember it</td>
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<td>When I forget something, I fear that I may have a serious memory problem, like Alzheimer’s disease</td>
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<tr>
<td>My memory is worse than most other people of my age</td>
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<tr>
<td>I have confidence in my ability to remember things</td>
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<tr>
<td>I feel unhappy when I think about my memory ability</td>
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<td>I worry that others will notice my memory is not very good</td>
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<tr>
<td>When I have trouble remembering something, I am not too hard on myself</td>
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<tr>
<td>I am concerned about my memory</td>
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<td>My memory is really going downhill lately</td>
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<tr>
<td>I am generally satisfied with my memory ability</td>
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<tr>
<td>I don’t get upset when I have trouble remembering something</td>
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<tr>
<td>I worry that I will forget something important</td>
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<tr>
<td>I am embarrassed about my memory ability</td>
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<tr>
<td>I get annoyed or irritated with myself when I am forgetful</td>
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<tr>
<td>My memory is good for my age</td>
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<tr>
<td>I worry about my memory ability</td>
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</tbody>
</table>
66. Any changes in your memory?

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Much worse</th>
<th>Worse</th>
<th>Unchanged</th>
<th>Better</th>
<th>Much better</th>
</tr>
</thead>
<tbody>
<tr>
<td>Has your memory changed in the last 5 years?</td>
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<tr>
<td>Has your memory changed in the last 12 months?</td>
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</table>

Please describe what changes you have noticed if any:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Do you wish to add any comments?

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
MEMORY MISTAKES

Below is a list of common memory mistakes that people make.

67. Decide how often you have done each one in the last two weeks, then select the appropriate box.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>All the time</th>
<th>Often</th>
<th>Sometimes</th>
<th>Rarely</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>How often do you forget to pay a bill on time?</td>
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<tr>
<td>How often do you misplace something you use daily, like your keys or glasses?</td>
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<tr>
<td>How often do you have trouble remembering a telephone number you just looked up?</td>
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<tr>
<td>How often do you not recall the name of someone you just met?</td>
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<td>How often do you leave something behind when you meant to bring it with you?</td>
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<tr>
<td>How often do you forget an appointment?</td>
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<tr>
<td>How often do you forget what you were just about to do; for example, walk into a room and forget what you went in there to do?</td>
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<tr>
<td>How often do you forget to run an errand?</td>
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<tr>
<td>How often do you have difficulty coming up with a specific word that you want?</td>
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<tr>
<td>How often do you have trouble remembering details from a newspaper or magazine article you read earlier that day?</td>
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<tr>
<td>How often do you forget to take medication?</td>
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<tr>
<td>How often do you not recall the name of someone you have known for sometime?</td>
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<tr>
<td>How often do you forget to pass on a message?</td>
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<tr>
<td>How often do you forget what you were going to say in a conversation?</td>
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<tr>
<td>How often do you forget a birthday or anniversary that you used to know well?</td>
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<tr>
<td>How often do you forget a telephone number you that use frequently?</td>
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<tr>
<td>(Please tick once on each line)</td>
<td>All the time</td>
<td>Often times</td>
<td>Rarely</td>
<td>Never</td>
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<tr>
<td>-----------------------------------------------------------------------------------------------</td>
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<tr>
<td>How often do you retell a story or joke to the same person because you forgot that you had already told him or her?</td>
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<tr>
<td>How often do you misplace something that you put away a few days ago?</td>
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<tr>
<td>How often do you forget to buy something you intended to buy?</td>
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<tr>
<td>How often do you forget details about a recent conversation?</td>
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</tbody>
</table>

Do you wish to add any comments?

_________________________________________________________________________________________

_________________________________________________________________________________________
### DIFFICULTIES IN EVERYDAY LIFE

This is the last set of questions!

68. Below is a list of statements. Decide how often you have done each one in the **last two weeks**, then select the appropriate box.

<table>
<thead>
<tr>
<th>(Please tick once on each line)</th>
<th>Never</th>
<th>Rarely</th>
<th>Some times</th>
<th>Often</th>
<th>All the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>When interrupted while reading, I have trouble finding my place again</td>
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<tr>
<td>I need a written list when I do errands</td>
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<tr>
<td>I forget appointments, dates or meetings</td>
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<tr>
<td>I forget to return phone calls</td>
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<tr>
<td>I have trouble getting my keys into a lock</td>
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<tr>
<td>I forget errands I planned to do</td>
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<tr>
<td>I have trouble recalling names of people I know</td>
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<tr>
<td>I find it hard to keep my mind on a task or a job</td>
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<tr>
<td>I have trouble describing a programme I have just watched on television</td>
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<tr>
<td>I have trouble expressing what I mean to say</td>
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<tr>
<td>I fail to recognise people I know</td>
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<tr>
<td>I have trouble getting out a word that’s on the tip of my tongue</td>
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<tr>
<td>I find it hard to understand what I read</td>
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<tr>
<td>I forget names of people soon after being introduced</td>
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<tr>
<td>I lose my train of thought when I listen to somebody else</td>
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<tr>
<td>I forget what day of the week it is</td>
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<tr>
<td>I cannot keep my mind on one thing</td>
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<tr>
<td>I have trouble manipulating buttons and zips</td>
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<tr>
<td>I have trouble sewing, mending, making minor household repairs</td>
<td></td>
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</tbody>
</table>
(Please tick once on each line) | Never | Rarely | Sometimes | Often | All the time |
--- | --- | --- | --- | --- | --- |
I have trouble fixing my mind on what I’m reading |   |   |   |   |   |
I forget right away what people say to me |   |   |   |   |   |
I forget to pay bills, record cheques, or mail letters |   |   |   |   |   |
My mind just goes blank at times |   |   |   |   |   |
I forget the date of the month |   |   |   |   |   |
I have trouble manipulating tools, scissors, corkscrews or can-openers |   |   |   |   |   |
I make mistakes in writing or calculating |   |   |   |   |   |

Do you wish to add any comments?  
_________________________________________________________________________  
_________________________________________________________________________  
_________________________________________________________________________

How long did you take to complete this questionnaire?  , (minutes) 

Date of submitting this survey: DD/MM/YYYY (day/month/year)  
____ / ____ / ________

Although it is not possible to give you individual feedback about your responses on this survey, if you wish we can email you a summary of what was found about the group as a whole, once the study is over (in approximately 2013).  

Please enter your email address below if you wish to receive this summary:  
_________________________________________________________________________

THANK YOU!  
The survey is now finished. Thank you very much for taking the time and trouble to complete this questionnaire. We are most grateful.  

Before submitting, we do have another question for you...
A FURTHER STUDY - WE NEED MORE HELP!

Your answers are very important to us. If you enjoyed answering these questions and would like to help further...

We will be following up this survey with a second study, for which we are selecting people with and without sleep difficulties. As this further study will be carried out in person and not online or through a written survey you must therefore live in Western Australia where we are based.

If you are willing to be contacted about the second study and you are aged 55-75, please tick ‘yes’ and complete your contact details below. These details will be kept separately from your earlier answers to preserve your confidentiality.

We will contact you in due course to explain what will be involved. If you then decide that, after all, it’s not for you, there is no obligation to take part.

I live in Western Australia

☐ Yes  ☐ No

I am between the ages of 55 - 75

☐ Yes  ☐ No

I am happy for you to contact me about taking part in a further study.

☐ Yes  ☐ No

The next page will be kept separately from your earlier answers to preserve your confidentiality.

If you have any questions please do not hesitate to contact us.

Chief Investigator: Alix Mellor
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Supervisors:
A/Prof Flavie Waters Phone: (08) 9347 6429 / 6650
Dr Romola Bucks Phone: (08) 6488 3232
Your contact details:

First name: ____________________________________________
Family/surname: _______________________________________
Address House number: __________________________________
Address Street: _________________________________________
Town/City: ____________________________________________
State:: ________________________________________________
Post Code: _____________________________________________
email: _________________________________________________
Home phone with prefix: _____________________________
Mobile phone: _________________________________
Preferred method of contact - post/email/phone/mobile: ________________________________
If by phone/mobile - best time to contact me: ________________________________

Thank you again for your kind help with this project. We are most grateful for your time. If you have any questions please do not hesitate to contact us.

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