Age-related Prospective Memory Decline:
Contributions from Sleep and Opportunities for Intervention

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B.A (Honours)

This thesis is presented for the degree of Doctor of Philosophy, and in partial fulfilment of the requirements for the Master of Clinical Neuropsychology of

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School of Psychological Science

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Thesis Declaration

I, Lara Fine, certify that:

This thesis has been substantially accomplished during enrolment in this degree.

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The research involving human data reported in this thesis was assessed and approved by The University of Western Australia Human Research Ethics Committee. Approval #s: 5361; 2086; 7799. Written patient consent has been received and archived for the research involving patient data reported in this thesis.

This thesis contains published work and work prepared for publication, some of which has been co-authored.

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Date: 7th October 2019
Abstract

It is well established that older adults experience prospective memory deficits. However, research evaluating prospective memory interventions for older adults is limited. This thesis explores two novel avenues to improve older adults’ prospective memory. First, sleep is recognised as an important determinant of prospective memory performance in younger adults, and of older adults’ cognition generally. Thus, this thesis examines whether sleep disruption relates to poorer prospective memory in older adults; whether sleep disruption mediates age-related prospective memory decline; and whether improving older adults’ sleep can improve their prospective memory. Second, regarding potential cognitive interventions for prospective memory, age-related prospective memory decline is often conceptualised as decline in the executive aspects of prospective memory specifically. However, these aspects have, largely, been ignored in the intervention literature. Thus, this thesis evaluated a novel executive function intervention for prospective memory in older adults.

To evaluate the relationship between sleep disruption and prospective memory and ageing (Chapter 2), older adults undertook assessment of sleep using actigraphy, and prospective memory using a laboratory task. Results indicated longer awakenings were associated with poorer prospective memory. Furthermore, the relationship between older age and poorer prospective memory appeared to operate via longer awakening length.

Building on these results, Chapter 3 examined whether an intervention to improve older adults’ sleep could also improve their prospective memory. Older adults with poor sleep were allocated either to a cognitive behavioural therapy for insomnia (CBT-I) group, or waitlist control. Participants underwent assessment pre and post-intervention of prospective memory, and sleep with actigraphy and questionnaires. Contrary to
prediction, despite subjective insomnia symptoms improving, CBT-I did not improve objective sleep, nor prospective memory.

In a second intervention study, using a neuropsychological approach to treatment, (Chapter 4), older adults were allocated to receive (1) goal management training (an executive function intervention), (2) an established intervention for prospective memory’s retrospective components (implementation intentions), or (3) no intervention. Prospective memory was assessed pre and post-intervention. Results suggested some older adults had difficulty comprehending goal management training. Post-hoc analysis suggested that for those who did comprehend it, prospective memory improved relative to no intervention, but only for participants with poorer baseline prospective memory. Implementation intentions was similarly effective for participants with poorer baseline prospective memory. Direct comparison of the interventions did not suggest significant differences between them.

Overall, the novel contributions made by this thesis are substantial. It includes studies that are among the first to examine (1) the relationship between prospective memory and sleep in older adults, (2) whether treating sleep problems can improve prospective memory, and (3) whether goal management training is effective at improving prospective memory in older adults.

In summary, this thesis found there are possible opportunities to improve older adults’ prospective memory through sleep and executive function strategies. However, several cautionary points were raised. First, interventions appear likely to be specifically effective for older adults with poorer prospective memory, rather than all older adults. Second, whilst a relationship between sleep and prospective memory was demonstrated, suggesting improving sleep could improve prospective memory, older adults’ sleep disturbance may be more resistant to treatment with CBT-I. This finding needs further exploration, particularly as to whether potential treatment resistance relates to
neurological changes underpinning poor sleep. Finally, when using executive interventions to improve prospective memory, researchers and clinicians will need to consider the complexity of the strategy and amount of training older adults will need. This is especially pertinent if intervention is targeted at older adults with poorer prospective memory, who may have potential underlying cognitive deficits affecting their comprehension of the intervention.
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1. Details of the work:


Location in thesis: Chapter 2.

Student contribution to work: This study was designed by the candidate in collaboration with her supervisors, Dr Michael Weinborn and Professor Romola Bucks. The candidate recruited participants, administered the cognitive battery, entered cognitive data, and collected and processed sleep data, in conjunction with other PhD and Honours students who were also part of the larger research project (the Healthy Ageing Research Program, Directors Professor Bucks and Dr Weinborn). The candidate assisted with experimental programming, in conjunction with Dr Ryan Li and Dr Amanda Ng. The candidate conducted all statistical analyses with guidance from her supervisors, wrote the manuscript, and made revisions in accordance with feedback provided by supervisors, co-authors and journal reviewers.

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CHAPTER 1

General Introduction
Prospective memory (PM) is the ability to remember and execute a task in the future. PM is pivotal in enabling independent living, by supporting financial, social, and health behaviours (Woods, Weinborn, Velnoweth, Rooney, & Bucks, 2012). Failures of PM are not just an inconvenience but can also be life threatening, for example in health-related behaviours such as remembering medications. As reviewed in the current chapter, age-related decline in PM has been well researched. However, the role of sleep disruption in age-related PM decline has not yet been examined, despite the links between sleep and cognition amongst older adults, highlighted below. The potential relationship between sleep and age-related PM deficits is important because improving sleep may be a way to improve PM in older adults. Another avenue to improving older adults’ PM is through cognitive interventions. Some research has evaluated cognitive interventions for PM. However, this chapter highlights that the focus of these interventions has been quite narrow, mostly limited to targeting the retrospective memory component of PM. The literature on age-related PM decline is drawn upon to emphasise that PM interventions should also aim to compensate for the executive function deficits that are thought to play a particularly pivotal role in age-related PM decline.

An overview of prospective memory

Successful PM performance has several components (Kliegel, Martin, McDaniel, & Einstein, 2002). First, a mental link is drawn between the PM task to be accomplished and a particular environmental cue that will trigger task execution. This cue can be based either on a particular event occurring (event-based) or on a set clock-time (time-based). Second, the intention to execute the PM task is maintained whilst other tasks are undertaken. Third, the cue is successfully detected, and, fourth, other ongoing tasks are temporarily paused. Finally, the PM task is correctly recalled and executed. Each of these components of successful PM are themselves supported by
other cognitive domains. Retrospective memory is important for successfully encoding the link between the cue and the PM task, and later recalling it when needed (West, 2005). Elements of executive function are important for (1) planning the fulfilment of the PM task, (2) maintaining the PM intention, (3) monitoring the environment for the PM cue, (5) controlling memory retrieval, and (6) activating appropriate behaviours after cue detection (Kliegel et al., 2002).

Several theories seek to explain PM task performance. The Prospective Memory Decision Control (PMDC) Theory describes an interplay between cognitive control and cognitive capacity that accounts for PM performance (Strickland, Loft, Remington, & Heathcote, 2018). The Preparatory Attentional and Memory Process (PAM) Theory posits that individuals must always allocate attentional resources to monitor for PM targets in a resource demanding process that detracts from performance on ongoing tasks (Smith, 2003; West, 2005). A dominant theory used throughout this thesis – the Multiprocess Framework – suggests strategic monitoring for the PM cue is only necessary under certain more demanding task conditions, e.g. time-based cues, non-focal event-based cues, low cue distinctiveness, and low cue-action association (McDaniel & Einstein, 2000). Otherwise, detection of the cue is expected to occur relatively automatically, without the allocation of attentional resources to conscious monitoring. The Multiprocess Framework can be linked to PM’s underpinning cognitive domains, with strategic monitoring tied to executive functioning, and automatic detection conceptualised as a spontaneous retrieval process, presumably supported by retrospective memory (McDaniel & Einstein, 2011).

As PM is defined by delaying the execution of an intention whilst completing other tasks, the study of PM has typically involved a classic, computerised paradigm where a PM task is embedded in another, ongoing task (Einstein & McDaniel, 1990). For example, a participant completes a general knowledge quiz or lexical decision task.
At the same time, they are told to respond to a particular cue (e.g. a word from a particular category, a particular syllable, a pre-assigned clock-time) when they become aware of it during the ongoing task. The number of times the participant correctly responds to these cues serves as a measure of their PM ability. Task difficulty can be manipulated by varying the salience or focality of the cue, using just one or many cues, having more or less frequently appearing cues, and changing the time interval between when the PM instructions are given and the appearance of the first cue. Response time data are used to measure whether the inclusion of the PM task changes the speed of the participant’s response to the ongoing task. For instance, increased response time to the ongoing task is taken to indicate that participants are engaging more resources in monitoring for the PM cue, at the expense of responding quickly to the ongoing task.

As well as these computerised paradigms, other approaches measure PM more naturally. These measures do not allow the same degree of experimenter control or calculation of ongoing task costs, but have PM tasks that are more similar to what might be encountered in real-life situations. One example is the Memory for Intentions Screening Test (MIST; Raskin, 2009) which has eight PM tasks mirroring behaviours one might complete in real life, such as self-addressing a postcard or asking to take a break. The task instructions are administered gradually as the participant works on a word search, which they must periodically interrupt to complete the tasks. The tasks vary by cue type (time or event-based) and delay interval (2 or 15 minutes). Another naturalistic PM measure is Virtual Week (Rendell & Craik, 2000), where participants play a board game with squares that simulate the passage of time, and complete various PM tasks at particular locations on the board. Tasks include buying items when out shopping or making phone calls at specific times. One round of the board represents one virtual day with participants required to complete 10 tasks per ‘day’ for 7 virtual days.
In summary, PM can be measured under laboratory conditions, producing fine-grained, sensitive measures that allow a high degree of experimenter control. PM performance can also be assessed under more naturalistic conditions with tasks that mimic real-world PM demands. Both types of measurement have proved important for better understanding the component processes that contribute to PM performance, and plotting the trajectory of PM performance across the lifespan.

**The neurobiology of prospective memory**

As expected, given the multiple components of a PM task, and the other cognitive functions that underpin successful PM, the neurobiology of PM involves a distributed network. Frontal regions play a particularly important role, consistent with accounts of this area being salient for supporting executive functions (Stuss, 2011), which themselves are pivotal for successful PM (Kliegel et al., 2002). The importance of the frontal lobes for PM is underscored by clinical case studies that report an isolated and profound deficit in PM after frontal lobe damage (Bisiacchi, 1996; Cockburn, 1995). Clinical case studies also point to the importance of the medial temporal lobes (Huppert & Beardsall, 1993; Palmer & McDonald, 2000), although neuroimaging data are not so firm on their role (Cona, Scarpazza, Sartori, Moscovitch, & Bisiacchi, 2015). It is suggested that the medial-temporal lobes are involved in PM in so far as retrospective memory is required for encoding and recalling the what of the PM task, rather than controlling the maintenance, monitoring and execution of the PM task (West, 2005).

As well as research emphasising the general importance of the frontal lobes, studies have linked particular brain regions to each of the components of PM, using electrophysiological and neuroimaging data. The initial encoding/planning of the PM task is associated with signals from frontal-polar regions (West & Ross-Munroe, 2002), particularly left anterior prefrontal cortex (Momennejad & Haynes, 2013; Momennejad & Haynes, 2012). This indicates executive control may play a supporting role in
elaborative processing for PM encoding, as it does in retrospective memory (West, 2005). Holding the PM task in mind whilst completing other tasks is associated with activation of bilateral frontal polar and precuneous regions, lateral prefrontal, and dorsal parietal regions (Burgess, Quayle, & Frith, 2001; Cona et al., 2015). A dissociation between medial and lateral prefrontal cortex is reported, where the former shows more activation under conditions of automatic detection of the PM cue, and the latter when strategic monitoring is required, e.g. for a less focal PM cue (Cona et al., 2015; McDaniel & Einstein, 2000). Detection of the PM cue is linked to signals from occipital-parietal cortex (West & Ross-Munroe, 2002). Disengagement from the ongoing task in order to switch to the PM task appears to again be supported by the frontal lobes, and also the anterior cingulate cortex (Cona et al., 2015; West, 2005). Retrieval of the PM intention is linked to activity in ventral parietal cortex and cingulate cortex (Cona et al., 2015). Execution of the PM task is thought to be facilitated by activation of the right thalamus, lateral anterior pre-frontal cortex and deactivation of the right middle frontal gyrus (Burgess et al., 2001; Burgess, Scott, & Frith, 2003; Cona et al., 2015; West, 2005).

Overall, research into the neurobiology of PM is generally supportive of a componential approach to understanding PM, and furthermore suggests that altering PM task conditions (e.g. cue type) can alter the demand on each of these components (e.g. the need for strategic as opposed to automatic monitoring, revealed by dissociation between medial and lateral activation in the prefrontal cortex).

Age-related decline in prospective memory

As reviewed, PM is a multicomponent process that relies on other complex cognitive abilities, such as executive function and retrospective memory. Overall, investigations of age-related PM decline suggest age effects occur to differing degrees dependent on PM task features, broadly consistent with the Multiprocess Framework
These task-feature dependent age effects can also be considered to reflect varying demand on the components of PM, and thus the cognitive domains underpinning these components (Kliegel et al., 2016). Studying age-related PM decline thus not only tells us about how PM ability changes, it also can provide insight into how ageing impacts on the processes underpinning PM, and, importantly, the dynamic integration of these abilities.

The importance of studying age-related PM decline is underscored by noting that PM is identified by older adults as a primary source of their everyday cognitive failures (McDaniel & Einstein, 2007), significantly impacts activities of daily living and quality of life (Woods et al., 2012), and could be used to help predict development of dementia (Salmon, 2011). However, even more important for the people who are the subject of these studies, delineating under what circumstances PM shows age effects can directly inform what interventions may successfully improve PM in older adults.

The presence of age-related decrements in PM ability was initially theorised by Craik (1986) who suggested age differences would be greatest for memory processes relying on self-initiation, as PM does. However, early empirical research revealed an interesting paradox: in the laboratory, older adults did indeed show poorer performance compared to younger adults on PM tasks, but their PM in everyday life was often better than younger adults (Henry, MacLeod, Phillips, & Crawford, 2004). Factors such as motivation and ability to use external aids were hypothesised to account for older adult’s better naturalistic PM performance (Henry et al., 2004).

After identification of the age-PM paradox, research intensified with multiple moderators of age effects on PM being proposed and evaluated. A prominent theme that marries well with the Multiprocess Framework is that the nature of the PM cue determines to what extent age effects are apparent. Specifically, if features of the cue support its automatic detection, then older adults would be likely to perform equally as
well as younger adults. However, if the cue is harder to detect, requiring the participant to consciously monitor the environment, then age effects would increase (McDaniel & Einstein, 2011). As previously mentioned, cue features that make detection harder include: being non-focal (having features unlikely to be processed as part of the ongoing task), being less salient, or being time-based rather than event-based (McDaniel & Einstein, 2000). PM tasks with such cues are indeed more consistently linked to stronger age effects (Einstein, Holland, McDaniel, & Guynn, 1992; Einstein, McDaniel, Richardson, Guynn, & Cunfer, 1995).

The primary explanation put forth to explain the greater age effects seen in PM tasks with harder to detect cues is that older adults have a deficit in strategic monitoring, which is assumed to underlie successful PM performance on these tasks (McDaniel & Einstein, 2000). The operation of strategic monitoring is generally inferred by an increase in costs to the ongoing task. However, strategic monitoring is not the only explanation put forth to explain age effects related to PM cue features. For time-based cues, Park and others (1997) showed smaller costs to the ongoing task than event-based cues, but nevertheless, older adults performed more poorly on the time-based task. Their interpretation is that higher-level attentional control and meta-cognition explain these data better than older adults’ reduced capacity for maintenance, rehearsal or monitoring (McDaniel & Einstein, 2011).

Beyond features of the PM cue, there are numerous other aspects of PM tasks proposed to moderate age effects. Each of these inform conceptions about what aspects of cognition underlie age-related PM decline, and thus how interventions might seek to improve PM in older adults. Older adults’ PM performance declines more than younger adults when their planning time is interrupted, suggesting they rely more heavily on pre-planning for successful PM (McDaniel & Einstein, 2000). Reduced attentional or working memory resources is inferred to underlie older adults’ poorer PM performance.
under circumstances where the ongoing task is more difficult or absorbing (Kidder, Park, Hertzog, & Morrell, 1997; McDaniel & Einstein, 2000; Schnitzspahn, Ihle, Henry, Rendell, & Kliegel, 2011). Older adults also appear more vulnerable to increased demands on the retrospective memory component of PM: their performance is poorer when the delay between instruction and PM cue presentation is increased, when the number of PM cues is increased, and when the required PM action involves more steps (Kidder et al., 1997; McDaniel & Einstein, 2011). Finally, poorer inhibition and switching ability is inferred to explain older adults’ difficulty with PM tasks where order of task accomplishment is pre-specified (Ihle, Hering, Mahy, Bisiacchi, & Kliegel, 2013), and difficulties executing PM tasks despite accurate cue detection and recall of intention (Zimmermann & Meier, 2006).

From these studies, task features that increase age effects in PM could be categorised as falling into two main groups: those that increase retrospective memory demands and those that increase executive function demands. Drawing this distinction can help guide the selection of interventions for PM, by suggesting interventions should support retrospective memory and executive function. Task features that would theoretically increase retrospective memory demands include longer delay intervals, responding to multiple PM cue types, and executing multi-step PM actions. Increased requirements for planning, switching, and inhibition fall into the category of executive functions (Miyake et al., 2000). Strategic monitoring, attentional control and metacognition are also associated with executive function (Fernandez-Duque, Baird, & Posner, 2000; McDaniel & Einstein, 2011). Attentional and working memory capacity (as opposed to control) do not fit so neatly, but have also been linked to executive abilities (McCabe, Roediger, McDaniel, Balota, & Hambrick, 2010). Linking the proposed moderators of age effects in PM to either executive function or retrospective
memory is consistent with definitions of PM that refer to the importance of each of these domains in underpinning PM (Kliegel et al., 2002; West, 2005).

As well as linking task-specific features that increase PM age effects to either executive function or retrospective memory, the degree to which ability in each of these cognitive domains contributes to PM performance can be evaluated. Azzopardi and others (2015) used structural equation modelling to separate variance linked to executive flexibility from that linked to memory/speed in a sample of 197 participants. They concluded that executive flexibility is the primary mediator of age effects on PM. Martin and others (2003) also demonstrated a mediating role of executive function in the relationship between age and PM in a sample of 80 participants. Whilst they did not consider if retrospective memory played a similar role, they found executive function fully mediated age effects in event-based and time-based PM tasks that had a single cue-action pair, and partially mediated age effects in a PM task that had multiple cues and actions. Similarly, Mattli and others (2014) found the executive component of a PM task contributed more to older adults’ PM performance than the retrospective component. Conversely, in a study of habitual, naturalistic PM, Cauvoto (2017) reported that retrospective memory and not executive function significantly contributed to PM performance. Overall, while most studies emphasise the primacy of executive function ability in determining older adults’ PM, some research suggests retrospective memory is also important, particularly for habitual PM. These results could suggest that supporting these two underpinning cognitive domains would be an important way to intervene to improve older adults’ PM.

In summary, the literature on age-related PM decline suggests older adults display more prominent PM deficits compared to younger adults under certain task conditions that increase load on executive function in particular, and also retrospective memory. Interventions that support these underpinning domains may therefore prove a
fruitful way to improve PM functioning amongst older adults, as explored further in Chapter 4 of this thesis.

**Cognitive ageing and sleep**

Looking beyond PM specifically, a current focus of cognitive ageing research is the role that sleep disruption may play in age-related cognitive decline (Zhang, Zhong, Li, & Chang, 2017). Disturbed sleep amongst older adults has been linked to domain specific and global cognitive deficits (Miyata, Noda, Iwamoto, & Kawano, 2013; Song et al., 2015; Spira et al., 2017), and indicators of neurodegeneration (Branger et al., 2016; Lim, Kowgier, Yu, Buchman, & Bennett, 2013). Poor sleep is also specifically implicated in deficits in the domains underpinning PM: executive function and retrospective memory. For example, a link between older adults’ retrospective memory performance, sleep physiology and subsequent hippocampal functioning has been described in Mander et al. (2013) and in Van Der Werf et al. (2009). Similarly, Anderson and Horne (2003) and Lafortune and others (2014) connected older adults’ performance on executive tasks to markers of the restorative action of sleep on the prefrontal cortex. These findings, demonstrating a neural basis for the link between poor sleep and deficits in retrospective memory and executive function, suggest sleep is also an important factor to consider for age-related PM deficits (Scullin et al., 2019). Indeed, outside of an ageing context, naturalistic and experimentally disturbed sleep is linked to poorer PM performance, confirmed in a recent meta-analysis (Leong, Cheng, Chee, & Lo, 2019). Given the high prevalence of sleep disturbance amongst older adults (Bloom et al., 2009), researching the consequences of sleep on older adults’ PM is an important avenue of investigation. Furthermore, if sleep disruption plays a role in age-related PM decline, there may be an opportunity to improve PM by treating sleep problems.

The literature on the potential role of sleep in cognitive ageing, and the effect of sleep disturbance on PM informs Chapters 2 and 3 of this thesis. Together, these
chapters explore sleep as a potential avenue of intervention for PM in older adults by evaluating whether sleep disruption affects older adults’ PM, whether sleep disruption is causally related to age-related PM decline, and whether improving older adults’ sleep can improve their PM. Subsequently, the thesis moves towards evaluating another avenue to improve PM in older adults: a novel executive function intervention for PM, presented in Chapter 4.

**Prospective memory intervention in older adults**

Consistent with the growth in literature on age-related PM decline, there has been increasing focus on designing and evaluating interventions to improve PM performance in older adults. Multiple methods of intervention have been evaluated, consistent with conceptions of PM as a multi-component process. This section reviews the varied approaches used to improve PM in the literature so far. Table 1.1 presents additional information on the samples, design and effect sizes for the studies described in this section. As will be demonstrated, some components of PM have been well targeted by intervention, whereas others have received scant attention.

To structure this review, contrasts are drawn between different intervention approaches. First, methods to improve PM can rely on either *internal* or *external* interventions (Hering, Rendell, Rose, Schnitzspahn, & Kliegel, 2014). External interventions involve outsourcing PM to aids or devices. Internal interventions are participant-driven techniques to improve PM performance. Internal interventions are further divided into those that attempt to remediate PM by repeated practice on the entire PM process, and those that focus on strategies to support a particular component of PM. Additionally, some interventions use a multi-component approach, which may include elements of external interventions, internal interventions that target particular component processes in PM, repeated practice on PM tasks, and psycho-education.
External interventions

External interventions to improve PM involve the provision of equipment that generates reminders to complete PM tasks, mainly electronic devices. The use of such devices is well-researched in the fields of brain injury and neurodegenerative disease (e.g. Thöne-Otto & Walther, 2003). Whilst some studies have reviewed factors to consider when designing such technology for use by older adults (e.g. Caprani, Greaney, & Porter, 2006), there is little research evaluating their stand-alone effectiveness at improving PM in healthy older adults. The use of external devices has, however, been included in multi-component interventions for PM. Insel and colleagues (2016) trained older adults in the use of a medication organiser, alongside other strategies to increase reliance on external cues for PM tasks, resulting in improved medication adherence, although not sustained at follow-up. Schmidt, Berg, and Deelman (2001) incorporated training in the use of an agenda into their intervention which primarily utilised implementation intentions (discussed below), resulting in improved subjective PM, and a small improvement in objectively measured PM, which again did not persist at follow-up. Whilst firm conclusions cannot be drawn from these multi-component studies, a potential issue in the use of external interventions is the long-term sustainability of their use, given the ongoing effort required in, for example, programming an electronic agenda.

Internal interventions

As highlighted, internal interventions to improve PM involve either attempted remediation of PM deficits by repeated practice on the entire PM process, or strategies to support a particular component of PM. Some researchers have used multi-component approaches that combine the above elements, and may also include external interventions and psycho-education.
Remediation interventions

One method used to facilitate repeated practice of PM is the Virtual Week paradigm (Rendell & Craik, 2000). Rose and colleagues (2015) took this approach with a group of healthy older adults who practiced the Virtual Week as a computer game for 12, one-hour sessions, with increasing difficulty and feedback on performance. In comparison to a control group, they found improvement in performance on the Virtual Week task over the course of the training, and on a naturalistic PM task (phoning the experimenter). However, performance on a laboratory-based PM task did not improve. Perhaps due to concerns raised about the generalisability of the remediation approach (Hering et al., 2014; Waldum, Dufault, & McDaniel, 2016), the majority of other PM intervention studies use strategies to support a particular theoretically defined component of PM.

Strategy Interventions

As described earlier, the components of a PM task can be broadly categorised into those that rely on retrospective memory, and those that rely on executive functions and associated processes. This distinction can also be used to group strategy oriented intervention approaches for PM. The most well-established intervention for PM supports the retrospective memory component using a strategy known as implementation intentions (Chen et al., 2015).

Implementation intentions

Implementation intentions involve forming and rehearsing a verbal statement and/or visualisation that makes explicit the link between a PM task and a cue. For example, a person may form and repeat the statement, “when the evening news starts [cue], then I will take my medication [task]”, and visualise this taking place. Implementation intentions are assumed to strengthen the mnemonic link between cue and PM task, supporting encoding and retrieval and thus improving older adults’ PM
performance by compensating for retrospective memory decline (Chen et al., 2015; McFarland & Glisky, 2011). Whilst this is the primary mechanism proposed to account for the effects of implementation intentions, within a Multiprocess Framework, implementation intentions are thought to increase the likelihood of spontaneous retrieval, thus reducing reliance on strategic monitoring, at which older adults are thought to have a deficit (McDaniel & Scullin, 2010). As such, implementation intentions could also be considered a strategy targeted at an executive component of PM (strategic monitoring). This highlights that, whilst useful, an absolute distinction between retrospective interventions and executive interventions may be somewhat of an oversimplification. Regardless, meta-analysis has demonstrated strong positive effects for implementation intentions at improving PM performance in older adults, assessed by laboratory-based and naturalistic tasks (Chen et al., 2015). Implementation intentions – used as part of multi-component interventions – have also been shown to improve self-reported PM in daily life (Burkard, Rochat, Blum, et al., 2014; Farzin, Ibrahim, Madon, & Basri, 2018).

Some researchers have assumed that implementation intentions are most suited for focal, event-based PM tasks (Foster, McDaniel, & Rendell, 2017; Waldum et al., 2016). However, recent findings have demonstrated they can be effective for other types of PM tasks. Whilst implementation intentions do not easily lend themselves to time-based PM tasks, some researchers have taken the approach of training older adults to first transform a time-based task to an event-based PM task, and then to apply implementation intentions (Insel et al., 2016). In regards to non-focal versus focal PM tasks, Chen (2015) concluded that implementation intentions were equally effective for both amongst younger adults. Fewer studies assess effects on non-focal tasks amongst older adults, although results are again mostly positive. Two studies report positive effects for non-focal tasks (Chasteen, Park, & Schwarz, 2001; Zimmermann & Meier, 2006), and another suggests they are generally effective for non-focal tasks amongst
older adults, except for the most elderly (Schnitzspahn & Kliegel, 2009). However, one study found implementation intentions were not effective for older adults completing non-focal PM tasks (Lee, Shelton, Scullin, & McDaniel, 2016).

Several studies have assessed whether implementation intentions are effective for all older adults, or whether pre-existing differences in cognitive ability affect their success. However, no clear consensus has emerged, partially due to the variety of cognitive domains assessed. With regards to general cognition, one study concluded that participants with lower baseline ability demonstrate greater gains from intervention (Brom et al., 2014), whilst another concluded the opposite (Schnitzspahn & Kliegel, 2009). When executive function and working memory are considered, two studies report lower baseline ability predicts greater gains in PM performance following intervention (Brom & Kliegel, 2014; Insel et al., 2016), and another reported equal benefit across high and low ability groups (McFarland & Glisky, 2011). However, one study found participants with lower working memory ability did not respond to implementation intentions (Burkard, Rochat, Juillerat Van der Linden, Gold, & Van der Linden, 2014). Finally, a study examining baseline differences in episodic memory reported an equivalent response to implementation intentions across high and low ability groups (Shelton et al., 2016).

In summary, implementation intentions are well established as a PM intervention for older adults. They have the most evidence for use with focal event-based PM tasks, but can also be effective for non-focal tasks, and time-based tasks with adaptations. Some research suggests implementation intentions may be less effective for older adults with poorer baseline cognitive ability (Burkard, Rochat, Juillerat Van der Linden, et al., 2014; Schnitzspahn & Kliegel, 2009). However, other studies find older adults with poorer ability have more to gain from implementation intentions or at least
Executive function strategies

In comparison with the large body of literature examining the effectiveness of implementation intentions as a strategy to improve the retrospective component of PM, few researchers have targeted intervention to the executive components of PM (although it is acknowledged that by some accounts, implementation intentions also has an executive component; McDaniel & Scullin, 2010). This is surprising, given the primacy given to executive function in explaining age-related PM deficits (Azzopardi et al., 2015; Kliegel et al., 2002; Mattli et al., 2014). The few studies that do test executive interventions all use different approaches, and so no single executive intervention is well established for improving PM in older adults.

In a study targeting planning ability, Kliegel and colleagues (2007) asked participants to plan aloud, gave them prompts as to what they might include in their plans, and provided a visual planning scheme for participants to complete. They found older adults who received the planning intervention performed better on a complex, multi-step laboratory-based PM task.

Another study targeted task-switching with the aim of improving older adults’ ability to successfully switch from the ongoing task to the PM task (Brom & Kliegel, 2014). Participants in the task-switching condition practiced a 15-25 minute self-paced task-switching training program daily for five days. Participants were then evaluated as to how often they remembered to perform a naturalistic PM task (testing their blood pressure at home). Task-switching training did not improve PM performance, in comparison to participants in the same study who received implementation intentions training.
Waldum and colleagues (2016) trained strategic monitoring as part of their multi-component PM intervention. Participants took part in eight weekly training sessions, which included prompts, practice, discussion and feedback to support strategic monitoring in non-focal and time-based PM tasks. Participants were concurrently enrolled in a retrospective memory and attention training intervention program. Whilst improvements were seen in time-based (but not event-based) PM on laboratory-based tasks administered at the end of the intervention, the additional attention and memory training limits the degree to which effects can be attributed to training in strategic monitoring.

In summary, two studies reported training in planning and monitoring improved PM performance, whereas another reported that training in task-switching did not appear to be effective at improving PM. The planning and monitoring studies both administered training within the PM task environment, suggesting this may improve generalisability. Given the emphasis on executive function in explaining age-related PM decline, future research should continue to evaluate executive function interventions for PM. Potential new interventions should be directly compared to the more established retrospective intervention – implementation intentions – in order to ascertain where resources for the application of PM interventions are best directed. Moving towards the evaluation of the effectiveness of PM interventions with naturalistic PM measures will also be important to demonstrate their generalisability and efficacy at improving everyday PM functioning.

**Multi-component interventions**

Several studies have sought to increase the effectiveness of strategy interventions for PM by including additional components. These studies all combined implementation intentions with other elements. However, only one study compared the effectiveness of the components alone and together (Brom & Kliegel, 2014). It is thus
difficult to conclude how much the additional elements add to the effectiveness of implementation intentions.

As reviewed in the above section on external interventions, two studies combined implementation intentions with an external intervention (a medication organiser in Insel et al., 2016; an agenda in Schmidt et al., 2001). Additional training was then provided to teach participants to shift from using self-initiation to externally driven cues for PM tasks. Both studies reported the training was effective, but this was not sustained at follow-up.

Farzin and colleagues (2018) combined implementation intentions with repeated practice on the Virtual Week paradigm in weekly, two-hour sessions for six weeks. The training resulted in significant improvements in subjective and objective, laboratory-based PM, sustained at follow-up.

Brom and Kliegel (2014) combined implementation intentions with an at-home task-switching training program, as discussed above. They not only evaluated the effects of the combined training, but also the implementation intention and task-switching elements individually, and concluded that only implementation intentions were effective.

Finally, Waldum and colleagues (2016) combined implementation intentions with training in strategic monitoring and clock checking. Participants received education on the strategies and when to use them, practiced them in PM tasks, and discussed their use in daily life over eight weekly sessions, as discussed above. Interestingly, improvements were seen in time-based PM (targeted by strategic monitoring) but not event-based PM (targeted by implementation intentions). As mentioned, findings from this study should be interpreted cautiously due to participants’ concurrent enrolment in other cognitive training programs.
Overall, multi-component interventions generally report success in improving PM. However, because all the studies included implementation intentions – which already has a large evidence base – and rarely evaluated the individual effectiveness of each element, it is not clear how much the additional components add. Direct comparison with studies using implementation intentions alone is also complicated by these studies use of multiple-treatment sessions, whereas stand-alone implementation intentions interventions are typically delivered in a single session. Future research into PM interventions should compare the effectiveness of different interventions to determine how best to ameliorate age-related PM decline. This is the approach taken in Chapter 4 of this thesis, which compares implementation intentions with an executive intervention to improve PM not yet tested amongst older adults: goal management training.
Table 1.1.

Details on select studies examining interventions for prospective memory decline amongst older adults.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Topic</th>
<th>Sample size</th>
<th>Design**</th>
<th>Effect size***</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brom et al., 2014.</td>
<td>Response to II moderated by cognitive ability.</td>
<td>N = 39</td>
<td>Single time point. Treatment vs. control receiving standard instructions. Randomisation specified.</td>
<td>$\eta^2_p = 0.31$. Interaction with WM: $\eta^2_p = 0.12$</td>
</tr>
<tr>
<td>Brom and Kliegel, 2014.</td>
<td>Response to II moderated by cognitive ability.</td>
<td>N = 62</td>
<td>Single time point. Both treatments (switching and II) compared individually to standard instruction control and to combined switching plus II group.</td>
<td>$\eta^2_p = 0.08$. Switching not effective. Switching as a moderator: high ability $D = .28 (p &gt; 0.05)$; Low ability $D = 1.44$.</td>
</tr>
<tr>
<td>Burkard, Rochat, Blum et al., 2014.</td>
<td>Response to II moderated by cognitive ability.</td>
<td>N = 45</td>
<td>Single time point. Treatment vs. control receiving standard instructions. Randomisation specified.</td>
<td>Treatment condition by working memory ability: $\eta^2_p = 0.134$</td>
</tr>
<tr>
<td>Chen et al., 2015.</td>
<td>Meta-analysis of laboratory studies on II.</td>
<td>K = 10, N = 468 (224 treatment, 244 control)</td>
<td>Meta analysis</td>
<td>$D = 0.68$</td>
</tr>
<tr>
<td>Kliegel et al., 2007.</td>
<td>Executive function interventions: Planning.</td>
<td>N = 30 (15 control, 15 treatment)</td>
<td>Single time point. Treatment vs. control receiving standard instructions</td>
<td></td>
</tr>
<tr>
<td>Authors</td>
<td>Topic</td>
<td>Sample size*</td>
<td>Design**</td>
<td>Effect size***</td>
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<tr>
<td>McFarland and Glisky, 2011.</td>
<td>Response to II moderated by cognitive ability.</td>
<td>N = 32</td>
<td>Single time point. Treatment vs. control receiving standard instructions</td>
<td>( \eta^2_p = 0.29 ) for whole group.</td>
</tr>
<tr>
<td>Rose et al., 2015.</td>
<td>Remediation intervention.</td>
<td>N = 59</td>
<td>Repeated measures. Treatment vs. active and no contact control groups collapsed together.</td>
<td>Not reported</td>
</tr>
<tr>
<td>Schmidt, Berg, and Deelman, 2001.</td>
<td>II with external interventions.</td>
<td>N = 65</td>
<td>Repeated measures. Treatment vs. active and no contact control groups collapsed together.</td>
<td>( B = 0.36 )</td>
</tr>
<tr>
<td>Schnitzspahn and Kliegel, 2009.</td>
<td>II for non-focal PM. Response to II moderated by cognitive ability.</td>
<td>N = 71</td>
<td>Single time point. Treatment vs. control receiving standard instructions.</td>
<td>Event based: ( r = -0.32 ) (young old); ( r = -0.35 ) (old old) Time based: ( r = -0.36 ) (young old); ( r = -0.05 ) (old old) ( \eta^2_p = 0.15 )</td>
</tr>
<tr>
<td>Shelton et al., 2016.</td>
<td>Response to II moderated by cognitive ability.</td>
<td>N = 72</td>
<td>Repeated measures. Treatment vs. control receiving standard instructions. Randomisation specified.</td>
<td>( \eta^2_p = 0.15 )</td>
</tr>
<tr>
<td>Waldum et al., 2016.</td>
<td>Executive function interventions: Strategic monitoring.</td>
<td>N = 37</td>
<td>Repeated measures. Treatment vs. no contact control.</td>
<td>Not reported</td>
</tr>
<tr>
<td>Zimmermann and Meier, 2006.</td>
<td>II for non-focal PM.</td>
<td>N = 185</td>
<td>Single time point. Treatment vs. control receiving standard instructions.</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

*Sample size breakdown by group given when provided in article.

**Random allocation to group indicated by 'randomisation specified' where explicitly stated so in article.

***All effect sizes come from statistically significant (\( p < 0.05 \)) statistics unless indicated.

II: implementation intentions; WM: working memory; PM: prospective memory; AD: Alzheimer’s disease.
The current thesis

The above sections have provided an overview of PM, discussed age-related decline in PM, and considered approaches taken so far to improve PM in older adults. This review has suggested that sleep and executive components are two underexplored potential avenues to improve PM.

In the cognitive ageing literature, sleep has emerged as an important determinant of older adults’ cognitive ability (Miyata et al., 2013; Song et al., 2015; Spira et al., 2017). Outside of an ageing context, sleep is also important for younger adults’ PM (Leong et al., 2019). However, no research has yet considered whether sleep disruption is related to PM performance in older adults, and, pivotally, whether improving sleep can improve PM in older adults. Chapters 2 and 3 of this thesis address these questions. In Chapter 2, associations between older adults’ objectively measured habitual sleep and event-based PM performance were examined. Hypotheses were that greater sleep disturbance would be related to poorer PM, and that greater sleep disturbance would mediate the relationship between older age and poorer PM. Chapter 3 built on the results of Chapter 2 with a pilot study investigating whether an intervention to improve older adults’ objectively measured sleep could also improve PM. Predictions were that reduced sleep disturbance following a course of cognitive behavioural therapy for insomnia (CBT-I) would be associated with improvements in PM on a comprehensive lab-based task.

Within the realm of more typical cognitive interventions for PM, there are also gaps in the literature. Specifically, research into age-related PM decline has emphasised the primacy of executive function in determining older adults’ PM performance (Azzopardi et al., 2015; Kliegel et al., 2002; Mattli et al., 2014). Indeed, decline in older adults’ PM is often conceptualised as decline in the executive aspects of PM specifically
(McDaniel & Einstein, 2011). However, interventions to improve PM in older adults have mostly ignored the executive components of PM in favour of focusing almost exclusively on strategies to support the retrospective components of PM. Thus, more research is needed to evaluate interventions that support the executive components of PM, and to do so in comparison to the most well-established intervention for PM: implementation intentions (Chen et al., 2015). This is the direction taken in Chapter 4 in this thesis. Older adults had their PM assessed, then received either an executive intervention (goal management training), implementation intentions or no intervention. PM was then re-assessed and associations between the intervention conditions and improvement in PM were explored. It was predicted that both goal management training and implementation intentions would improve PM, with goal management training being expected to show a benefit over implementation intentions.

This thesis concludes with a summary and general discussion of the overall findings of the studies. Limitations and future directions for research are provided.
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Foreword to Chapter 2.

The preceding chapter has highlighted that sleep disturbance affects prospective memory function in younger adults, and may be implicated in age-related cognitive decline generally. Thus, the following chapter examines whether sleep disturbance is associated with poorer prospective memory in older adults, and whether sleep disturbance is implicated in age-related prospective memory decline. In examining what role sleep plays in older adults’ prospective memory, this chapter contributes to the broader aim of this thesis: exploring potential avenues to intervention for prospective memory in older adults. Specifically, the findings of this chapter help inform whether addressing sleep problems is a way to potentially improve prospective memory in older adults. This chapter was written for and published as a manuscript in a peer-reviewed journal:


The manuscript has been altered to fit the format of the thesis.
CHAPTER 2

Sleep Disruption Explains Age-Related Prospective Memory Deficits: Implications for Cognitive Ageing and Intervention
Abstract

The high prevalence of sleep disruption amongst older adults may have implications for cognitive ageing, particularly for higher-order aspects of cognition. One domain where sleep disruption may contribute to age-related deficits is prospective memory – the ability to remember to perform deferred actions at the appropriate time in the future. Community-dwelling older adults (55-93yrs, N = 133) undertook assessment of sleep using actigraphy and participated in a laboratory-based prospective memory task. After controlling for education, sleep disruption (longer awakenings) was associated with poorer prospective memory. Additionally, longer awakenings mediated the relationship between older age and poorer prospective memory. Other metrics of sleep disruption, including sleep efficiency and wake after sleep onset, were not related to prospective memory, suggesting that examining the features of individual wake episodes rather than total wake time may help clarify relationships between sleep and cognition. The mediating role of awakening length was partially a function of greater depression and poorer executive function (shifting), but not retrospective memory. This study is among the first to examine the association between objectively-measured sleep and prospective memory in older adults. Further, this study is novel in suggesting sleep disruption might contribute to age-related prospective memory deficits; perhaps, with implications for cognitive ageing more broadly. Our results suggest there may be opportunities to prevent prospective memory decline by treating sleep problems.
Introduction

Prospective memory is the ability to remember to perform a deferred action at the appropriate time in the future. It is vital for independent living and well-being (Woods, Weinborn, Velnoweth, Rooney, & Bucks, 2012), yet declines with age (Kliegel et al., 2016). Identifying factors contributing to PM deficits may help preserve older adults’ independence. One potential factor is sleep disruption.

Sleep and cognition in older adults

Older adults are more likely than other age groups to experience disturbed sleep (Bloom et al., 2009). This increased sleep disruption is likely attributable to both age-dependent changes in circadian rhythm and sleep architecture, and also higher rates of sleep pathology such as insomnia and sleep apnoea (Bloom et al., 2009). Consequently, older adults often have reduced total sleep time (TST) and sleep efficiency (TST as a proportion of time in bed), take longer to fall asleep (sleep latency) and have increased wake after sleep onset (WASO; Vitiello, 2009). Findings of increased WASO presumably reflect more frequent and longer awakenings (Vitiello, 2009), however WASO does not on its own indicate the duration of individual wake episodes, only total wake time over the entire night (Lim, Kowgier, Yu, Buchman, & Bennett, 2013). Previous research has typically overlooked the features of individual wake episodes (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004b).

Sleep disruption in older adults is associated with poorer performance in multiple cognitive domains and an increased risk of cognitive decline (Yaffe, Falvey, & Hoang, 2014). However, consensus on the nature of this association has been complicated by variability in the ways that sleep and cognition are measured (for reviews see Brewster, Varrasse, & Rowe, 2015; Holanda Júnior & de Almondes, 2016). Whilst some studies test individual cognitive domains such as processing speed,
memory or executive function (e.g. Miyata, Noda, Iwamoto, & Kawano, 2013; Spira et al., 2017), others examine global cognition with screening tests (Blackwell et al., 2014; Song et al., 2015) or indicators of neurodegenerative disease (Branger et al., 2016; Lim et al., 2013). Assessment of sleep is similarly diverse and includes various combinations of TST, WASO, sleep latency, sleep efficiency, night-to-night variability, time in various sleep stages and subjective quality (Brewster et al., 2015). In large-scale community studies of older adults, the focus has generally been on the duration of wake and sleep, and whilst the overall picture supports a sleep-cognition association, both significant and non-significant findings are reported (Blackwell et al., 2011, 2006; Lambiase, Gabriel, Kuller, & Matthews, 2018; Miyata et al., 2013; Spira et al., 2017).

Fewer studies have assessed the frequency and average duration of individual wake episodes, which may better capture potential interference with slow-wave/rapid eye movement (REM) sleep in large-scale settings where polysomnography is not feasible (Blackwell et al., 2014; Lim et al., 2013).

The association between sleep disturbance and poorer cognition in older adults could help explain age-related PM deficits. Alternatively, concurrent difficulties with sleep and cognition could simply be a function of general decline across multiple aspects of health with advancing age. Regardless, the high incidence of sleep problems amongst older adults and the importance of PM functioning for independent living underscore the need to understand if and how sleep disruption impacts PM.

**Sleep, depression and the cognitive processes underpinning prospective memory**

PM is underpinned by retrospective memory and executive function – particularly the ability to shift between tasks (Kliegel et al., 2016). Retrospective memory and task shifting are both affected by poor sleep (Waters & Bucks, 2011). These particular sleep-related cognitive deficits are linked to hippocampal and prefrontal functioning, and possibly reflect an interference with sleep-dependent neuro-
restorative processes (Anderson & Horne, 2003; Lafortune et al., 2014). If sleep disruption interferes with the restorative processes supporting neural function generally, and retrospective memory and shifting in particular, then PM is likely to be similarly impacted.

Depression and sleep problems are associated in older adults, with sleep disruption being both a symptom of depression, and possibly also a cause (Roberts, Shem, Kaplan, & Strawbridge, 2000). Depression is linked to deficits in retrospective memory, executive functions (including shifting) and PM (Austin, Mitchell, & Goodwin, 2001; Zhou et al., 2017), suggesting sleep disruption may also affect PM via an association with depression.

**Sleep and prospective memory: previous research**

Although the effect of sleep disruption on PM has not yet been investigated in older adults, previous research supports a relationship between sleep and PM in younger adults. In both laboratory-based and more naturalistic tasks, experimentally-induced sleep deprivation has been found negatively to impact PM performance (Diekelmann, Wilhelm, Wagner, & Born, 2013a, 2013b; Esposito, Occhionero, & Cicogna, 2015; Grundgeiger, Bayen, & Horn, 2014). Two studies have examined the impact of sleep disruption on PM using actigraphs (wrist-worn devices that monitor motor activity to assess sleep over multiple nights) and a single-item PM task: remembering to press the actigraph button at bedtime. Such PM tasks are considered “event-based”, in contrast to “time-based” tasks where the PM cue is a particular clock time. In the first study, “bad” sleepers in a mixed-aged sample performed less well on the PM task (Fabbri, Tonetti, Martoni, & Natale, 2014). However, the second study found sleep did not predict PM in older adults (Cavuoto et al., 2016). The effect of sleep disruption on PM is, thus, not yet clear, with previous research limited by use of single-item PM tasks which provide limited data and associated sensitivity for detecting deficits (Einstein et al., 2005).
Previous research has also not considered the role that mood and cognitive factors that support PM (e.g., retrospective memory, shifting) may play in the potential impact of sleep on PM. Clarification of this relationship and underpinning psychological mechanisms is particularly relevant for older adults, who commonly suffer sleep disruption and for whom PM functioning is critical.

**Aims and hypotheses**

The current study aimed to determine the relationship between sleep and PM in a sample of community-dwelling older adults. Participants had their sleep measured with actigraphy and completed an Einstein and McDaniel (1990) event-based PM task, generating multiple PM data points. We hypothesised that less sleep (decreased TST, sleep efficiency) and more wakefulness (increased WASO, awakening length, awakening number) would be associated with poorer event-based PM.

Secondly, drawing on potential links between older adults’ sleep problems and cognitive ageing, this study tested whether sleep disruption mediated age-related PM deficits. We hypothesised that, consistent with the extant literature, older age would be related to poorer PM, but this relationship would be partially or fully mediated by greater sleep disruption (less sleep and more wakefulness as described above).

Finally, we assessed the role of performance in other cognitive domains and severity of depression in the potential relationships between age, sleep and PM. We hypothesised that the proposed mediating role of sleep disruption in the relationship between age and PM would, in turn, be partially a function of poorer retrospective memory/shifting and greater depression.

**Method**

**Participants**

Community dwelling older adults \((N = 147)\) aged 50 and above were recruited to the Healthy Ageing Research Program at the University of Western Australia,
through the West Australian Participant Pool, and the Western Australia Memory Study conducted at the Australian Alzheimer’s Research Foundation. Participants who reported severe neurological or psychiatric conditions (e.g. Parkinson’s disease, stroke, schizophrenia), previous loss of consciousness over 30 minutes, concerning levels of alcohol use (>15 on the Alcohol Use Disorders Identification Test [AUDIT]; Babor, Higgins-Biddle, Saunders, Monteiro, & World Health Organization, 2001) or frequent benzodiazepine use (>3 times week), were excluded from the analysis. Participants were also excluded if they achieved a score below age/education defined cut-offs on the Mini-Mental State Examination (MMSE; Folstein, Folstein & McHugh, 1975; Iverson, 1998) or if their performance on the comprehensive neuropsychological battery raised concerns about early dementia/ MCI (based on the McKhann et al., 2011 criteria). After applying these criteria, 133 participants were included in the analysis. All were fluent English speakers.

Procedure

Participants first attended a short visit to be consented and receive questionnaires and sleep assessment equipment. Sleep equipment use ranged from 6 to 11 days, with 95% of participants using the equipment for 7 nights. Participants returned for a cognitive assessment immediately following the sleep assessment, where a comprehensive battery of neuropsychological battery assessing memory, attention, processing speed, visual-spatial skills, language and executive functions was completed at the participants’ preferred time of day, with multiple breaks to prevent fatigue, taking on average 3.5 hours (see General Methods in Supplementary Materials).

Sleep assessment

Participants were provided with an actigraph (Tri-axial wGT3X-BT activity monitor, Actigraph LLC, FL, USA) and a diary to record bed (when they started trying to sleep) and rise time (when they woke for the last time; Carney et al., 2012). Further
details on actigraphy are provided in the General Methods in Supplementary Materials. Actigraphy data were autoscored using the Cole-Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992) and Actilife software (version 6.11.8). Autoscored data were visually inspected for problematic non-wear time and concordance with the diary. Nights with non-wear time during the rest period or within 5 minutes of bed/rise time were excluded from the analysis (1% of nights were affected by non-wear time). Autoscored bed and rise times were adjusted to match the diary or, if there was a discrepancy of more than 60 minutes, visual manual scoring was used, taking into account the diary and typical movement patterns of bed/rise time. Visual manual scoring was also used when diary times were missing, and in total 8% of nights required visual manual scoring. Once the rest period was thus defined, TST, number and length of awakenings per night, WASO and sleep efficiency were calculated for each night and averaged for each participant to serve as measures of sleep disruption.

**Prospective memory task**

PM was assessed with a typical, laboratory PM paradigm (Einstein & McDaniel, 1990) where an event-based PM task was embedded in an ongoing word categorization task. The task was presented on a standard computer with E-Prime 2.0 Software (Psychology Software Tools, Pittsburgh, PA), and responses made on the keyboard.

The ongoing word categorisation task required participants to indicate quickly and accurately whether a lower-case word on the left of the screen fitted a category defined by an upper-case word on the right. When the word was a match (e.g. eagle BIRD) participants were required to press the “L” key, marked with a “Y”. When the word was not a match (e.g. owl FURNITURE), participants were required to press the “A” key, marked with an “N”.

The PM task required participants to press an alternate key – the “6” key marked with an “X”– whenever they saw the syllable “tor” presented in the lower case word on
the left (e.g. tornado, motorcycle). This phonological PM cue was “non-focal” to the ongoing categorization task because detecting the PM cue is not part of the information required to make ongoing word categorisations. Previous research suggests non-focal PM tasks are particularly sensitive to age-related PM declines (Kliegel et al., 2016).

The word pairs were created from an updated version of the Battig and Montagne (1969) category norms (Van Overschelde, Rawson, & Dunlosky, 2004). The task consisted of four blocks, the first with 50 matching and 50 non-matching word pairs, the last three each with 100 matching and 100 non-matching word pairs. The word pairs were randomly presented within blocks. The first block measured baseline word categorisation. The three subsequent blocks each contained 8 words with the PM cue, half paired with a matching, and half with a non-matching category word. PM cues were presented randomly within every 25 trials, were never presented in the first 5 trials, and always had at least 2 categorisation trials between them. Each block began with the presentation of a blank screen for 250ms, a fixation cross for 250ms, and then the first word pair. Subsequent word pairs were presented after the participant made their response, or after 5000ms.

Participants received computerised instructions for the word categorization task, completed practice trials with feedback, a distractor Sudoku puzzle and then the first block. Next, participants were given instructions for the PM task, completed more practice items with a PM cue, and then the three PM blocks. In between each block, participants were asked to repeat the instructions to the experimenter (to confirm their understanding), given a one-minute break, and asked to work on a new Sudoku puzzle to prevent rehearsal of the PM instructions.

A PM response was scored as correct if it was made on the trial containing a PM cue. PM accuracy was calculated as the proportion of correct responses to PM cues.
across the three PM blocks. Ongoing task accuracy was calculated as the proportion of correct responses to word categorisation trials across the four blocks.

**Questionnaires**

To determine inclusion as described in the *Participants* section, participants completed questionnaires on their demographics (including education in total years completed [missing *N* = 2]), medical history, and alcohol use (using the AUDIT; Babor et al., 2001). To assess probable sleep disorders, participants completed the Insomnia Severity Index (ISI; >14 = clinically significant symptoms; missing *N* = 15; Bastien, Vallieres, & Morin, 2001) and the Berlin Questionnaire measuring Obstructive Sleep Apnoea (OSA) risk (Netzer, Strohs, Netzer, Clark -, & Strohl, 1999). Participants with high OSA risk or who reported an OSA diagnosis (*N* = 12) were deemed to have a positive OSA status.

Participants also completed the Patient Health Questionnaire (PHQ-9; >9 = clinically significant symptoms; Kroenke & Spitzer, 2002) to measure depression severity. When used in the mediation analysis, the PHQ total score was calculated without the question pertaining to sleep problems and was used as a continuous variable. Descriptive reports of depression rates in the sample were based on the unaltered PHQ-9 score.

**Additional cognitive measures**

Verbal and visual retrospective memory was measured with the Delayed Memory Index (*M*±*SD*100±15) of the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; missing *N* = 3; Randolph, Tierney, Mohr, & Chase, 1998). This index score is adjusted for age with higher scores indicating better memory.

Shifting was measured with the difference in completion time between Trail Making Test A and B (Trails B score subtract Trails A score; missing *N* = 2; Reitan &
Wolfson, 1985). When used in the mediation analysis, scores were adjusted for age by converting to Z-scores using normative data for older adults (Tombough in Strauss, Sherman, & Spreen, 2006), and then reflected so that positive scores indicate better performance. Raw difference scores are reported in the sample descriptives (Table 2.2.).

**Statistical analysis**

Conditional Process Analysis for SPSS (Hayes, 2013) was used to test hypotheses about the association between sleep and PM, and the mediating role of sleep, other cognitive domains, and depression in the relationship between age and PM. When examining sleep disruption, we were mindful of the need to capture both awakenings and duration whilst also avoiding problems with multi-collinearity (Thompson & Borrello, 1985). As sleep efficiency, WASO, and awakening number were highly correlated in our sample (Table 2.1) we felt it was not appropriate to include them in the model concurrently. Out of these three variables, we elected to include awakening number, along with TST and awakening length in our primary analysis, as our main concern was to capture best the pattern of awakenings, rather than solely wake time. However, for completeness we also ran additional analyses substituting WASO and sleep efficiency for awakening number (reported in the supplementary materials, pp. 162). Education was included as a covariate to control for premorbid cognitive ability. Statistical significance was set at 0.05 (two-tailed), and 5000 bootstrapped samples, with bias-corrected 95% confidence intervals (CI). Direct and indirect effects were calculated using a series of ordinary least squares regressions and the bootstrapping procedure. The indirect effect represents the degree to which the predictor (age) is related to the outcome (PM) via its impact on the mediator or serial mediators (sleep variables, retrospective memory, shifting and depression). Each indirect effect is considered significant when the CI does not cross zero.
Table 2.1.

Simple bivariate correlations between sleep variables using 5000 bootstrapped samples.

<table>
<thead>
<tr>
<th></th>
<th>Sleep Efficiency</th>
<th>TST</th>
<th>WASO</th>
<th>Awakening Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Awakening Length</td>
<td>-.32‡</td>
<td>.08</td>
<td>.36‡</td>
<td>-.23‡</td>
</tr>
<tr>
<td>Awakening Number</td>
<td>-.74‡</td>
<td>-.03</td>
<td>.75‡</td>
<td></td>
</tr>
<tr>
<td>WASO</td>
<td>-.96‡</td>
<td>-.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TST</td>
<td>.28‡</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: ‡ p < 0.01; WASO: wake after sleep onset; TST: total sleep time.

To test the validity of our sleep measure, we assessed whether actigraphy variables differed between participants with and without probable sleep disorders. A series of independent t-tests with 5000 bootstrapped samples were run comparing awakening length, awakening number, TST, WASO and sleep efficiency between participants with positive or negative OSA status, and with or without clinically significant insomnia symptoms.

Results

Sample characteristics are presented in Table 2.2. As shown in Table 2.3, participants with a positive OSA status had significantly poorer sleep efficiency (t (131) = -2.33, p < 0.05, Cohen’s d = 0.45), longer WASO (t (131) = 2.96, p < 0.01, d = 0.58), and longer awakenings (t (131) = 2.32, p < 0.05, d = 0.46). Participants with clinically significant insomnia symptoms (score >14 on the ISI) also had poorer sleep efficiency (t (116) = -2.20, p < 0.05, d = 0.85) and longer WASO (t (116) = 2.50, p < 0.05, d = 0.96).
Table 2.2.

Demographic, cognitive and sleep characteristics of the sample.

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Proportion</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>133</td>
<td>40% men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>131</td>
<td></td>
<td>13.88 (3.57)</td>
<td>3 – 27</td>
</tr>
<tr>
<td>Age (years)</td>
<td>133</td>
<td></td>
<td>71.71 (7.68)</td>
<td>55 – 93</td>
</tr>
<tr>
<td>Depression (PHQ-9 Score)</td>
<td>133</td>
<td>4%*</td>
<td>2.38 (2.83)</td>
<td>0 – 13</td>
</tr>
</tbody>
</table>

Cognitive measures

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MMSE</td>
<td>133</td>
<td>27.87 (1.56)</td>
<td>24 – 30</td>
<td></td>
</tr>
<tr>
<td>Trails B – Trails A (seconds)</td>
<td>131</td>
<td>5%*</td>
<td>45.41 (26.68)</td>
<td>4.36 – 166.44</td>
</tr>
<tr>
<td>RBANS Delayed Memory Index</td>
<td>130</td>
<td>1%*</td>
<td>102.21 (11.84)</td>
<td>60 – 126</td>
</tr>
<tr>
<td>PM Accuracy (%)</td>
<td>133</td>
<td>64.55 (25.59)</td>
<td>4.33 – 100</td>
<td></td>
</tr>
<tr>
<td>Ongoing Task Accuracy (%)</td>
<td>133</td>
<td>96.85 (1.87)</td>
<td>84.33 – 99.33</td>
<td></td>
</tr>
</tbody>
</table>

Sleep measures

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>OSA status</td>
<td>133</td>
<td>26%</td>
<td>positive</td>
<td></td>
</tr>
<tr>
<td>Insomnia symptoms (ISI score)</td>
<td>118</td>
<td>8%*</td>
<td>6.24 (5.11)</td>
<td>0 – 20</td>
</tr>
<tr>
<td>Awakening Length (minutes)</td>
<td>133</td>
<td></td>
<td>3.49 (1.27)</td>
<td>1.27 – 7.99</td>
</tr>
<tr>
<td>Awakening Number (per night)</td>
<td>133</td>
<td></td>
<td>12.17 (5.77)</td>
<td>2.57 – 29</td>
</tr>
<tr>
<td>WASO (minutes)</td>
<td>133</td>
<td>40.42 (20.47)</td>
<td>4.57 – 117.14</td>
<td></td>
</tr>
<tr>
<td>TST (minutes)</td>
<td>133</td>
<td>413.68 (48.15)</td>
<td>236.50 – 556.67</td>
<td></td>
</tr>
<tr>
<td>Sleep Efficiency (%)</td>
<td>133</td>
<td>90.42 (4.39)</td>
<td>70.41 – 98.01</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* percentage of participants with clinically significant symptoms of depression (>9 on PHQ-9) or insomnia (>14 on ISI [Insomnia Severity Index; Bastien, Vallières, & Morin, 2001]) or impaired score on cognitive measure (Z<–2.0). MMSE: Mini-Mental State Examination (Folstein, Folstein & McHugh, 1975); PHQ-9: Patient Health Questionnaire (Kroenke & Spitzer, 2002); RBANS: Repeatable Battery for the Assessment of Neuropsychological Status (Randolph, Tierney, Mohr & Chase, 1998); PM: PM; OSA: obstructive sleep apnoea; WASO: wake after sleep onset; TST: total sleep time.
Table 2.3.

Means (standard deviations) for Insomnia Severity Index (ISI) & actigraphy-derived sleep metrics in participants with and without symptoms of obstructive sleep apnoea (OSA) and insomnia.

<table>
<thead>
<tr>
<th></th>
<th>OSA Negative (N = 98)</th>
<th>OSA Positive (N = 35)</th>
<th>No/sub-threshold insomnia (N = 109)</th>
<th>Clinically significant insomnia (N = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISI Score</td>
<td>5.86 (5.04)</td>
<td>7.25 (5.23)</td>
<td>5.39 (4.32)</td>
<td>16.44 (1.74)</td>
</tr>
<tr>
<td>Awakening Length</td>
<td>3.34 (1.26)</td>
<td>3.91 (1.22) †</td>
<td>3.39 (1.27)</td>
<td>3.90 (1.21)</td>
</tr>
<tr>
<td>Awakening Number</td>
<td>11.75 (5.46)</td>
<td>13.35 (6.48)</td>
<td>12.08 (5.82)</td>
<td>15.81 (6.57)</td>
</tr>
<tr>
<td>WASO (minutes)</td>
<td>37.36 (19.63)</td>
<td>48.97 (20.62) ‡</td>
<td>38.82 (20.10)</td>
<td>56.03 (15.57) †</td>
</tr>
<tr>
<td>TST (minutes)</td>
<td>412.18 (49.39)</td>
<td>417.87 (44.91)</td>
<td>413.57 (49.37)</td>
<td>424.89 (57.05)</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>90.94 (4.41)</td>
<td>88.96 (4.05) †</td>
<td>90.69 (4.47)</td>
<td>87.32 (3.39) †</td>
</tr>
</tbody>
</table>

Note. † p < 0.05; ‡ p < 0.01; WASO: wake after sleep onset; TST: total sleep time.
Sleep and prospective memory

Table 2.4 shows the model (i.) examining the role of sleep disruption, specifically TST, awakening number and awakening length, in the relationship between age and PM task accuracy. After controlling for education, the mediation model explained 17% of the variance in PM, $F(5, 125) = 4.94, p < 0.001$, approximately 6% more than the model with age and education alone ($R^2 = 0.11$). Older age was associated with fewer but longer awakenings. As predicted, longer awakenings were associated with poorer PM. However, the other sleep variables were not related to PM. As predicted, there was a significant and negative total effect of age on PM. However, when the sleep variables were added, age was no longer significantly related to PM. Additionally, there was a significant indirect effect via longer awakenings ($path a^l b^l = -0.27, CI = -0.66$ to -0.04), indicating that longer awakenings mediated the relationship between older age and poorer PM. The indirect paths via the other sleep variables were not significant ($path a^l b^l$ via awakening number: -0.13, CI = -0.38 to 0.04; via TST: -0.01, CI = -0.10 to 0.05). Thus, only awakening length was used in subsequent, serial mediation analyses.

Additional analyses substituting WASO and sleep efficiency for awakening number (reported in the supplementary materials, pp. 162) largely replicated the above results, and did not find any additional significant relationships for WASO or sleep efficiency.
Table 2.4.

**Serial mediation models for predicted relationships between age, sleep disruption, cognition, mood, and prospective memory in older adults.**

<table>
<thead>
<tr>
<th>Model</th>
<th>Predictor</th>
<th>Mediator 1</th>
<th>Mediator 2</th>
<th>Outcome</th>
<th>Covariate</th>
<th>Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(a^1) (a^2) (d^{e1}) (b^1) (b^2) (e^1) (e^2) (c) (c')</td>
</tr>
<tr>
<td>i.</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.07‡ -4.20† 1.59‡ -0.62† -0.23</td>
</tr>
<tr>
<td></td>
<td>Awakening Length</td>
<td></td>
<td></td>
<td>PM Accuracy</td>
<td>Education</td>
<td>-0.25‡ 0.52</td>
</tr>
<tr>
<td></td>
<td>Awakening Number</td>
<td>TST</td>
<td></td>
<td></td>
<td></td>
<td>0.80 0.01</td>
</tr>
<tr>
<td>ii.</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.06‡ 0.11 -0.69 -3.42‡ 0.75‡ 1.26† 0.57 -0.49 -0.35</td>
</tr>
<tr>
<td></td>
<td>Awakening Length</td>
<td></td>
<td>Retrospective Memory</td>
<td>PM Accuracy</td>
<td>Education</td>
<td>0.04‡ 0.13 -3.98† 5.04† 1.39† 0.07‡ -0.61† -0.57</td>
</tr>
<tr>
<td>iii.</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.05‡ 0.04‡ -0.13 -3.98† 5.04† 1.39† 0.07‡ -0.61† -0.57</td>
</tr>
<tr>
<td></td>
<td>Awakening Length</td>
<td>TST</td>
<td>Shifting</td>
<td>PM Accuracy</td>
<td>Education</td>
<td>0.05‡ 0.04‡ -0.13 -3.98† 5.04† 1.39† 0.07‡ -0.61† -0.57</td>
</tr>
<tr>
<td>iv.</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.06‡ -0.09‡ 0.38† -3.94† -1.44 1.62† -0.02 -0.62† -0.47</td>
</tr>
</tbody>
</table>

Note. †: p < 0.05; ‡: p < 0.01; \(a\): path between predictor and mediator; \(b\): path between mediator and outcome variable, controlling for predictor; \(d\): path between mediators; \(c\): total effect between predictor and outcome variable; \(c'\): direct effect between predictor and outcome variable, after taking account of indirect effect(s); \(e\): path between covariate and outcome variable/mediator. TST: total sleep time; PM: prospective memory.
Sleep, prospective memory and other cognitive variables

To determine whether the mediating effect of longer awakenings in the relationship between age and PM was, itself, a function of retrospective memory, shifting, or depression, serial mediation models were conducted (Table 2.4). When retrospective memory was included, the model (ii.) explained 25% of the variance in PM, $F(4, 123) = 10.41, p < 0.001$ (total effect: $R^2 = 0.10$). The indirect effect via awakening length remained significant ($path a'b^l = -0.20, CI = -0.48$ to $-0.01$). However, this effect was not carried by the impact of longer awakenings on retrospective memory ($path a'd^{l1}b^2 = -0.03, CI = -0.14$ to $0.05$), nor was the indirect effect via retrospective memory significant ($path a^2b^2 = 0.08, CI = -0.15$ to $0.33$). Nonetheless, better retrospective memory predicted better PM. These findings suggest awakening length and retrospective memory were independent predictors of PM.

When shifting was included, the model (iii.) explained 17% of the variance in PM, $F(4, 122) = 6.21, p < 0.001$ (total effect: $R^2 = 0.09$). The serial indirect effect via awakening length and shifting was significant ($path a'd^{l1}b^2 = -0.03, CI = -0.12$ to $-0.004$). Both the individual indirect effects via awakening length alone, and shifting alone, were also significant ($path a'b^l = -0.20, CI = -0.56$ to $-0.01$; $path a^2b^2 = 0.19, CI = 0.03$ to $0.50$). These findings suggest poorer shifting predicted poorer PM, and that age-related PM decline is partially mediated in a serial fashion by longer awakenings and poorer shifting.

Sleep, prospective memory and depression.

When depression was included, the model (iv.) explained 17% of the variance in PM, $F(4, 126) = 6.45, p < 0.001$ (total effect: $R^2 = 0.11$). The serial indirect effect via awakening length and depression was significant ($path a'd^{l1}b^2 = -0.03, CI = -0.11$ to $-0.003$). Both the individual indirect effects via awakening length alone, and depression alone were also significant ($path a'b^l = -0.25, CI = -0.63$ to $-0.03$; $path a^2b^2 = 0.13, CI$
= 0.002 to 0.34), although depression did not independently predict PM. These findings suggest that longer awakenings are associated with both greater depression and poorer PM, and additionally that age-related PM decline is partially mediated in a serial fashion by longer awakenings and greater depression.

**Discussion**

**Sleep and prospective memory**

The first aim was to determine if there was an association between sleep disruption and PM in older adults. As hypothesised, sleep disruption in the form of longer awakenings was related to poorer event-based PM whilst, contrary to predictions, number of awakenings and TST were not related to PM. Additional analyses assessing the relationship between sleep efficiency and PM, and WASO and PM were also non-significant (see supplementary materials, pp. 162).

The particular significance of awakening length is consistent with other research suggesting that fragmentation of sleep by awakenings is predictive of poorer cognition in older adults (Blackwell et al., 2014). Additionally, the non-significant findings for WASO could possibly suggest that measures of total wake duration are less sensitive at capturing the particular scenario of sleep disruption that is most detrimental. Indeed, there was only a small association (0.36) between WASO and average awakening length in our sample, and visual inspection of the relationship suggests those participants with a ‘concerning’ amount of WASO (40mins; Ohayon, Carskadon, Guilleminault, & Vitiello, 2017) had substantial variability in their average awakening lengths (Figure 2.1). Whilst polysomnography data are needed as confirmation, we can speculate that the particular significance of awakening length may result from its utility in capturing a scenario of significant disturbance to slow-wave and REM sleep, due to repeated long awakenings playing havoc with the cyclical nature of sleep through the night. In contrast, the duration of WASO on its own does not indicate whether wake
time is accrued in multiple moderate length awakenings, a single very long awakening, or a high frequency of brief awakenings that do not interfere with sleep cycles. Overall, these findings underscore the importance of considering the duration of individual wake episodes, rather than solely total wake time, in future research examining the links between sleep and cognition.

**Figure 2.1.**

*Relationship between wake after sleep onset (WASO) and awakening length.*

---

**Sleep, prospective memory and cognitive ageing**

Secondly, this study aimed to evaluate whether sleep disruption mediates age-related PM deficits. As predicted, greater age was associated with poorer PM, and this effect was no longer significant when sleep disruption was included in the model. Additionally, the indirect path assessing the association between age and PM via
awakening length was significant, suggesting sleep disruption in the form of longer awakenings explained the relationship between age and PM. This suggests the concurrent existence of sleep and cognition problems in older adults is not merely a function of general, age-related decline. Rather, it is possible that sleep disruption contributes to age-related PM deficits, and, perhaps, cognitive ageing more generally. Research documenting that older adults with poorer sleep have reduced white matter integrity (Sexton et al., 2017), cortical thinning (Lim et al., 2016) and reduced glial and neuronal integrity (Cross et al., 2013) hints at potential neural underpinnings for this proposed relationship. If sleep disruption does, indeed, play a causal role in (rather than being a symptom of) these neural consequences, future research should aim to uncover possible mechanisms (e.g., interruption of sleep-dependent neuro-restoration), and whether particular stages of sleep are critical, e.g. slow-wave, REM sleep. Additionally, treatment of sleep problems may be suggested as an avenue for preventing or halting cognitive decline.

**Sleep, prospective memory, other cognitive domains and depression**

The mediating role of awakening length in the relationship between older age and poorer PM was partially a function of poorer shifting, but not retrospective memory. This is consistent with arguments that executive functions are particularly vulnerable to sleep disruption (Holanda Júnior & de Almondes, 2016), and suggests flow-on effects for PM. Shifting was also a mediator of the relationship between age and PM independent of sleep, consistent with previous research emphasising the importance of executive function in age-related PM decline (McDaniel & Einstein, 2011).

As predicted, the mediating role of awakening length was also partially a function of greater depression. However, awakening length continued to account for the majority of the indirect effect, and depression was not an independent predictor of PM.
This suggests that, whilst interrelationships exist between age, sleep, depression and PM, the importance of sleep to PM function is not completely due to associations with mood.

Sleep in community dwelling older adults

In considering the pattern rather than simply the duration of wakefulness, our study additionally revealed a previously unreported dissociation between number and length of awakenings: older age was associated with fewer but longer awakenings. Given the association between age-related sleep disruption and poor cognition, future research should verify this finding, and establish if long awakenings are a particular driver of the sleep-cognition relationship, perhaps via interference with slow-wave or REM sleep.

In relation to recently published recommendations, the majority of our sample had adequate sleep efficiency, however, only 35% met recommendations for TST (Hirshkowitz et al., 2015). Moreover, whilst normative actigraphy data for older adults are minimal, this finding is broadly consistent with other research (Kurina et al., 2015). The high prevalence of sleep disturbance in this non-clinical sample underscores the importance of better understanding how sleep disruption affects PM, and potential implications for cognitive ageing.

Limitations

Actigraphy has been criticised for overestimating sleep and underestimating wake, particularly as sleep becomes more disturbed. Nonetheless, it allows reliable measurement of habitual sleep in a person’s own home and over a longer term than polysomnography, with high agreement (Ancoli-Israel et al., 2003). Additionally, the greater actigraphically defined sleep disruption exhibited by participants with probable sleep disorders suggests our measure successfully captured sleep disruption.

Our sample was relatively well educated and healthy and, whilst rates of depression were similar to reported community prevalence (Pirkis et al., 2009), rates of
probable insomnia and OSA were lower (Bloom et al., 2009). Although it could be
argued this reduces generalizability, actigraphy suggests our sample was experiencing a
similar level of sleep disturbance to other older adult samples (Kurina et al., 2015).

Finally, as cognitive performance varies as a function of time-of-day and
individual chronotype (Schmidt, Collette, Cajochen, & Peigneux, 2007), we may have
suppressed some sleep disruption effects in allowing our participants to complete the
PM task at their preferred time of day. One previous study has found time-of-day effects
on PM (Fabbri, Tonetti, Martoni, & Natale, 2015), however their task was likely linked
to morning/evening cues, and a moderating effect of circadian time was not supported in
a meta-analysis of sleep deprivation and cognition (Lim & Dinges, 2010).

Conclusion

This study demonstrates that greater sleep disruption, specifically longer
awakenings, is associated with poorer PM, and suggests sleep disruption might
contribute to age-related PM deficits. Thus, there may be an opportunity to improve PM
by treating sleep problems, helping older adults live better for longer.
References


Cavuoto, M. G., Ong, B. E. N., Pike, K. E., Nicholas, C. L., Bei, B., & Kinsella, G. J. (2016). Objective but not subjective sleep predicts memory in community-dwelling


Foreword to Chapter 3.

The preceding chapter demonstrates that sleep disturbance is related to prospective memory in older adults and, furthermore, suggests sleep disturbance helps explain age-related prospective memory deficits. Thus, improving sleep may be an avenue to improve prospective memory in older adults. The following chapter directly evaluates this possibility by testing whether a well-validated intervention to improve older adults’ sleep also improves prospective memory. For consistency with the other empirical chapters included in this thesis, this chapter was written in the style of a manuscript to be submitted for journal publication.
CHAPTER 3

Improving Sleep to Improve Prospective Memory in Older Adults: A Pilot Study

Using Cognitive Behavioural Therapy for Insomnia
Abstract

New evidence suggests older adults’ prospective memory may be affected by disturbed sleep, suggesting intervention to improve sleep could also improve prospective memory (PM; Fine et al., 2019; Scullin et al., 2019). This pilot study assessed whether an established intervention for poor sleep – cognitive behavioural therapy for insomnia (CBT-I) – improved prospective memory in older adults, via the expected benefits of CBT-I on awakening length and sleep efficiency. Twenty-two older adults with poor sleep were allocated to either a four-session CBT-I group or waitlist control. Participants underwent assessment pre and post-intervention of prospective memory with a comprehensive lab-based task, and sleep with actigraphy and questionnaires. Contrary to prediction, CBT-I was not associated with improvements in objective sleep or prospective memory. Neither pre-intervention insomnia symptom severity nor baseline PM moderated the effect of CBT-I on PM. We discuss potential limitations of this study in terms of inclusion criteria, and the severity of participants’ sleep and PM difficulties. Overall, these non-significant findings underscore, rather than discount, the importance of considering objective sleep when evaluating effects of CBT-I, in that subjective improvements in sleep following CBT-I do not necessarily equate to objective improvement in sleep or PM. Future research should focus on objective sleep as an outcome measure (and potentially also an inclusion criteria), when seeking evidence for the benefits of CBT-I on PM.
Introduction

Prospective memory (PM; ability to perform a deferred action at the appropriate time in the future) is a vital cognitive skill that affects older adults’ quality of life and ability to live independently (Woods, Weinborn, Velnoweth, Rooney, & Bucks, 2012). Recent studies suggest older adults’ PM may be adversely affected by poor sleep (Fine et al., 2019; Scullin et al., 2019). Thus, interventions to improve older adults’ sleep may also improve their PM. One such intervention, cognitive behavioural therapy for insomnia, or CBT-I, has been well-validated (Bloom et al., 2009). While the above listed studies provide intriguing initial evidence that poor sleep is related to poor PM, these studies are limited by use of cross-sectional data. Therefore, the nature of this relationship – specifically, whether poor sleep causes impairments in PM – has yet to be evaluated. We addressed this question by manipulating sleep through a course of CBT-I and evaluating whether PM ability subsequently increased in a sample of community dwelling older adults with sleep complaints.

Prospective memory complaints are among the most common cognitive issues reported by older adults (McDaniel & Einstein, 2007). Additionally, over half of older adults complain of significant sleep disruption (Almeida & Pfaff, 2005; Bloom et al., 2009). A potential relationship between older adults’ sleep difficulties and PM is suggested by findings that older adults with poor sleep have deficits in the cognitive domains underpinning PM: executive function and retrospective memory (e.g., Anderson & Horne, 2003; Kliegel et al., 2016; Lafortune et al., 2014; Rose et al., 2015; see Chapter 1 for a full review). Furthermore, two recent studies have linked poorer event-based PM in older adults to longer awakenings as measured by actigraphy during habitual sleep (Fine et al., 2019; Chapter 2 in this thesis), and shorter REM sleep duration measured by polysomnography.
(PSG; Scullin et al., 2019). Thus, recent direct evidence suggests older adults’ PM complaints may be related to sleep problems, however, the direction of this relationship it is not yet clear.

Mirroring themes seen in the general sleep-cognition literature (Yaffe, Falvey, & Hoang, 2014), researchers exploring the sleep-PM link have considered whether sleep disruption may in fact cause, or at least contribute to, age-related PM decline. Whilst the two studies assessing sleep and PM in older adults concluded that sleep statistically mediated age-related PM deficits (Fine et al., 2019 [Chapter 2, this thesis]; Scullin et al., 2019), the study designs did not allow for convincing evidence of causality. Of note, however, some evidence for this causal relationship is provided by experimental sleep deprivation studies in younger adults (Diekelmann, Wilhelm, Wagner, & Born, 2013; Grundgeiger, Bayen, & Horn, 2014; Occhionero, Cicogna, & Esposito, 2017). Specifically, these studies show that participants who undergo sleep deprivation have poorer PM performance the next day, compared to participants who sleep normally. While these sleep deprivation studies provide initial evidence for a causal relationship, extended periods of habitual sleep disruption in older adults are, arguably, not equivalent to the simple restriction of total sleep time for a brief period (Wilckens, Erickson, & Wheeler, 2012). Instead, older adults experience reduced total sleep time over months and years. Further, older adults’ sleep disruption is not marked by deprivation alone, but by taking longer to fall asleep and having more frequent and longer awakenings through the night, reducing overall sleep efficiency (Vitiello, 2009). Whilst it is not reasonable to produce this sort of long-standing sleep disturbance in older participants to test causality, improving sleep with CBT-I and then assessing effects on PM could present a way to demonstrate a causal relationship between sleep and PM. If manipulating sleep with CBT-I does, indeed,
improve PM, then the hypothesis that poor sleep directly contributes to poorer PM in older adults would receive support. Conversely, if CBT-I does not improve PM, it may suggest that sleep and PM problems simply co-occur rather than being causally related, or that a third factor, such as general brain network disruption (Porter, Buxton, Avidan, & Porter, 2015), is responsible for both sleep disruption and poor cognition. Thus, the results of the current study could clarify the nature of the sleep-PM association, and perhaps suggest more broadly whether treating sleep problems can improve cognition, specifically, PM.

Treatment of sleep problems is especially relevant for older adults not just because of the potential links between sleep and cognition, but also because rates of sleep disturbance are particularly high in this population. Older adults have higher rates of sleep pathology such as insomnia and sleep apnoea, and also experience sleep disruption due to age-dependent changes in circadian rhythms and sleep architecture (Bloom et al., 2009). The high prevalence of sleep problems in older adults has led to a tendency to view poor sleep as an inevitable consequence of aging (Vitiello, 2009). However, an alternative view recognises that the sleep problems of older adults are deserving of intervention, and can be effectively treated (Irwin, Cole, & Nicassio, 2006).

CBT-I is the recommended first line treatment for addressing sleep problems in adults and older adults (Morgenthaler et al., 2006; Ree, Junge, & Cunnington, 2017). This is true even for the large number of older adults with insomnia symptoms who also have sleep apnoea (Fung, Martin, Josephson, & et al., 2016; Sweetman, Lack, Lambert, Gradisar, & Harris, 2017). By targeting a range of non-specific sleep issues (e.g. poor sleep hygiene) as well as dysfunctional beliefs and attitudes about sleep, CBT-I is useful even where sleep problems occur in the context of other co-morbid health conditions (Stepanski & Rybarczyk, 2006). CBT-I involves education, supporting clients to change behaviours that
interfere with sleep, and cognitive interventions to restructure unhelpful beliefs, reduce worry and reduce cognitive hyper-arousal. The number of sessions and method of delivery for CBT-I vary, but older adults have been shown to benefit from as few as four sessions of CBT-I, delivered in a group format (Lovato, Lack, Wright, & Kennaway, 2014). Whilst the treatment name refers to ‘insomnia’, many studies report CBT-I improves sleep in participants who are broadly defined as having difficulty sleeping (Nau, Mccrae, Cook, & Lichstein, 2005). This use of less stringent participant inclusion criteria is likely due to a lack of quantitative descriptors of poor sleep in standard insomnia diagnostic criteria (American Psychiatric Association, 2014), and the need to be sensitive to normal developmental changes in sleep patterns when defining sleep problems (Nau et al., 2005).

Evidence for the effectiveness of CBT-I is primarily based on improvements in sleep measured by subjective report. For example, a large meta-analysis reported significant benefits on subjective sleep efficiency, wake after sleep onset (WASO), sleep onset latency (SOL) and number of awakenings (Straten et al., 2017). Significant improvements are also evident for ratings of overall sleep quality and insomnia symptoms (Straten et al., 2017). Fewer studies have considered whether CBT-I improves objective sleep, and results are more mixed. In a review of 10 randomised controlled trials that included measures of objective sleep, effects were significant for sleep efficiency and WASO, were significant but had a risk of bias for total wake time, and were not significant for total sleep time (TST) and SOL (Okajima, Komada, & Inoue, 2010).

Amongst older adults, evidence for improvement in objective sleep following CBT-I is also mixed. Irwin and colleagues (2014) did not find any significant effects on objective sleep in their large sample (N = 123) of older adults who received intensive CBT-I treatment (4 months). Kay and others (2015) reported significant effects for objectively
measured sleep efficiency, TST, and WASO, but not SOL \((N = 63)\) after 8 sessions of CBT-I. Lovato and others (2014) reported significant effects for TST and WASO, but not sleep efficiency in their large sample \((N = 118)\) of older adults receiving brief treatment (4 weeks). Martin and others (2017) reported significant effects for sleep efficiency, total wake time, number of awakenings, but not TST, in their sample of predominately male veterans \((N = 42)\) receiving brief treatment (4 weeks).

In summary, CBT-I is recommended as a treatment for sleep problems primarily based on improvements in subjective sleep, with some additional evidence for improvements in objective sleep. The discrepancy between objective and subjective sleep findings may or may not be important, depending on whether objective baseline measures suggest deficient sleep. If sleep is objectively ‘normal’ at baseline, as it often is (paradoxical insomnia; Parrino, Milioli, De Paolis, Grassi, & Terzano, 2009), then lack of improvement in objective measures is not particularly concerning. If sleep is not normal, and stays that way despite improved self-report, it may be that tolerance rather than remission from sleep problems is taking place (Pillai, Roth, & Drake, 2016). In the face of discrepancies between objective and subjective sleep improvement, using other outcome measures following CBT-I can help demonstrate its utility. For example, measures of daily functioning – important for the diagnostic criteria for insomnia (American Psychiatric Association, 2014) – could help determine whether treatment with CBT-I has real-world benefits for patients.

One objective way to assess the benefit of CBT-I for daily function is to measure improvement in cognition. Two studies so far have taken this approach. The first included a small \((N = 5)\) mixed sample of adults and reported that CBT-I improved vigilance (Gosselin, Campbell, Grenier, & J, 2016). The second included a moderate-sized sample of
older adults \((N = 48)\) and reported marginal improvement \((p = .072)\) in attentional switching, linked to objective WASO (Wilckens et al., 2016). The current study sought to add to these preliminary results regarding the benefit of CBT-I for cognition by considering an aspect of cognition that is highly relevant to older adults’ daily functioning: PM.

To summarise, this pilot study aimed to assess whether a course of CBT-I can improve PM in community dwelling older adults. Specifically, we compared the PM performance of older adults pre and post CBT-I with a waitlist control group (primary outcome), and tested whether improvement in objective sleep (secondary outcome) mediated the hypothesized improvement in PM following CBT-I. We addressed limitations in the previous literature by using an objective measure of sleep (actigraphy), and by examining whether CBT-I can lead to improvement in a cognitive ability with implications for daily functioning, namely PM. We also hoped to be able to draw conclusions regarding whether any relationship between sleep and PM is causal. That is, if PM improves in the intervention group but not the waitlist control group, it is likely that sleep disturbance causes decreased PM. The objective sleep metrics used were sleep efficiency and awakening length. Sleep efficiency is the measure most frequently found to improve following CBT-I and, in comparison to WASO and SOL, is able to capture problems with both sleep onset and early morning awakening (Okajima et al., 2010). Awakening length has previously been implicated in the sleep-PM association, and enables a more fine-grained measure of the pattern of sleep disruption, because it is calculated from the duration of individual wake episodes rather than solely total wake time (Fine et al., 2019). We assessed PM with a comprehensive laboratory-based measure that mirrors typical PM tasks encountered in daily life, and includes both time-based and event-based tasks. We hypothesized that CBT-I would be associated with better post-intervention time-based and
event-based PM, via the beneficial impact of CBT-I on reduced average awakening length (Hypothesis 1) and increased sleep efficiency (Hypothesis 2).

**Method**

**Study Design**

The study employed a quasi-randomised controlled design. Participants were allocated to either a CBT-I or waitlist control condition and underwent pre-intervention and post-intervention assessment of objective sleep and PM. Our initial aim was to achieve fully-random allocation, however, due to difficulties with recruitment, in three instances participants were allocated to their preferred condition (two to CBT-I and one to waitlist control) in order to retain them in the study (potential implications addressed in the Discussion). Further, there were not sufficient personnel for assessors to be blinded to participants’ condition.

**Participants**

Community-dwelling older adults were recruited through ongoing research programs (the Healthy Ageing Research Program [HARP]; the Western Australia Memory Study [WAMS] at the Australian Alzheimer’s Research Foundation [AARF], Research Director: R. N. Martins) or through the West Australian Participant Pool (WAPP, Director: R. S. Bucks). See General Methods in the Supplementary Materials for further information.

Inclusion criteria for the study were:

a) Aged 55 years and above;

b) Subjective sleep problems as indexed by a score above 7 on the Insomnia Severity Index (ISI; Bastien, Morin, Bastien, Vallie, & Morin, 2001) or above 5 on the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989);

Exclusion criteria were:
a) Presence of a serious neurological condition (e.g. stroke, epilepsy, or performance suggestive of likely dementia on a comprehensive neuropsychological battery, based on the McKhann et al., 2011 criteria);

b) Uncontrolled/ severe psychiatric conditions (e.g. psychotic disorders, major depressive episode depression not under adequate treatment by a medical or mental health professional [American Psychiatric Association, 2014]);

c) Moderate to high risk of an alcohol use disorder (score >15 on the Alcohol Use Disorders Identification Test [AUDIT]; Babor, Higgins-Biddle, Saunders, Monteiro, 2015).

Participants were permitted to have primary sleep disorders (e.g. sleep apnoea, periodic limb movement disorder, restless leg syndrome – but not narcolepsy) as we wanted to ensure our sample was representative of typical older adults with sleep difficulties, who often have insomnia comorbid with other sleep disorders (Bloom et al., 2009). It was a requirement that treatment for other sleep disorders remained stable over the course of the study.

Participants in ongoing research projects (HARP and WAMS) were identified for potential inclusion through file review of previously completed sleep questionnaires. Individuals from WAPP who had not previously participated in research projects were posted study information and invited to complete and return sleep questionnaires if they were interested in participating. Potential participants from HARP, WAMS and WAPP who

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1 Initially, it was stipulated that other primary sleep disorders should be adequately treated according to current best practice from two weeks prior to the start of the study and its full duration. However, the criterion was changed to specify treatment (or lack of) must remain unchanged during the course of the study, as previously undiagnosed sleep apnoea was uncovered in some participants through their participation in a contemporaneous PSG study. Three of the seven participants who were assessed by PSG had sleep apnoea. Of the remaining 14 participants who did not undergo PSG, six had high risk of obstructive sleep apnoea on the Berlin questionnaire (Netzer, Strohs, Netzer, Clark-, & Strohl, 1999).
met criteria based on sleep questionnaires then underwent telephone screening to assess suitability based on the broader inclusion criteria. As shown in Figure 3.1, 26 participants met inclusion criteria and were enrolled in the study. Two participants withdrew before randomization, and two withdrew after randomization but before the study commenced. Twenty-two participants completed the study, and all except one (who was missing sleep assessment data due to equipment failure) were included in the analysis.

Figure 3.1.

**Participant flowchart.**
Procedure

The study was conducted between October 2015 and March 2018 with four separate rounds of recruitment and data collection in that period. Each round followed the procedure shown in Figure 3.2. After being identified and screened by phone, eligible participants were consented, allocated to treatment condition, and provided with wrist-mounted actigraphs for the pre-intervention sleep assessment. Participants used the actigraphs at home for approximately one week and then returned for pre-intervention cognitive assessments. All participants had a weekend included in their actigraphy data. Participants in the CBT-I condition then commenced treatment, following a standard CBT for primary insomnia protocol (Edinger & Carney, 2008). This protocol consisted of an individual assessment session, four group treatment sessions one week apart, and two group follow-up sessions at 4-6 weeks and 8-12 weeks after the final treatment session (based on participant availability). After the CBT-I participants’ final follow-up session, all participants received actigraphs for the post-intervention sleep assessment, and underwent the post-intervention cognitive assessment. Thus, allowing for the full treatment duration, we aimed for an interval of approximately 120 days between pre and post-assessments.

As part of related research activities, but not directly relevant to this study, some participants (three waitlist control and four CBT-I) also underwent PSG recordings on one night during the period when they wore the actigraphs. Due to concerns that it would not be representative of participants’ typical sleep, the actigraphy data collected on this night were not included when averaging the sleep metrics, though all participants still had a minimum five nights of actigraphy data.
Figure 3.2.

Study procedure.
Sleep assessment

For pre and post-intervention sleep assessment, participants were provided with an actigraph (Tri-axial wGT3X-BT activity monitor, Actigraph LLC, FL, USA) and a diary to record bed (when they started trying to sleep) and rise time (when they woke for the last time (Carney et al., 2012). Further details on actigraphy are provided in the General Methods in Supplementary Materials. Actigraphy data were autoscored using the Cole-Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992) and Actilife software (version 6.11.8). Autoscored data were visually inspected for problematic non-wear time (not wearing the actigraph from at least 5 minutes before bedtime until at least 5 minutes after rise time). If such problematic non-wear time was identified, the night was excluded from the analysis (one night [< 1%] was affected by non-wear time). Autoscored bed and rise times were adjusted to match the diary or, if there was a discrepancy of more than 60 minutes, visual manual scoring was used, taking into account the diary and typical movement patterns of bed/rise time. Visual manual scoring was also used when diary times were missing, and, in total, 53 nights (18%) required visual manual scoring. Once the rest period was defined, sleep efficiency, TST, WASO, number of awakenings, and average awakening length were calculated for each night and averaged for each participant. To measure objective improvement in sleep, the difference between pre and post-intervention sleep efficiency and average awakening length were calculated for each participant, with a positive score for sleep efficiency and a negative score for awakening length indicative of improvement.²

² As some commentators have criticised the use of simple difference scores to index improvement (Hayes & Rockwood, 2016), we also calculated residual scores from post-intervention scores regressed on pre-intervention scores using linear regression in SPSS, and used these residual scores instead of difference scores, with no substantive differences from our primary findings.
Cognitive assessment and PM measure

For the pre and post-intervention cognitive assessment, participants completed a comprehensive battery of neuropsychological tests including PM (see General Methods in Supplementary Materials), with multiple breaks to prevent fatigue, taking on average 3.5 hours. The assessment was conducted by experienced research assistants under the supervision of an endorsed Clinical Neuropsychologist.

The PM measure was the Western Australia Prospective Memory test (WAProm): a laboratory-based measure based on the general methodology of the well-validated research version of the Memory for Intentions Screening Test (MIST; Raskin, 2009). Previous research has supported the reliability and validity of the WAProm (McCabe et al., 2018). The WAProm has 10 PM tasks completed over 30 minutes by participants whilst working on a standard word-search, which serves as the ongoing task. These PM tasks vary by cue-type (time-based and event-based), delay between instruction and cue (5 minutes and 15 minutes), and retrospective memory load (1-step and 2-step instructions; only event-based items had 2-step instructions). An example of a 1-step time-based item is, “In 5 minutes, point to the floor.” An example of a 2-step event-based item is, “When I pick up the key, remind me to lock the door and put the keys in my pocket.” Table 3.1 shows the breakdown of cue-type, delay time and retrospective memory load for each task. The WAProm is scored by awarding 1 point for each PM task executed at the correct time, and either 1 or 2 points (based on retrospective memory load) for execution of the correct action/s, giving a maximum total score of 22 points.
Time-based and event-based WAProm scores were calculated for each participant at pre-intervention and post-intervention. For consistency with the time-based score, the event-based score included only the 1-step instruction items. Thus, both scores were based on 4 low retrospective memory load items.

Time-based and event-based WAProm scores were corrected for age, education and gender in the following way. Linear regression was used to predict WAProm scores from age, education and gender in a study-based normative sample of healthy community-dwelling older adults who took part in WAMS ($N = 212$; described in supplementary materials, pp. 163). Coefficients generated in the regression (supplementary materials, pp. 165) were used to calculate predicted WAProm scores for each participant in the current study. Achieved scores were subtracted from predicted scores to generate residual scores (unstandardized). A positive residual score indicated

<table>
<thead>
<tr>
<th>PM task number</th>
<th>Cue-type</th>
<th>Delay time</th>
<th>Retrospective memory load</th>
<th>Possible points</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Event</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>Event</td>
<td>5</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>Time</td>
<td>15</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>Time</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>Event</td>
<td>15</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>Time</td>
<td>15</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>Event</td>
<td>15</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>Event</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>Time</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>Event</td>
<td>5</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
that a participant’s achieved WAProm score was better than predicted, and a negative residual score indicated their achieved score was poorer than predicted.

Participants also completed the California Verbal Learning Test – Adult Version (CVLT; Woods, Delis, Scott, Kramer, & Holdnack, 2006), and the Trail Making Test A and B (Reitan & Wolfson, 1985; Tombaugh, 2004). The CVLT long-delay free recall standardized score was used as a measure of retrospective memory. The standardized difference score for Trail Making Test A versus Trail Making Test B served as a measure of executive function (Strauss, Sherman, & Spreen, 2006). This score was reflected so that a higher score indicated better executive function.

**Questionnaires**

At pre and post-intervention visits, participants completed the ISI and PSQI to measure insomnia symptoms and subjective sleep quality (Bastien et al., 2001; Buysse et al., 1989), and the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) to measure mood. Each of the three axis scores from the DASS (depression score, anxiety score, stress score) were calculated.

**Intervention**

The CBT-I intervention was delivered by provisionally registered psychologists undertaking a Masters degree in Clinical Psychology or Clinical Neuropsychology, closely supervised by a clinical psychologist with expertise in treatment of insomnia.

Participants in the CBT-I intervention condition attended an individual assessment session with a therapist. They then completed the intervention, which consisted of four, 2-hour group sessions, and two, 1-hour follow-up sessions, following a standard CBT for primary insomnia protocol (Edinger & Carney, 2008). The intervention included:

1) Education on sleep, insomnia, and age-related sleep changes;
2) Sleep hygiene;
3) Stimulus control;
4) Sleep restriction;
5) Cognitive restructuring addressing negative automatic thoughts and dysfunctional beliefs about sleep;
6) Stress management;
7) Problem solving;
8) Meditation and aspects of mindfulness.

Participants used sleep diaries (Carney et al., 2012) and the ISI (Bastien et al., 2001) to monitor their progress.

**Statistical Analysis**

Conditional Process Analysis for SPSS (version 3.1, Hayes, 2013) linear mediation analysis was used to assess whether receiving CBT-I was associated with better post-intervention time-based and event-based PM, via the beneficial impact of CBT-I on reduced average awakening length (Hypothesis 1) and increased sleep efficiency (Hypothesis 2). Intervention group was used as the predictor and coded as 1 = CBT-I, and 0 = waitlist control. Pre-intervention time-based or event-based PM was included as a covariate. As described above and in the supplementary materials (pp. 163), the pre-intervention and post-intervention PM measures were unstandardised residual scores from WAProm raw scores, corrected for age, gender and education. Statistical significance was set at .05 (two-tailed), and all analyses calculated using 5000 bootstrapped samples and bias-corrected 95% confidence intervals (CI). Direct effects of CBT-I, and indirect effects of CBT-I via improved sleep were calculated using a series of ordinary least squares regressions and the bootstrapping procedure. Each indirect effect is considered significant when the CIs do not cross zero.

As a supplement, group-level changes in PM, sleep, insomnia symptoms, and other aspects of cognition are presented in the supplementary materials, pp. 167.
Results

Sample characteristics and descriptive statistics are presented in Table 3.2. One-way analysis of variance (bootstrapped 5000 samples) was used to test for baseline differences in demographics, mood, sleep, PM and other cognitive domains. Whilst acknowledging that small samples and skewed data may affect these analyses, there were no significant differences between the control and CBT-I conditions, pre-intervention. Demographic variables were furthermore controlled for in the PM measures through calculation of residual scores as described above. The average interval between pre and post-intervention cognitive assessments was equivalent between the conditions. Depression, anxiety and stress levels in both groups were, on average, within the normal range pre and post-intervention (Lovibond & Lovibond, 1995). Average PSQI scores suggested poor subjective sleep quality (score >5) in both groups pre and post-intervention. Average ISI scores suggested subthreshold insomnia symptoms in both groups at pre-intervention. However, in comparison to scores at recruitment which were above the cut-off of 7 for all participants, some participants’ ISI scores had reduced below 7 by the time pre-intervention assessments were conducted (control N = 4; CBT-I N = 4). At post-intervention, average ISI scores suggested ongoing subthreshold insomnia in the control condition, whilst symptoms in the CBT-I condition had reduced below clinical thresholds. No side effects or problems with adherence were reported.
Table 3.2.

Descriptive statistics.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Min – Max</th>
<th>Full sample ((N = 21)) mean (std. deviation)</th>
<th>Control condition ((N = 10)) mean (std. deviation)</th>
<th>CBT-I condition ((N = 11)) mean (std. deviation)</th>
<th>Control vs. CBT-I comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at start (years)</td>
<td>56 – 81</td>
<td>69.67 (6.53)</td>
<td>70.30 (7.38)</td>
<td>69.09 (5.96)</td>
<td>(F = 0.34) (p = .569) (\eta^2 = 0.02)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>10 – 20</td>
<td>15.00 (2.92)</td>
<td>14.30 (2.41)</td>
<td>15.64 (3.30)</td>
<td>(F = 0.48) (p = .497) (\eta^2 = 0.03)</td>
</tr>
<tr>
<td>Percentage male</td>
<td></td>
<td>47.60</td>
<td>50.00</td>
<td>45.50</td>
<td>(F = 0.05) (p = .821) (\eta^2 = 0.004)</td>
</tr>
<tr>
<td>Interval between cognitive assessments (days)</td>
<td>36 – 256</td>
<td>139.43 (67.23)</td>
<td>139.10 (82.91)</td>
<td>139.73 (53.4)</td>
<td></td>
</tr>
</tbody>
</table>

Mood and subjective sleep

<table>
<thead>
<tr>
<th></th>
<th>Control condition ((N = 10)) mean (std. deviation)</th>
<th>CBT-I condition ((N = 11)) mean (std. deviation)</th>
<th>Pre-intervention Control vs. CBT-I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression (DASS)</td>
<td>1.30 (1.34)</td>
<td>2.55 (3.01)</td>
<td>(F = 1.44) (p = .246) (\eta^2 = 0.08)</td>
</tr>
<tr>
<td>Anxiety (DASS)</td>
<td>2.60 (2.88)</td>
<td>1.36 (1.86)</td>
<td>(F = 0.60) (p = .446) (\eta^2 = 0.03)</td>
</tr>
<tr>
<td>Stress (DASS)</td>
<td>5.10 (4.12)</td>
<td>6.36 (3.38)</td>
<td>(F = 0.76) (p = .394) (\eta^2 = 0.04)</td>
</tr>
<tr>
<td>ostive global sleep quality (PSQI)*</td>
<td>Pre-intervention Control</td>
<td>Post-intervention Control</td>
<td>Pre-intervention CBT-I</td>
</tr>
<tr>
<td>---</td>
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</tr>
<tr>
<td></td>
<td>12.33 (3.28)</td>
<td>11.40 (2.22)</td>
<td>12.20 (3.65)</td>
</tr>
<tr>
<td>Insomnia symptoms severity (ISI)</td>
<td>9.20 (3.77)</td>
<td>9.30 (3.77)</td>
<td>11.00 (5.08)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Actigraphy derived sleep</th>
<th>Pre-intervention Control</th>
<th>Post-intervention Control</th>
<th>Pre-intervention CBT-I</th>
<th>Post-intervention CBT-I</th>
<th>$F$</th>
<th>$p$</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep efficiency (percentage)</td>
<td>90.28 (4.39)</td>
<td>90.38 (4.10)</td>
<td>91.67 (4.17)</td>
<td>92.13 (4.26)</td>
<td>0.61</td>
<td>.444</td>
<td>0.03</td>
</tr>
<tr>
<td>Average awakening length (mins)</td>
<td>3.65 (1.26)</td>
<td>4.01 (1.69)</td>
<td>3.51 (1.74)</td>
<td>3.59 (1.56)</td>
<td>0.01</td>
<td>.906</td>
<td>0.00</td>
</tr>
<tr>
<td>Total sleep time (mins)</td>
<td>412.05 (47.32)</td>
<td>422.38 (38.27)</td>
<td>385.29 (51.24)</td>
<td>418.46 (47.02)</td>
<td>1.16</td>
<td>.296</td>
<td>0.06</td>
</tr>
<tr>
<td>Wake after sleep onset (mins)</td>
<td>41.53 (22.10)</td>
<td>40.98 (18.09)</td>
<td>33.86 (20.87)</td>
<td>31.62 (18.02)</td>
<td>0.79</td>
<td>.384</td>
<td>0.04</td>
</tr>
<tr>
<td>Number of awakenings</td>
<td>12.49 (6.56)</td>
<td>12.06 (7.15)</td>
<td>10.61 (6.62)</td>
<td>10.58 (6.05)</td>
<td>0.54</td>
<td>.472</td>
<td>0.03</td>
</tr>
<tr>
<td>Nights of data collected</td>
<td>6.30 (0.48)</td>
<td>6.70 (0.82)</td>
<td>6.82 (0.98)</td>
<td>7.00 (0.63)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control condition (N = 10)</td>
<td>CBT-I condition (N = 11)</td>
<td>Pre-intervention Control vs. CBT-I comparison</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------------------------</td>
<td>-------------------------------</td>
<td>-----------------------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mean (std. deviation)</td>
<td>mean (std. deviation)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm total score</td>
<td>9 – 21</td>
<td>16.30 (2.87)</td>
<td>16.20 (4.37)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm event-based score**</td>
<td>4 – 8</td>
<td>6.60 (1.26)</td>
<td>6.50 (1.27)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm time-based score</td>
<td>2 – 8</td>
<td>4.30 (1.77)</td>
<td>5.20 (2.25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm ongoing task score</td>
<td>6 – 35</td>
<td>18.70 (7.52)</td>
<td>20.60 (8.47)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm total residual score</td>
<td>-4.83 – 9.45</td>
<td>3.03 (2.73)</td>
<td>2.93 (3.76)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm event-based residual score **</td>
<td>-1.58 – 2.68</td>
<td>0.95 (1.26)</td>
<td>0.85 (1.05)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAProm time-based residual score</td>
<td>-1.99 – 5.12</td>
<td>0.47 (1.65)</td>
<td>1.37 (1.99)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other cognitive variables</td>
<td>Control condition (N = 10)</td>
<td>CBT-I condition (N = 11)</td>
<td>Pre-intervention Control vs. CBT-I comparison F (p)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
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<td>-------------------------</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mean (std. deviation)</td>
<td>mean (std. deviation)</td>
<td>F</td>
<td>p</td>
<td>$\eta^2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Min – Max Pre-intervention Post-intervention</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Retrospective memory (CVLT long delay Z score)</td>
<td>-2.00 – 2.00 0.10 (1.20) 0.95 (0.98)</td>
<td>0.18 (0.81) 0.45 (0.79)</td>
<td>0.03</td>
<td>.855</td>
<td>0.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive Function (TMT A – B Z score)</td>
<td>-1.39 – 1.77 0.75 (0.91) 0.60 (0.85)</td>
<td>0.78 (0.58) 0.82 (0.84)</td>
<td>0.01</td>
<td>.934</td>
<td>0.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note:* *N* = 1 missing from CBT-I condition, and *N* = 1 missing from control condition. **Calculated with one-instruction items only. Actigraphy averaged over the sleep data collection period, excluding nights when some participants (*N* = 3 control, 4 CBT) underwent out-of-home polysomnography. CBT-I: Cognitive Behavioral Therapy for Insomnia; DASS: Depression Anxiety Stress Scales (Lovibond, & Lovibond, 1995); PSQI: Pittsburgh Sleep Quality Index (Buysse, et al., 1989); ISI: Insomnia Severity Index (Bastien, Vallières, & Morin, 2001); WAProm: Western Australia PM Test, WAProm residual score with age, education and gender accounted for based on values generated in study based normative sample of community-dwelling healthy older adults (*N* =212) described in the supplementary materials. CVLT: California Verbal Learning Test – Adult version (Delis, Kramer, Kaplan, & Thompkins, 1987); TMT A - B: difference between Trail Making Test A score and Trail Making Test B score (Tombaugh, 2004).
Linear mediation analyses (Table 3.3) assessed whether receiving CBT-I was associated with better post-intervention PM, via the beneficial impact of CBT-I on reduced average awakening length (Hypothesis 1) and increased sleep efficiency (Hypothesis 2) 3.

Models testing awakening length as a mediator of CBT-I’s effect on post-intervention PM explained 37% of the variance in event-based PM, $F(3, 17) = 3.30, p = .046$ (Model 1), and 48% of the variance in time-based PM, $F(3, 17) = 5.30, p = .009$ (Model 2). Contrary to predictions, receiving CBT-I was not associated with reduced awakening length, or improved PM. Indirect effects were not significant (Model 1 path $ab: 0.16, CI:\ -0.32$ to 0.70; Model 2 path $ab: -0.003, CI:\ -0.19$ to 0.68).

Models testing sleep efficiency as a mediator of CBT-I’s effect on post-intervention PM predicted 49% of the variance in time-based PM, $F(3, 17) = 5.34, p = .009$ (Model 4). Contrary to predictions, receiving CBT-I was not associated with increased sleep efficiency, or improved time-based PM. Indirect effects were not significant (path $ab: 0.01, CI:\ -0.40$ to 0.27). For event-based PM, whilst 28% of the variance was accounted for, the overall model was not significant, $F(3, 17) = 2.21, p = .124$ (Model 3).

3 All models were also tested with change in sleep represented as residual scores from regression predicting post-intervention sleep from pre-intervention sleep, rather than change scores. There were no substantive differences to the models reported here.
Table 3.3.

Mediation models for predicted relationships between sleep intervention, change in sleep quality and PM.

<table>
<thead>
<tr>
<th>Model Number</th>
<th>Mediator</th>
<th>Outcome</th>
<th>a</th>
<th>b</th>
<th>c</th>
<th>c'</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N = 21</td>
<td>Change in awakening length</td>
<td>Event-based PM, T2</td>
<td>-0.39</td>
<td>-0.41</td>
<td>0.37</td>
<td>0.21</td>
<td>0.47†</td>
</tr>
<tr>
<td>2. N = 21</td>
<td>Event-based PM, T2</td>
<td>Time-based PM, T2</td>
<td>-0.29</td>
<td>0.01</td>
<td>-0.26</td>
<td>-0.25</td>
<td>0.83‡</td>
</tr>
<tr>
<td>3. N = 21</td>
<td>Change in sleep efficiency</td>
<td>Event-based PM, T2</td>
<td>0.70</td>
<td>-0.06</td>
<td>0.37</td>
<td>0.40</td>
<td>0.54†</td>
</tr>
<tr>
<td>4. N = 21</td>
<td>Event-based PM, T2</td>
<td>Time-based PM, T2</td>
<td>0.34</td>
<td>0.03</td>
<td>-0.26</td>
<td>-0.27</td>
<td>0.82‡</td>
</tr>
</tbody>
</table>

Note. †: p < .05; ‡: p < .01; X: predictor; M: mediator; Y: outcome; D: covariate. T2 = post-intervention, T1 = pre-intervention; a: path between intervention group and change in sleep; b: path between change in sleep and T2 PM (PM), controlling for intervention group; ab: indirect effect of intervention group on T2 PM, via change in sleep; c: total effect between intervention group and T2 PM; c’: direct effect between intervention group and T2 PM, after taking account of indirect effect; d: path between T1 PM and T2 PM. Intervention group: 1 = CBT-I (Cognitive behavioural therapy for insomnia); 0 = waitlist control. Change in sleep is difference between T1 awakening length/ sleep efficiency and T2 awakening length/ sleep efficiency, measured with actigraphy. Increased sleep efficiency is positive change score. Reduced awakening length is negative change score. PM measured with the Western Australia PM Test (WAProm), converted to a residual score with age, gender and education correction. All analyses bootstrapped 5000 samples.
Post-hoc Analyses

Due to the unexpected finding that CBT-I was not associated with either improved sleep or improved PM, we considered whether the effects of CBT-I were moderated by either pre-intervention insomnia symptom severity or pre-intervention PM. We thus had four, post-hoc hypotheses. We predicted that the beneficial impact of CBT-I on sleep and post-intervention PM might only be apparent for participants who had clinically significant pre-intervention insomnia symptoms (Hypothesis 3: models with awakening length moderated by insomnia symptom severity; Hypothesis 4: models with sleep efficiency moderated by insomnia symptom severity). We also predicted that the beneficial impact of CBT-I on sleep and post-intervention PM might only be apparent for participants with poorer than average pre-intervention PM ability (Hypothesis 5: models with awakening length moderated by pre-intervention PM; Hypothesis 6: models with sleep efficiency moderated by pre-intervention PM).

Pre-intervention clinically significant insomnia symptoms were defined as an ISI score above 7 (Bastien et al., 2001). Whilst all participants had clinically significant insomnia at recruitment, we noticed that some participants had reduced symptom severity by the time of pre-intervention assessments, as described above. Participants were coded as 1 = clinically significant insomnia, and 0 = no insomnia pre-intervention. The number of participants in each insomnia category is shown in Table 3.4, broken down by intervention condition. The dichotomised insomnia symptom severity variable was then tested as a potential moderator of the relationship between CBT-I and sleep improvement (a path), and the relationship between CBT-I and post-intervention PM (c path).

To define poorer than average PM, WAProm residual scores from the standardisation sample were converted to percentiles (see supplementary materials, pp.
The value corresponding to the 50\textsuperscript{th} percentile was used to code the pre-intervention PM residual score of each participant in the current study as 1 = below average or 0 = average or above (event-based PM residual score 50\textsuperscript{th} percentile = 0.12; time-based PM residual score 50\textsuperscript{th} percentile = 0.16, pre-intervention). The number of participants in each pre-intervention PM category is shown in Table 3.4, broken down by intervention condition. The dichotomised pre-intervention PM variable was then tested as a moderator of the relationship between sleep improvement and post-intervention PM ($b$ path), and CBT-I and post-intervention PM ($c$ path).

Table 3.4.

| Counts of participants within each insomnia severity and pre-intervention prospective memory category. |
|--------------------------------------------------|----------------|----------------|---------------|
|                                                  | Control Condition | CBT-I Condition | Total         |
| Pre-intervention insomnia symptom severity       | No insomnia       | 4              | 4             | 8             |
|                                                  | Clinically significant insomnia | 6 | 7 | 13 |
|                                                  | Total             | 10             | 11            | 21            |
| Pre-intervention event-based PM Score            | Average or above  | 7              | 4             | 11            |
|                                                  | Below average     | 3              | 7             | 10            |
|                                                  | Total             | 10             | 11            | 21            |
| Pre-intervention time-based PM Score             | Average or above  | 5              | 7             | 12            |
|                                                  | Below average     | 5              | 4             | 9             |
|                                                  | Total             | 10             | 11            | 21            |

Note: Insomnia symptom severity based on the Insomnia Severity Index (ISI; (Bastien, Vallières, & Morin, 2001) where a score of 0 - 7 is no insomnia, and above 7 is clinically significant insomnia. PM (PM) measured with the Western Australia PM Test (WAProm), converted to a residual score with age, gender and education correction. Residual scores categorized as average or above (50\textsuperscript{th} – 100\textsuperscript{th} percentile) or below average (0 – 49\textsuperscript{th} percentile) using a study-based normative sample (supplementary materials).
Table 3.5.

*Moderated mediation models for predicted relationships between sleep intervention, insomnia symptoms, change in sleep quality and prospective memory.*

<table>
<thead>
<tr>
<th>Model Number</th>
<th>Mediator</th>
<th>Outcome</th>
<th>a</th>
<th>e</th>
<th>a*e</th>
<th>b</th>
<th>c</th>
<th>f</th>
<th>c*f</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>5. N = 21</td>
<td>Change in awakening</td>
<td>Event-based PM, T2</td>
<td>-0.10</td>
<td>0.002</td>
<td>-0.49</td>
<td>-0.37</td>
<td>-0.23</td>
<td>-0.22</td>
<td>0.76</td>
<td>0.50†</td>
</tr>
<tr>
<td>6. N = 21</td>
<td>length</td>
<td>Time-based PM, T2</td>
<td>-0.02</td>
<td>0.06</td>
<td>0.42</td>
<td>-0.04</td>
<td>0.51</td>
<td>0.34</td>
<td>-1.24</td>
<td>0.84‡</td>
</tr>
<tr>
<td>7. N = 21</td>
<td>Change in sleep efficiency</td>
<td>Event-based PM, T2</td>
<td>3.12</td>
<td>1.78</td>
<td>-4.04</td>
<td>-0.01</td>
<td>-0.17</td>
<td>-0.21</td>
<td>0.91</td>
<td>0.56†</td>
</tr>
<tr>
<td>8. N = 21</td>
<td></td>
<td>Time-based PM, T2</td>
<td>2.94</td>
<td>1.53</td>
<td>-4.18</td>
<td>-0.05</td>
<td>0.65</td>
<td>0.41</td>
<td>-1.43</td>
<td>0.86‡</td>
</tr>
</tbody>
</table>

*Note.* †: *p < .05; ‡: *p < .01; X: predictor; M: mediator; W: moderator; Y: outcome; D: covariate; T2 = post-intervention, T1 = pre-intervention; a: path between intervention group and change in sleep; e: path between T1 insomnia symptoms and change in sleep; a*e: interaction between intervention group and T1 insomnia symptoms on change in sleep; b: path between change in sleep and T2 PM (PM), controlling for intervention group; f: path between T1 insomnia symptoms and T2 PM; c*f: interaction between intervention group and T1 insomnia symptoms on T2 PM (PM); d: path between T1 PM and T2 PM. Intervention group: 1 = CBT-I (Cognitive behavioural therapy for insomnia); 0 = waitlist control. Change in sleep is difference between T1 awakening length/ sleep efficiency and T2 awakening length/ sleep efficiency, measured with actigraphy. Increased sleep efficiency is positive change score. Reduced awakening length is negative change score. PM measured with the Western Australia PM Test (WAProm), converted to a residual score with age, gender and education correction. Insomnia symptoms measures with the Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001). All analyses bootstrapped 5000 samples.
Table 3.5 shows linear moderated mediation analyses assessing whether – contingent on insomnia severity – receiving CBT-I was associated with better post-intervention PM, via the beneficial impact of CBT-I on reduced awakening length (Hypothesis 4) or increased sleep efficiency (Hypothesis 5). Models testing awakening length as a mediator of CBT-I’s effect on post-intervention PM explained 52% of the variance in time-based PM, $F(5, 15) = 3.28, p = .034$ (Model 6). Contrary to predictions, there was no interaction between insomnia symptoms and the effects of CBT-I on time-based PM directly, or indirectly via awakening length (index of moderated mediation: 0.02, CIs -0.82 to 0.87). For event-based PM, whilst 40% of the variance was accounted for, the overall model was not significant $F(5, 15) = 1.96, p = .143$ (Model 5).

Models testing sleep efficiency as a mediator of CBT-I’s effect on post-intervention PM explained 53% of the variance in post-intervention time-based PM, $F(5, 15) = 3.32, p = .032$ (Model 8). Contrary to predictions, there was no interaction between insomnia symptoms and the effects of CBT-I on time-based PM directly, or indirectly via sleep efficiency (index of moderated mediation: 0.20, CIs -1.64 to 1.99). For event-based PM, whilst 32% of the variance was accounted for, the overall model was not significant, $F(5, 15) = 1.40, p = .280$ (Model 7).
Table 3.6.

**Moderated mediation models for predicted relationships between sleep intervention, change in sleep quality, pre-intervention prospective memory and post-intervention prospective memory.**

![Diagram](image)

<table>
<thead>
<tr>
<th>Model Number</th>
<th>Mediator</th>
<th>Outcome</th>
<th>Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td>9. N = 21</td>
<td>Change in awakening length</td>
<td>Event-based PM, T2</td>
<td>-0.29 0.05 0.22 -0.57 0.98 -1.71</td>
</tr>
<tr>
<td>10. N = 21</td>
<td></td>
<td>Time-based PM, T2</td>
<td>-0.29 0.93 -1.81 -1.36 -0.78 1.18</td>
</tr>
<tr>
<td>11. N = 21</td>
<td>Change in sleep efficiency</td>
<td>Event-based PM, T2</td>
<td>0.36 0.14 -0.17 0.22 1.07 -1.62</td>
</tr>
<tr>
<td>12. N = 21</td>
<td></td>
<td>Time-based PM, T2</td>
<td>0.36 -0.08 -2.02 0.57 -1.03 1.57</td>
</tr>
</tbody>
</table>

**Note.** †: \( p < .05 \); ‡: \( p < .01 \); X: predictor; M: mediator; W: moderator; Y: outcome; D: covariate; T2 = post-intervention; T1 = pre-intervention; a: path between intervention group and change in sleep; b: path between change in sleep and T2 PM (PM), controlling for intervention group; f: path between T1 PM and T2 PM; b*f: interaction between change in sleep and T1 PM on T2 PM; c*f: interaction between intervention group and T1 PM on T2 PM. Intervention group: 1 = CBT-I (Cognitive behavioural therapy for insomnia); 0 = waitlist control. Change in sleep is difference between T1 awakening length/ sleep efficiency and T2 awakening length/ sleep efficiency, measured with actigraphy. Increased sleep efficiency is positive change score. Reduced awakening length is negative change score. PM measured with the Western Australia PM Test (WAProm), converted to a residual score with age, gender and education correction. All analyses bootstrapped 5000 samples.
Table 3.6 shows linear moderated mediation analyses assessing whether – contingent on pre-intervention PM – receiving CBT-I was associated with better post-intervention PM, via the beneficial impact of CBT-I on reduced awakening length (Hypothesis 6) or increased sleep efficiency (Hypothesis 7). The overall models testing awakening length as a mediator of CBT-I’s effect on post-intervention PM were not significant for event-based PM, \( F(5, 15) = 1.66, p = .205 \), 36% variance explained (Model 9), or time-based PM, \( F(5, 15) = 1.63, p = .212 \), 35% variance explained (Model 10).

The overall models testing sleep efficiency as a mediator of CBT-I’s effect on post-intervention PM were not significant for event-based PM, \( F(5, 15) = 1.22, p = .349 \), 29% variance explained (Model 11), or time-based PM, \( F(5, 15) = 1.51, p = .244 \), 34% variance explained (Model 12).

**Discussion**

This pilot study assessed whether a course of CBT-I improved PM in older adults (primary outcome), via the beneficial impact of CBT-I on sleep (secondary outcome). Overall, we were not successful in improving objective sleep with CBT-I. Thus, our hypotheses that CBT-I would be associated with better post-intervention PM, via the beneficial impact of CBT-I on reduced average awakening length (Hypothesis 1) and increased sleep efficiency (Hypothesis 2) were not supported. In this discussion, we consider possible reasons why CBT-I was not associated with improved objective sleep in this study. Whilst this may be due to our participant inclusion criteria (use of sleep questionnaires to define sleep problems, rather than a formal diagnosis of insomnia), there is also some precedent to this finding in the literature, which will be discussed. We consider the implications of our results on our broader aims of improving PM, and evaluating whether there is a causal relationship between sleep disruption and poor PM. General limitations of this study are reviewed, in the hope that this pilot will prove
useful for informing future research. Overall, interpretation of our non-significant findings should be tempered by acknowledgement of the small sample size. This study serves predominately as a proof of concept and provides guidance for future procedures and analyses in forthcoming research. Indeed, following this study, the protocol for future research has been revised to a within subjects dose-response design.

Our results indicated an improvement in self-reported insomnia symptoms post-CBT-I. However, in our small sample, we were unable to demonstrate a positive effect of CBT-I on objectively measured awakening length or sleep efficiency. Additionally, in our group-level analyses (supplementary materials, pp. 167), we did not find effects of intervention condition on WASO or TST. The lack of improvement in objective sleep following CBT-I may have been due to our sample not being required to meet formal diagnostic criteria for insomnia (American Psychiatric Association, 2014). Instead, clinical cut-offs on two well-validated sleep symptom questionnaires (Bastien et al., 2001; Buysse et al., 1989) were used to define sleep problems. This approach has been taken in other CBT-I studies with older adults (Lovato et al., 2014; Martin et al., 2017), whilst some researchers have required participants to meet formal diagnostic criteria in order to be included (Irwin et al., 2014; Kay et al., 2015). Having less stringent criteria may have meant our participants had less severe sleep problems than individuals with a clinical diagnosis, and thus had less potential to show benefit following CBT-I. However, we also note there is no clear pattern of studies using formal insomnia diagnostic criteria having participants with more severe sleep problems at baseline, or showing more consistent benefit from treatment (Irwin et al., 2014; Kay et al., 2015; Lovato et al., 2014; Martin et al., 2017). Another factor which may have reduced the benefit of CBT-I was our observation that, by the beginning of the study, some participants’ rated their sleep problems as less severe than at recruitment. Overall, future research may benefit from applying stricter inclusion criteria (e.g. meeting formal
insomnia diagnostic criteria) and having less time between recruitment and the start of the study.

Whilst the lack of improvement in objective sleep might have been due to our inclusion criteria, some previous studies have also failed to find consistent effects of CBT-I on measures of objective sleep, especially in older adults, despite self-reported sleep typically improving (Irwin et al., 2014; Lovato et al., 2014). Similarly, in our study, despite the null findings for objective sleep, CBT-I was associated with a significant reduction in self-reported insomnia symptoms. Some research suggests self-reported improvement in the absence of objective sleep changes reflects tolerance for symptoms rather than recovery (Pillai et al., 2016). The extent to which this may apply to our study is unclear, as post-hoc comparison of our participants’ sleep with normative actigraphy data gives a mixed picture as to, firstly, whether participants had objective sleep problems pre-intervention, and secondly, whether their sleep continued to be objectively disturbed post-intervention. At pre-intervention, participants’ objective TST was below recommendations (420 – 480 minutes; Hirshkowitz et al., 2015), and their WASO was above the recommended range (0 – 40 minutes, although there is not a clear consensus on whether more than 40 minutes WASO is inappropriate for older adults; Ohayon et al., 2017). Their sleep efficiency, however, was within the normal range (>85%; Ohayon et al., 2017). After treatment, participants experienced normalisation of their TST regardless of whether they received treatment or not. WASO, however, did not significantly improve. In summary, it is not clear whether the discrepancy between subjective and objective sleep in our study is indicative of our participants’ experiencing tolerance rather than remission, and future research is needed in this area. Whilst these results from a small sample should not lead to dismissal of CBT-I as a treatment option for older adults, they do emphasize the importance of considering objective as well as self-report outcomes to better determine whether CBT-I leads to resolution of objective
sleep difficulties. Whilst resolution of subjective sleep difficulty may be an acceptable goal of CBT-I in some circumstances, this study addressed potential cognitive implications of objectively poor sleep, and so improving objective sleep was necessary. Future research might use objective sleep parameters in the inclusion criteria, as well as in outcome measurement, to better characterise how the symptoms of participants with objectively poor sleep change with treatment.

The discrepancy between improvement in subjective and objective sleep in this study could also be a function of expectation effects, due to our use of a waitlist control group rather than active control. As described by Boot and others (2013), participants who know they are receiving the treatment have an expectation of improvement and may thus be more motivated than control participants to perform better on outcome measures. This could explain why the CBT-I condition but not the control condition reported reduced insomnia symptoms after treatment. Future research should aim to assess and control expectation effects. This could be done by using a dose-response design (as is planned for future research in our lab), or an active control condition to reduce the effect of positive expectations, particularly for subjectively reported sleep. Possibilities for active control groups include relaxation/exercise programs to improve sleep such as tai chi, provision of education on sleep and sleep hygiene, or treatment with only one or some of the components of CBT-I, e.g. sleep restriction, cognitive restructuring or stress management.

In regards to our primary aim, we were not able to satisfactorily answer the question of whether CBT-I can improve PM in older adults. As objective sleep did not improve, our non-significant findings in regards to PM do not discount the possibility that improving sleep is still a viable pathway to improve PM. Furthermore, our study represents a successful proof of concept, in demonstrating that older adults can tolerate the pre and post-assessment procedures and intervention with minimal dropout.
Additionally, our experience may prove useful for guiding future research, in terms of participant inclusion criteria. As discussed, having more stringent criteria around participants’ sleep problems might increase the benefits of CBT-I on objective sleep. Similarly, in our study, the majority of participants had better than average baseline performance on the PM task, and only two participants performed below the 25th percentile, based on a normative sample (supplementary materials, pp. 163). Thus, specifically recruiting participants with PM deficits might result in clearer benefits for PM following CBT-I. Whilst this study included moderated mediation analyses to try to assess the impact of baseline PM on response to treatment, the small sample affected power to show effects. Overall, future studies might benefit from specifically recruiting participants with sleep and PM problems that are more evident, who thus might benefit more from intervention.

Another consideration for future research is the sensitivity of the PM measure to change. The PM measure used in this study – the WAProm – is a newly developed test that has not yet been used in a repeated measures design. However, the WAProm closely follows the format of the Memory for Intentions Screen Test (MIST; Raskin, 2009), which is sensitive to change (Fleming, Shum, Strong, & Lightbody, 2005). Future research might consider using other measures of PM that allow a more fine-grained analysis of reaction time and ongoing task performance to detect change (e.g. Einstein & McDaniel, 1990). However, it must be noted that this may come at a cost to generalizability, ecological validity and burden on participants, which were all factors considered when selecting the WAProm for this study.

A second aim of this study was to draw conclusions regarding a potential causal relationship between sleep and PM, by investigating whether manipulating sleep with CBT-I would affect PM. Ultimately, as our manipulation was not successful, we cannot draw conclusions about whether this study supports a causal relationship between sleep
and PM. More research is needed to test whether habitual sleep disruption experienced by older adults affects PM, potentially using longitudinal and experimental designs. One possibility hinted at by our finding that CBT-I did not improve awakening length or sleep efficiency is that older adults’ objective sleep disturbance may be more resistant to treatment than younger adults. If so, this could suggest a more stable, neurological basis for poor sleep in older adults, for example age-related atrophy in the medial prefrontal cortex resulting in reduced slow-wave activity (Mander et al., 2013), or reduced grey matter volume in the orbitofrontal cortex and inferior frontal gyri contributing to sleep fragmentation (Lim et al., 2016). If disturbed sleep has a neurological basis in some older adults, this could relate to co-existing cognitive problems through interruption of sleep-dependent restorative processes supporting healthy cognition (Anderson & Horne, 2003; Lafortune et al., 2014; Mendelsohn & Larrick, 2013). Furthermore, the neurological changes that interfere with sleep might have broader consequences for brain networks that support cognition (Mander, 2013; Yaffe et al., 2014). These two possibilities are not necessarily mutually exclusive: age-related neurological brain changes might result in both disturbed sleep and poor cognition, with cognitive problems being further exacerbated by the consequences of poor sleep. These possible scenarios need further exploration in future research with larger samples, to determine if older adults’ sleep disturbance is indeed more resistant to treatment with CBT-I, and whether treatment resistance is related to neurological changes in brain regions that support consolidated sleep.

This study also aimed to address limitations in previous research by exploring whether the benefits of CBT-I extend beyond subjective sleep, namely objective sleep and cognition. As emphasized in reviews of the CBT-I literature (Harvey & Tang, 2003), insomnia disorder is defined by impact on daily functioning (American Psychiatric Association, 2014), highlighting the need for research on CBT-I to show benefits of
treatment in areas other than sleep. Additionally, research suggesting that self-reported recovery from sleep problems may in fact just reflect tolerance reinforces the need to show objective sleep improvements (Pillai et al., 2016). Unfortunately, this study was not able to show objective improvement in sleep or cognition despite delivering a comprehensive, manualized version of CBT-I through trained therapists, under supervision from a clinical psychologist with expertise in insomnia treatment. Our findings reinforce that it is not a given that improvement in subjective ratings of insomnia symptoms will correspond to objective improvement in sleep and daily function. This highlights the need for researchers and clinicians to consider the target of their treatment approaches, and strike a balance between restructuring unreasonable expectations of sleep and promoting actual sleep improvement, especially for objectively poor sleepers (Pillai et al., 2016).

As well as having less stringent inclusion criteria in regards to severity of problems with sleep and PM, there are some other general limitations of this research. As a pilot study, our sample size was small and this constrained our ability to detect effects, as evident in our regression analyses where models were non-significant, despite the high proportions of variance explained. Another significant limitation is the difficulties we experienced with randomisation, in that some participants insisted on allocation to their preferred condition. This limitation of the pilot is now addressed in the protocol for future research, which utilises a within-study, dose-response design. Finally, the ISI was used to monitor progress in therapy, as well as being an outcome measure. Thus, CBT-I participants were exposed multiple times to the questionnaire. Whilst we would have preferred to use a different outcome measure for subjective sleep, our small sample meant that missing data on other measures (namely the PSQI) limited us to using the ISI. Having a larger sample in future research should overcome this
issue with missing data. Despite these issues, we feel this study provided an important contribution by generating important points for consideration in future studies.

To summarise from above, our experiences with this pilot study have led to several recommendations for future research in this area. Regarding trial design, there is a need to better control expectation effects and improve recruitment, which could be done with an active control group or a within subjects dose-response design. Regarding inclusion criteria, future research should consider requiring participants to meet formal diagnostic criteria for insomnia, defining poor sleep with objective measures (such as actigraphy), and recruiting participants with PM complaints. Additional outcome measures could be incorporated into future studies to help better understand the causal relationships between sleep and PM. Specifically, the collection of brain imaging data could help ascertain whether there are neurological underpinnings to poor sleep and poor PM in older adults. A longitudinal study would be the optimal way to explore causality in the links between sleep and PM. The addition of other PM measures (e.g. self and informant report of PM in daily life, computerised PM paradigms) should also be considered when designing future studies.

In conclusion, whilst we were unable to fully satisfy our aims, this pilot study was informative in guiding future research into whether CBT-I can be used to improve PM. Whilst our results were non-significant in regards to demonstrating changes in objective sleep, we feel this underscores rather than discounts the importance of considering objective sleep. Our findings suggest subjective improvement in insomnia symptoms is not equivalent to objective improvement in sleep parameters, and is not associated with improvement in cognition. Future research should continue to focus on objective sleep as an outcome (and potentially also as inclusion criteria) to ensure improvement in symptoms reflects actual recovery, rather than tolerance.
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Foreword to Chapter 4.

The preceding two chapters have evaluated sleep as a potential avenue to intervention for older adults’ prospective memory. This thesis now turns to another opportunity for intervention: a compensatory executive function intervention to improve prospective memory in older adults. Chapter 1 reviewed literature on existing prospective memory interventions for older adults, and concluded that the majority of studies have focused on the retrospective memory component of prospective memory. This is despite the emphasis on executive dysfunction as a driver of age-related prospective memory decline. The following chapter thus helps fill a gap in the literature by evaluating a compensatory executive function intervention for prospective memory. This chapter was written for submission to a peer-reviewed journal.
CHAPTER 4

Improving Prospective Memory Performance in Community-Dwelling Older Adults: Goal Management Training and Implementation Intentions
Abstract

Prospective memory (the ability to remember and execute a deferred task in the future) declines with age, with significant implications for older adults’ activities of daily living and quality of life. Prospective memory interventions have focused primarily on the retrospective component of prospective memory (e.g., implementation intentions). However, executive dysfunction is also implicated in age-related prospective memory decline. Thus, we tested a compensatory executive intervention for prospective memory (goal management training) for the first time in older adults, compared to an established intervention (implementation intentions) or no intervention. Community-dwelling older adults (58-92 years; N = 157) were randomly allocated to receive goal management training, implementation intentions or no intervention. Prospective memory was assessed before and after the intervention with a well-validated laboratory-based prospective memory measure. Implementation intentions relative to no intervention improved prospective memory for participants with poorer baseline prospective memory. Participants who received goal management training were more likely to have difficulty comprehending the intervention. For those participants who comprehended goal management training, prospective memory improved relative to no intervention, but only for older adults with poorer baseline prospective memory. Direct comparison of implementation intentions and goal management training did not suggest significant differences between the interventions, either overall or in analyses accounting for baseline prospective memory ability or instruction comprehension. Older adults may have difficulty effectively using goal management training delivered in a single brief session to improve prospective memory. Whilst compensatory interventions may be most useful for older adults with poorer prospective memory, potential underlying cognitive deficits may interfere with this group’s ability to learn a complex, multi-step strategy like goal management training without significant effort.
Introduction

Prospective Memory (PM) is the ability to remember and execute a task in the future, which requires intact retrospective memory and executive function (McDaniel & Einstein, 2011). Older adults experience decline in PM, significantly impacting their activities of daily living and quality of life (Woods, Weinborn, Velnoweth, Rooney, & Bucks, 2012). Thus, it is vital to find ways of successfully intervening to improve older adults’ PM. One well-established strategy to compensate for PM deficits is to support the retrospective memory component of PM using a rehearsal strategy known as implementation intentions (Chen et al., 2015). Conversely, interventions targeting the executive function aspects of PM have not been well researched. The present study aimed to evaluate the efficacy of an executive compensation approach, specifically goal management training (GMT), in improving PM performance amongst community-dwelling older adults, in comparison to implementation intentions.

Successful PM performance requires forming a mental link between a PM task and a particular environmental cue, subsequently monitoring the environment for the PM cue whilst completing other activities, successfully detecting the cue, disengaging from other activities, and recalling and executing the PM task (Kliegel, Martin, McDaniel, & Einstein, 2002). Other cognitive abilities supporting each of these steps are thus important for PM. Retrospective memory is important for successfully encoding the link between the cue and the PM task, and later recalling it when needed (West, 2005). Elements of executive function are important for planning the fulfilment of the PM task, monitoring the environment for the cue, controlling memory retrieval, and activating appropriate behaviours after cue detection (Kliegel et al., 2002). These elements of executive function can be conceptualised as guided by the overarching ability to maintain intentions in goal-directed behaviour (Duncan, 1995), that is, to maintain the goals of the PM task in conjunction with ongoing task goals. As executive
functions and retrospective memory play important roles in PM performance, age-related declines in these two abilities are suspected to underlie age-related PM deficits (McDaniel & Einstein, 2011; Schnitzspahn, Stahl, Zeintl, Kaller, & Kliegel, 2013). Indeed, older adults identify PM failures as among their most frequent and concerning cognitive problems (McDaniel & Einstein, 2007).

Despite the impact of PM failures on older adults’ daily life, research on PM intervention for older adults is still in its infancy. Currently, the approach most consistently shown to improve PM in older adults is implementation intentions (Chen et al., 2015). Implementation intentions involve forming and rehearsing a verbal statement and/or visualisation that makes explicit the link between a PM task and a cue. For example, a person may form and repeat the statement, “when the evening news starts [cue], then I will take my medication [task]”, and visualise this taking place. Implementation intentions are assumed to strengthen the mnemonic link between cue and PM task, supporting encoding and retrieval and thus improving older adults’ PM performance by compensating for retrospective memory decline (Chen et al., 2015; McFarland & Glisky, 2011). By functioning as an internal and compensatory strategy, implementation intentions reduce reliance on external supports (e.g. reminders from external devices [Thöne-Otto & Walther, 2003] which have to be programmed and may be lost, and avoid the criticism levelled at remediation interventions (repeated practice on PM tasks or specific components of PM) for often failing to generalise (Hering, Rendell, Rose, Schnitzspahn, & Kliegel, 2014). Whilst implementation intentions successfully target the memory component of PM, compensatory interventions targeting executive components of PM have not been well studied in older adults, despite the significant contribution of executive dysfunction to PM failures (Schnitzspahn et al., 2013; West, 2005).

In clinical populations, PM failures have been addressed by using a
compensatory strategy targeting executive dysfunction known as GMT (Mahan, Rous, & Adlam, 2017; Robertson, 1996). GMT supports the maintenance of PM intentions and their activation at the appropriate time upon encountering the cue. GMT also provides prompts for the elements of executive function that are required for successful PM. Participants are taught the steps to successful PM goal completion, including periodically interrupting their cognitive processing of the ongoing task, directing their attention to overarching goals, planning the achievement of these goals, and monitoring performance (Stamenova & Levine, 2018). Flowcharts, checklists and metaphors are used to train these steps, and content-free cues to trigger reviewing of goal states (Krasny-Pacini, Chevignard, & Evans, 2014; Levine et al., 2000). Despite the emphasis placed on executive declines in explaining age-related PM deficits (Schnitzspahn et al., 2013), no studies have yet evaluated the ability of GMT to improve PM amongst older adults.

GMT has been widely used to support general executive deficits (Stamenova & Levine, 2018), however, just two studies have evaluated GMT for PM, both among individuals with brain injury. In the first study, participants were taught GMT, assisted by errorless learning techniques and brief psychoeducation, in a single 30-minute one-on-one session. Participants subsequently completed a time-based PM task: phoning the experimenters at set times over 10 days (Fish et al., 2007). When participants were provided with content-free cues to remind them to use GMT, their PM performance significantly improved. In the second study, participants received an intervention containing components of GMT (a content-free cue to review goal states), but did not demonstrate subsequent improvement in PM (Sweeney, Kersel, Morris, Manly, & Evans, 2010). These discrepant findings notionally support the promise of GMT for improving PM performance, but underscore the need for explicit training in all components of GMT (provided in Fish et al. [2007] but not in Sweeney et al. [2010]).
Building on these findings, the current study sought to examine the efficacy of GMT to improve PM performance in older adults. We compared the PM performance of participants who received GMT to those who received no intervention, and a group trained in an established intervention: implementation intentions. We measured PM performance before and after the intervention with an objective and well-validated laboratory PM task (Einstein & McDaniel, 1990) to control for differences in baseline PM ability. As suggested by previous research, our application of GMT included explicit training in all components and content-free cues. We successfully piloted our version of GMT in an unpublished study of PM and depression in young adults (Li, 2014). We included the implementation intentions condition to ensure participants were capable of responding to an established intervention by showing improvement in PM on our particular task.

We were also interested in how an intervention targeting the executive components of PM (GMT) would compare with one targeting the retrospective components (implementation intentions). GMT is considered an executive intervention because it focuses on planning, directing attention to overarching goals, and monitoring behaviour. Implementation intentions is considered a retrospective memory intervention because it aims to strengthen encoding. However, implementation intentions could also be conceptualised as supporting executive functions in PM because potential consequences of strengthened encoding are reduced demands on monitoring, and automatized switching from the ongoing to the PM task. Similarly, one component of GMT involves an attempt to strengthen encoding as participants are prompted to ‘learn’ the steps to successful goal achievement. Nevertheless, we contrast these two interventions in an effort to gauge the utility of focusing more on executive functions or more on retrospective memory when supporting PM in older adults.

We hypothesised that implementation intentions would predict significantly
better post-intervention PM accuracy than no intervention (Hypothesis 1), and that GMT would also predict significantly better post-intervention PM accuracy than no intervention (Hypothesis 2). We also hypothesised that GMT would predict significantly better post-intervention PM accuracy than implementation intentions (Hypothesis 3), based on research suggesting executive dysfunction (supported by GMT) is a greater contributor to age-related PM decline than retrospective memory (supported by implementation intentions; McDaniel & Einstein, 2011).

Method

Participants

Community-dwelling older adults (recruited through the West Australian Participant Pool, Director: R. S. Bucks, and Western Australia Memory Study, Australian Alzheimer’s Research Foundation, Research Director: R. N. Martins) attended the Healthy Ageing Research Program at the University of Western Australia. The study was approved by the Human Ethics Office at the University of Western Australia, and conducted in accordance with the guidelines of the Helsinki Declaration. Participants gave written informed consent to have their anonymised data included in the study. Participants \( N = 194 \) were randomised to an intervention condition (GMT, implementation intentions, or no intervention) and 172 participants went on to complete the PM task. Randomisation was through the assignment of a study number linked to randomisation sequence when participants confirmed their participation. Figure 4.1 shows inclusion tracking for each condition. Participants were excluded from further analysis if: their PM data were invalid based on examiner report, poor accuracy on the ongoing task (< 60%) or excessive PM false alarms (> 50); they achieved a score below age/education defined cut-offs (Crum, Anthony, Bassett, & Folstein, 1993) on the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975); they reported severe neurological or psychiatric conditions (e.g. Parkinson’s disease, stroke,
schizophrenia), previous loss of consciousness over 30 minutes, or concerning levels of alcohol use (> 15 on the Alcohol Use Disorders Identification Test [AUDIT] Babor, Higgins-Biddle, Saunders, Monteiro, & World Health Organization, 2001). Participants were also required to speak fluent English. After applying these criteria, 157 participants were included in the analysis.
Participant flowchart.

Randomised
$N = 223$

Attended
$N = 194$

No intervention
$N = 64$

Implementation intentions
$N = 60$

Goal management training
$N = 70$

Did not complete
$N = 7$
(not appropriate $N = 2$; computer error $N = 5$)

Did not complete
$N = 7$
(not appropriate $N = 1$; computer error $N = 4$; time constraints $N = 2$)

Did not complete
$N = 8$
(not appropriate $N = 2$; computer error $N = 4$; time constraints $N = 2$)

Complete
$N = 57$

Complete
$N = 53$

Complete
$N = 53$

PM data not valid
$N = 2$

Valid
$N = 55$

Valid
$N = 53$

PM data not valid
$N = 1$

Included
$N = 53$

Included
$N = 49$

Included
$N = 4$

Included
$N = 6$

Included
$N = 55$

Note: 1: Whilst we set out to have an even distribution of participants in each condition, not all participants who were allocated a condition at recruitment attended their visits.

2: PM (prospective memory) data validity: ongoing task accuracy > 60%; PM false alarm rate < 50; no experimenter report of invalid performance.

3: Inclusion criteria: score above age/education defined cut-offs on the Mini-Mental State Examination (MMSE; Folstein, et al., 1975), no reported severe neurological or psychiatric conditions, previous loss of consciousness over 30 minutes, or concerning levels of alcohol use (> 15 on the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 2001); fluent English speaker.

Procedure

Participants were contacted by telephone, completed the study questionnaires at home and then attended the study centre to complete the PM task as part of a larger neuropsychological battery (approx. 3.5 hours; see General Methods in Supplementary
Materials. This battery included the MMSE (Folstein et al., 1975) and questionnaires on demographics, medical history, and alcohol use, to determine inclusion as described in the Participants section. The battery also included the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, Tierney, Mohr, & Chase, 1998) and the Trail Making Test A and B (TMT A and B; Reitan & Wolfson, 1985) which were used to describe the samples’ overall cognitive ability (RBANS Total Scaled Score), retrospective memory (RBANS Delayed Memory Index) and executive functioning (TMT A B difference score, converted to Z-score as per Tombaugh in Strauss, Sherman, & Spreen, 2006). Assessment and intervention were conducted by graduate students in the School of Psychological Science, supervised by certified clinical psychologists and neuropsychologists. Assessors were not able to be blinded to intervention condition, as they were present when participants completed the PM assessment and intervention. However, PM was assessed pre and post intervention through a computerised paradigm, reducing the risk of bias.

The procedure for the PM task and intervention is shown in Figure 4.2. PM was assessed with a typical, laboratory PM paradigm (Einstein & McDaniel, 1990) where a PM task is embedded in an ongoing lexical decision task. Participants received ongoing task instructions, completed 10 practice trials, then completed the baseline ongoing task (block 1). Participants then took a 30-second break and received PM task instructions, followed by a distractor task to limit rehearsal of these instructions. Participants then completed the pre-intervention PM task (block 2). Next, PM task instructions were repeated, and participants received instructions for GMT, implementation intentions or no intervention. Participants in the no intervention or implementation intentions condition were told to ignore the stop sign graphic that appeared in the post-intervention PM task. This graphic was part of the GMT intervention (described below) and, as it appeared periodically for 15 seconds, it was presented to all participants to avoid those
in the GMT condition having the advantage of regular task breaks. Participants then completed another distractor task, and the post-intervention PM task (block 3). The PM assessment and intervention took approximately 30mins in total.
Figure 4.2.
Procedure for prospective memory measure and intervention.

Note: PM: prospective memory, GMT: goal management training, II: implementation intentions.
**Prospective memory measure and intervention**

**Ongoing task**

The task was presented on a standard computer with E-Prime 2.0 Software (Psychology Software Tools, Pittsburgh, PA). Participants were required to indicate quickly and accurately whether a letter string presented on the computer screen was a word or non-word. Responses were made on the computer keyboard. When the letter string was a word (e.g., ‘house’) participants were required to press the L key, covered by a “Y” sticker for ‘yes’. When the letter string was not a word (e.g., ‘plomp’) participants were required to press the A key, covered by an “N” sticker for ‘no’.

Word stimuli for the ongoing task were created from an Australian database of medium frequency 4-8 letter words (Dennis, 1995). Non-words were created from words in the same database by swapping the vowels so that they did not closely resemble, visually or phonetically, any English word, but were still pronounceable. Block 1 had 50 words and 50 non-words, and blocks 2 and 3 each had 92 words and 100 non-words. Each block began with an instruction “Get ready”, a 3s countdown on screen, a blank screen presented for 100ms, a fixation cross for 500ms, a blank screen for 250ms, and then the first letter string. Subsequent letter strings were separated by fixation crosses (500ms) and presented for 2250ms or until a response was made.

Ongoing task accuracy for each block was calculated as the proportion of correct responses to word/non-word trials. Average ongoing task response time (RT) for each block was calculated from correct trials only.

**Prospective memory task**

The PM task embedded in the word/non-word judgment task required participants to press an alternative key – the “6” key, covered by an “X” sticker –

\[\text{See supplementary materials for calculation of PM costs and analyses.}\]
whenever they were presented with a word from a target semantic category. For half the participants, the PM target semantic category was “fruit” (plum, grape, cherry, mango, banana, peach, pear, melon) in block 2 and “animal” (rabbit, sheep, mouse, tiger, giraffe, goat, elephant, zebra) in block 3, and the reverse was true for the other half of participants. Fruit/animal words were medium frequency and contained 4-8 letters. Each PM block had eight PM cues from the target semantic category randomly inserted once every 25 trials, but not in the first 5 trials.

In the post-intervention PM task (block 3), a stop sign with the GMT steps listed beneath it appeared for 15s after the 50th, 100th, and 150th trials. The title of each step was listed (“Stop, Define, List, Learn, Check”) but without any other descriptive information on what the step required, so participants in the no intervention or implementation intention conditions were unlikely to be able to spontaneously use GMT strategies.

To compare the effects of intervention condition on PM, pre-intervention and post-intervention PM accuracy (primary outcome) were calculated as the proportion of correct responses to PM cues in block 2 and block 3 respectively.

To assess comprehension of the intervention instructions (secondary outcome), examiners noted any relevant participant comments or behaviour. Two independent raters converted these examiner comments into a dichotomous ‘confusion’ variable, coded ‘1’ if a participant stated they were confused or misinterpreted instructions during intervention delivery or the post-intervention task, and otherwise coded ‘0’. Raters resolved discrepancies in their coding by discussion and consensus decision.

**Distractor task**

The distractor task used to prevent rehearsal of the instructions was a set of arithmetic problems that started simply (e.g., “2 + 2”), and became progressively more difficult (e.g., “306 x 109”). Participants were not given a calculator, but were reassured
the problems were designed to be difficult and were not expected to all be completed in the time provided.

**Implementation intentions training**

Implementation intentions training involved a visual and a verbal component\(^3\) (see supplementary materials for training script, pp. 171). Participants were first asked to imagine themselves seeing the PM cue, making a word/non-word judgment, and then pressing the “X” key. They were prompted to practice this visualisation for 30s. They then had to state out-loud three times, “When I see a fruit/animal word, I will press the ‘X’ key”.

**GMT training**

GMT involved a teaching component, a cueing component, and a rehearsal component (see supplementary materials for training script, pp. 171). First, participants were taught a five step strategy to support maintenance of goals and their successful completion with the aid of a flow-chart, and examples of how they should apply it to the PM task (Levine et al., 2000). These steps prompted directed attention, planning, executive control of memory processes, and monitoring. The five steps were:

1. **Stop**, pause and think about what you are doing,
2. **Define** the main goal,
3. **List** the sub-goals to achieve the main goal,
4. **Learn**, review and consolidate the first three steps, and
5. **Check** that you are on task.

Next, participants were introduced to the content-free cueing to trigger reviewing of goal states. They were told a stop sign graphic would appear during the PM task to remind them to review the five GMT steps. Finally, participants rehearsed GMT by completing a quiz testing their memory for each of the five steps. Incorrect
responses were followed by corrective feedback and repetition of the question. Once they had passed the quiz, participants completed a 20-trial practice of the PM task, with the stop sign appearing after the 10th trial and the PM cue at the 18th trial.

**Statistical analysis**

One-way analysis of variance (ANOVA) was used to compare intervention conditions on demographics, other cognitive variables, baseline ongoing task performance and pre-intervention PM task performance. Linear regression in SPSS was used to test the effect of intervention condition on post-intervention PM accuracy, controlling for pre-intervention PM accuracy (Hypotheses 1 - 3). For the regression analyses comparing implementation intentions to no intervention, and GMT to no intervention, condition was parameterised as 0 = no intervention and 1 = intervention condition, either implementation intentions or GMT. For the regression analyses comparing GMT to implementation intentions, condition was parameterised as 0 = implementation intentions and 1 = GMT. Statistical significance was set at .05 (two-tailed), and all analyses calculated using 5000 bootstrapped samples and bias-corrected 95% confidence intervals (CI).

**Results**

**Primary analysis**

Sample characteristics and descriptive statistics are presented in Table 4.1. There were no pre-existing differences between intervention conditions in age, education, gender, MMSE score, RBANS Total Scale Score, RBANS Delayed Memory Index or TMT A B difference Z-score. There was a significant intervention condition difference at baseline in the ongoing task RT. Participants who went on to receive implementation intentions had significantly slower baseline RTs than those who received GMT, $F(2,154) = 3.932, p = .022$, mean difference: 81ms. There were no other differences in RT or accuracy for either the baseline ongoing task or pre-intervention PM task. There
were no differences in post-intervention PM task costs between the intervention conditions, as described in the supplementary materials, pp.169.

Participants’ overall cognitive ability (RBANS Total Scaled Score) and retrospective memory (RBANS Delayed Memory Index) were comparable to normative samples (Randolph et al., 1998). Executive functioning (TMT A B difference score, converted to Z-score) was somewhat better than normative samples, but still within one standard deviation (SD) of population averages (Strauss et al., 2006). With regards to extreme scores, two participants had RBANS delayed memory index scores two SDs below the mean (one in the no intervention condition had a score of 68, and one in the II condition had a score of 64). Two participants had TMT A B difference Z-scores two SDs below the mean (both in the GMT condition: -3.94 and -2.47). The neuropsychological profiles of these participants were examined and none had other indications of broader cognitive impairment beyond these isolated low scores.
Table 4.1.
Sample characteristics and descriptive statistics.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>No intervention condition (N = 53)</th>
<th>II condition (N = 49)</th>
<th>GMT condition (N = 55)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Min - max</strong></td>
<td><strong>M (SD)</strong></td>
<td><strong>M (SD)</strong></td>
<td><strong>M (SD)</strong></td>
</tr>
<tr>
<td>Age (years)</td>
<td>58 – 92</td>
<td>72.78 (7.25)</td>
<td>72.34 (7.88)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>3 – 2.5</td>
<td>13.79 (3.41)</td>
<td>14.17 (3.11)</td>
</tr>
<tr>
<td>MMSE total score</td>
<td>22 – 30</td>
<td>28.23 (1.48)</td>
<td>28.28 (1.29)</td>
</tr>
<tr>
<td>RBANS Total Scale Score*</td>
<td>76 – 128</td>
<td>103.04 (12.01)</td>
<td>102.82 (13.23)</td>
</tr>
<tr>
<td>RBANS Delayed Memory Index Score**</td>
<td>64 – 126</td>
<td>102.32 (11.22)</td>
<td>100.08 (12.59)</td>
</tr>
<tr>
<td>TMT A B Difference Z-score***</td>
<td>-3.94 – 1.74</td>
<td>0.66 (0.80)</td>
<td>0.71 (0.73)</td>
</tr>
<tr>
<td>Percentage male</td>
<td>29</td>
<td>36</td>
<td>20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ongoing task descriptives</th>
<th>Full sample (N = 157)</th>
<th>No intervention condition (N = 53)</th>
<th>II condition (N = 49)</th>
<th>GMT condition (N = 55)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline OT accuracy (proportion correct)</td>
<td>.88 – 1.00</td>
<td>.97 (.02)</td>
<td>.97 (.02)</td>
<td>.98 (.01)</td>
</tr>
<tr>
<td>Baseline OT RT (ms)</td>
<td>529.66 – 1377.48</td>
<td>793.91 (150.22)</td>
<td>798.13 (165.51)</td>
<td>834.55 (159.70)</td>
</tr>
<tr>
<td>Pre-intervention OT accuracy (proportion correct)</td>
<td>.83 – 1.00</td>
<td>.96 (.02)</td>
<td>.97 (.01)</td>
<td>.96 (.01)</td>
</tr>
<tr>
<td>Pre-intervention OT RT (ms)</td>
<td>647.46 – 1379.94</td>
<td>865.07 (134.88)</td>
<td>875.36 (160.22)</td>
<td>878.22 (129.11)</td>
</tr>
<tr>
<td>Post-intervention OT accuracy (proportion correct)</td>
<td>.90 – 1.00</td>
<td>.97 (.01)</td>
<td>.96 (.02)</td>
<td>.97 (.02)</td>
</tr>
<tr>
<td>Post-intervention OT RT (ms)</td>
<td>632.08 – 1426.13</td>
<td>840.55 (120.62)</td>
<td>847.81 (147.26)</td>
<td>844.22 (101.81)</td>
</tr>
</tbody>
</table>
### PM task descriptives

<table>
<thead>
<tr>
<th></th>
<th>Min - max</th>
<th>Full sample $(N = 157)$</th>
<th>No intervention condition $(N = 53)$</th>
<th>II condition $(N = 49)$</th>
<th>GMT condition $(N = 55)$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
</tr>
<tr>
<td>Pre-intervention PM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>accuracy (proportion correct)</td>
<td></td>
<td>.81 (.19)</td>
<td>.83 (.18)</td>
<td>.81 (.19)</td>
<td>.79 (.21)</td>
</tr>
<tr>
<td>Post-intervention PM</td>
<td></td>
<td>.80 (.17)</td>
<td>.79 (.16)</td>
<td>.84 (.15)</td>
<td>.78 (.19)</td>
</tr>
<tr>
<td>PM accuracy (proportion correct)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Count of PM task examiner comments

| Confused (count) | No | 140 | 50 | 47 | 43 |
| Yes             |    | 17  | 3  | 2  | 12 |

*Note: M: mean; SD: standard deviation; II: implementation intentions; GMT: goal management training; MMSE: Mini-Mental State Examination (Folstein, Folstein & McHugh, 1975); RBANS: The Repeatable Battery for the Assessment of Neuropsychological Status (Randolph, Tierney, Mohr, & Chase, 1998); TMT A B Difference Z-score: Trail Making Test B score subtract Trail Making Test A score (Reitan & Wolfson, 1985) converted to Z-score (as per Tombough in Strauss, Sherman, & Spreen, 2006), OT: ongoing task; RT: reaction time; ms: milliseconds; PM: prospective memory.

*Missing data by condition: no intervention 2, II 3, GMT 0. **Missing data by condition: No intervention 2, II 2, GMT 0. ***Missing data by condition: No intervention 3, II 1, GMT 4.
To evaluate the primary outcome of PM performance, linear regression models testing the effect of intervention condition on post-intervention PM accuracy, controlling for pre-intervention PM accuracy, are presented in Table 4.2-i. Contrary to prediction (Hypothesis 1), whilst the overall model was significant, $F(2, 99) = 9.22, p < .001$, there was no association between receiving training in implementation intentions and better post-intervention PM, in comparison to no intervention, after controlling for pre-intervention PM, albeit the effect was at trend ($p = .055$). Likewise, when compared to no intervention, GMT (Hypothesis 2) did not produce significantly higher post-intervention PM accuracy, albeit the overall model was significant, $F(2, 105) = 10.97, p < .001$. Finally, there was no significant difference in post-intervention PM accuracy between GMT and implementation intentions (Hypothesis 3), although the overall model was significant, $F(2, 101) = 5.59, p = .005$. 


### Table 4.2.

Linear regression predicting post-intervention prospective memory accuracy with intervention condition, controlling for pre-intervention prospective memory accuracy.

<table>
<thead>
<tr>
<th>Model number</th>
<th>Step</th>
<th>Predictor 1</th>
<th>Predictor 2</th>
<th>R²</th>
<th>R² change (p)</th>
<th>Unstandardised coefficient (bootstrapped CIs 5000 samples)</th>
<th>Standardised coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N = 102</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.13</td>
<td></td>
<td>0.31 (0.16 – 0.53)</td>
<td>.35</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td></td>
<td>0.32</td>
<td>(0.17 – 0.53 )</td>
<td>.36</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention condition</td>
<td></td>
<td>.16</td>
<td>.03</td>
<td>0.32 (0.17 – 0.53 )</td>
<td>.36</td>
</tr>
<tr>
<td></td>
<td></td>
<td>II = 1, No intervention = 0</td>
<td></td>
<td></td>
<td></td>
<td>0.06 (-0.002 – 0.12)</td>
<td>.18</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.17</td>
<td></td>
<td>0.37 (0.10 – 0.72)</td>
<td>.42</td>
</tr>
<tr>
<td>2. N = 108</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.17</td>
<td>0</td>
<td>0.38 (0.10 – 0.68)</td>
<td>.42</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td></td>
<td></td>
<td>0.01 (-0.05 – 0.07)</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention condition</td>
<td></td>
<td>.17</td>
<td>.0</td>
<td>(p =.882)</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GMT = 1, II = 0</td>
<td></td>
<td></td>
<td></td>
<td>0.01 (-0.05 – 0.07)</td>
<td>.01</td>
</tr>
<tr>
<td>3. N = 104</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.08</td>
<td></td>
<td>0.24 (-0.01 – 0.54)</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.10</td>
<td>.02</td>
<td>0.24 (-0.02 – 0.53)</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention condition</td>
<td></td>
<td></td>
<td></td>
<td>(p =.101)</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GMT = 1, II = 0</td>
<td></td>
<td></td>
<td></td>
<td>-0.06 (-0.12 – 0.01)</td>
<td>-.16</td>
</tr>
</tbody>
</table>
### ii. Sensitivity analysis

<table>
<thead>
<tr>
<th>Model number</th>
<th>Step</th>
<th>Predictor 1</th>
<th>Predictor 2</th>
<th>R²</th>
<th>R² change (p)</th>
<th>Unstandardised coefficient (bootstrapped CIs 5000 samples)</th>
<th>Standardised coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. N = 97</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.15</td>
<td></td>
<td><strong>0.34</strong> (0.19 – 0.58)</td>
<td>.39</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td>Intervention condition</td>
<td>.19</td>
<td>.04</td>
<td><strong>0.35</strong> (0.21 – 0.60)</td>
<td>.41</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>II = 1, No intervention = 0</td>
<td></td>
<td></td>
<td><strong>0.06</strong> (0.01 – 0.12)</td>
<td></td>
</tr>
<tr>
<td>5. N = 93</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.10</td>
<td></td>
<td><strong>0.28</strong> (0.06 – 0.64)</td>
<td>.32</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td>Intervention condition</td>
<td>.11</td>
<td>.01</td>
<td><strong>0.29</strong> (0.07 – 0.64)</td>
<td>.33</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>GMT = 1, No intervention = 0</td>
<td></td>
<td></td>
<td>0.03 (-0.03 – 0.09)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. N = 90</td>
<td>1</td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td>.02</td>
<td></td>
<td>0.12 (-0.03 – 0.34)</td>
<td>.15</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Pre-intervention PM accuracy</td>
<td>Intervention condition</td>
<td>.03</td>
<td>.01</td>
<td>0.12 (-0.04 – 0.35)</td>
<td>.15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>GMT = 1, II = 0</td>
<td></td>
<td></td>
<td>-0.03 (-0.10 – 0.03)</td>
<td>-.10</td>
</tr>
</tbody>
</table>

Note: CIs: confidence intervals, if CIs do not cross zero the effect is significant at p < .05. Significant effects in bold. Primary analyses include all participants; Sensitivity analyses exclude participants who experienced confusion during intervention training or post-intervention PM task. PM: prospective memory. II: implementation intentions. GMT: goal management training.
Given the unexpected non-significant effects of intervention on PM performance, we conducted additional post-hoc analyses to better describe response to intervention in different cohorts. After observing that baseline PM accuracy in our sample was overall reasonably high but certainly not at ceiling (81%), and noting that previous research had concentrated on clinical samples (Levine et al., 2000), we considered whether the PM intervention effects might be more apparent for participants with poorer baseline PM. We thus used Conditional Process Analysis for SPSS Version 3.1 (Hayes, 2013) linear moderation analysis to test the potential moderating role of pre-intervention PM accuracy on the effect of intervention condition on post-intervention PM accuracy.

As shown in Table 4.3-i, the effect of implementation intentions on post-intervention PM accuracy, relative to no intervention, was indeed moderated by pre-intervention PM accuracy, $F(3, 98) = 7.80, p < .001$. Follow-up comparisons of the effect of implementation intentions at different levels of pre-intervention PM revealed that participants with relatively poor baseline PM (-1 SD from the mean) improved on average 18%; significantly better than the no intervention condition, $b = 0.12, SE = 0.04, p = .005$. Those scoring at the mean for baseline PM accuracy improved marginally (average improvement 3%) relative to no intervention, with a trend-level effect, $b = 0.06, SE = 0.03, p = .051$. Whilst those with the best baseline PM accuracy (+1SD from the mean) performed no differently in terms of post-intervention PM than those in the no intervention condition, $b = 0, SE = 0.04, p = .976$ (declined 13% on average). Figure 4.3 depicts the association between pre-intervention and post-intervention PM accuracy by intervention condition. It highlights that participants with the poorest baseline PM (-1 SD from the mean) exhibit the greatest improvement when comparing implementation intention to no intervention conditions on post-intervention PM accuracy. In contrast, pre-intervention PM accuracy was not a significant moderator of the effect of GMT on post-intervention PM accuracy, relative to the no intervention or implementation...
intention conditions.
Table 4.3.

*Moderated regression model predicting post-intervention prospective memory accuracy with intervention condition, moderated by pre-intervention prospective memory accuracy.*

<table>
<thead>
<tr>
<th>Model number</th>
<th>Predictor</th>
<th>Overall model $R^2$</th>
<th>Interaction effect $R^2$ Change (Sig.)</th>
<th>Unstandardised coefficient (bootstrapped CIs 5000 samples)</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. $N = 102$</td>
<td>Intervention condition (II = 1, No intervention = 0)</td>
<td>.19</td>
<td>0.33 (.06 – 0.75)</td>
<td>.015</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td>.04 (.040)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td>-0.33 (-0.81 – -0.02)</td>
<td>.040</td>
</tr>
<tr>
<td>8. $N = 108$</td>
<td>Intervention condition (GMT = 1, No intervention = 0)</td>
<td>.18</td>
<td>0.16 (-0.24 – 0.69)</td>
<td>.256</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td>.01 (.258)</td>
<td></td>
<td>0.48 (0.28 – 0.90)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td>-0.19 (-0.80 – 0.27)</td>
<td>.258</td>
</tr>
<tr>
<td>9. $N = 105$</td>
<td>Intervention condition (GMT = 1, II = 0)</td>
<td>.11</td>
<td>-0.16 (-0.57 – 0.24)</td>
<td>.247</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td>.01 (.441)</td>
<td></td>
<td>0.17 (-0.06 – 0.41)</td>
<td>.195</td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td>0.13 (-0.34 – 0.60)</td>
<td>.441</td>
</tr>
</tbody>
</table>
### ii. Sensitivity analysis

<table>
<thead>
<tr>
<th>Model number</th>
<th>Predictor</th>
<th>Overall model R²</th>
<th>Interaction effect R² Change (Sig.)</th>
<th>Unstandardised coefficient (bootstrapped CIs 5000 samples)</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>10. N = 97</td>
<td>Intervention condition (II = 1, No intervention = 0)</td>
<td>.22</td>
<td>.03 (.048)</td>
<td><strong>0.33</strong> (0.06 – 0.59)</td>
<td>.017</td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td></td>
<td><strong>0.52</strong> (0.29 – 0.74)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td><strong>-0.32</strong> (-0.76 – -0.003)</td>
<td>.048</td>
</tr>
<tr>
<td>11. N = 93</td>
<td>Intervention condition (GMT = 1, No intervention = 0)</td>
<td>.19</td>
<td>.07 (.005)</td>
<td><strong>0.42</strong> (0.14 – 0.70)</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td></td>
<td><strong>0.52</strong> (0.29 – 0.75)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td><strong>-0.48</strong> (-0.81 – -0.15)</td>
<td>.005</td>
</tr>
<tr>
<td>12. N = 90</td>
<td>Intervention condition (GMT = 1, II = 0)</td>
<td>.04</td>
<td>.01 (.358)</td>
<td>0.10 (-0.19 – 0.38)</td>
<td>.497</td>
</tr>
<tr>
<td></td>
<td>Pre-intervention PM accuracy</td>
<td></td>
<td></td>
<td>0.20 (-0.04 – 0.43)</td>
<td>.099</td>
</tr>
<tr>
<td></td>
<td>Interaction effect</td>
<td></td>
<td></td>
<td>-0.16 (-0.51 – 0.19)</td>
<td>.358</td>
</tr>
</tbody>
</table>

*Note:* CIs: confidence intervals, if CIs do not cross zero the effect is significant at p < .05. Significant effects in bold. Primary analyses include all participants; Sensitivity analyses exclude participants who experienced confusion during intervention training or post-intervention PM task. PM: prospective memory. II: implementation intentions. GMT: goal management training. Interaction effect is Intervention condition × Pre-intervention PM accuracy.
Figure 4.3.

Association between pre-intervention and post-intervention prospective memory accuracy by intervention condition.

Sensitivity analysis

After noting higher counts of confusion in the GMT condition (Table 4.1, coding described in Methods), we also considered whether there were significant differences between the proportion of participants expressing confusion (secondary outcome) in each intervention condition, and whether analyses should be repeated after excluding participants who demonstrated confusion. In the GMT condition, 21.8% of participants experienced confusion, significantly more (ratio > 1.96) than in the implementation intentions (4.1%) or no intervention conditions (5.7%). Thus, we conducted sensitivity analyses comparing the effect of GMT, implementation intentions and no intervention on post-intervention PM accuracy after excluding participants who were confused during the intervention delivery or post-intervention PM task. These

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5 GMT vs. no intervention: actual difference = 16.10, SE difference = 6.66, ratio = 2.42. GMT vs. II: actual difference = 17.70, SE difference = 6.70, ratio = 2.64. II vs no intervention: actual difference = -1.60, SE difference = 4.29, ratio = -0.37.
models are presented in Table 4.2-ii.

After excluding confused participants, receiving implementation intentions was associated with better post-intervention PM, in comparison to no intervention, $F(2, 94) = 11.06, p < .001$. Conversely, while the overall model was significant ($F(2, 90) = 5.61, p = .005$), GMT was not associated with better PM in comparison to no intervention (see Table 4.2-ii). When GMT was compared to implementation intentions among those who were not confused, there were no differences in outcome, $F(2, 87) = 1.48, p = .234$.

As for the primary analyses, we also repeated the moderation analyses to determine if those with poorer pre-intervention baseline might find either one or both interventions differentially effective.

Linear moderation analyses conducted without participants who experienced confusion in the task are presented in Table 4.3-ii. The moderating effect of pre-intervention PM accuracy on the effect of implementation intentions on post-intervention PM accuracy remained significant in the sensitivity analysis, $F(3, 93) = 8.95, p < .001$. Additionally, there was a significant moderating effect of pre-intervention PM accuracy on the effect of GMT, $F(3, 89) = 6.76, p < .001$. GMT was associated with better PM accuracy compared to no intervention among those participants with relatively poor baseline PM (-1 SD from the mean; $b = 0.12$, SE $= 0.04$, $p = .008$), who improved on average by 18%. GMT was not associated with better PM accuracy compared to no intervention for those participants whose baseline PM was average ($b = 0.03$, SE $= 0.03$, $p = .328$, declined 1% on average) or better (+1 SD from the mean, $b = -0.06$, SE $= 0.04$, $p = .198$, declined 18% on average). As in the simple model, the overall linear moderation model comparing GMT to implementation intentions was not significant, $F(3, 86) = 1.27, p = .290$. 

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Discussion

This study aimed to evaluate the efficacy of GMT as an intervention for PM in older adults. Regarding the primary outcome, an established intervention (implementation intentions) improved PM marginally in the sample as a whole, and significantly amongst older adults with poorer baseline PM. GMT unexpectedly did not improve PM performance in older adults in comparison to no intervention (Hypothesis 2) or implementation intentions (Hypothesis 3). Regarding the secondary outcome, a significantly higher proportion of participants who received GMT experienced confusion during intervention delivery or the post-intervention PM task, prompting us to conduct additional sensitivity analyses, discussed below.

Whilst our finding that GMT did not improve PM is unexpected, we believe our delivery of GMT was consistent with what previous research suggests is appropriate. We included an explicit training stage and content-free cues, which have been shown to increase efficacy (Fish et al., 2007; Krasny-Pacini et al., 2014). We required participants to pass a quiz testing their knowledge of GMT before they completed the post-intervention measures. Finally, we gave our participants the opportunity to practice GMT within the PM task environment before starting the post-intervention assessment.

Our prediction that GMT would improve PM was based on a number of previous research findings. First, executive changes have been identified as a significant source of PM deficits in older adults (Schnitzspahn et al., 2013) and thus an intervention targeting executive function would be expected to ameliorate PM deficits. Second, previous research supports GMT as an effective intervention for executive function, although we note positive outcomes with older adults specifically are primarily based on subjective report (Levine et al., 2007; van Hooren et al., 2007). Finally, GMT has shown promise as an effective intervention specifically for PM (Fish et al., 2007) in other populations. Our unexpected finding that GMT did not improve PM might be
taken as cautionary as to whether compensating for executive dysfunction is an appropriate strategy to improve PM performance in older adults. Alternatively, it may be that the delivery of GMT needs to be altered when used specifically for PM in older adults.

It is possible that GMT was not associated with improved PM accuracy in our study because the ‘dose’ of GMT was inadequate for these participants. Whilst we aimed to provide a thorough and explicit outline of GMT, explained how to use it in the task, and successfully piloted the intervention with young adults (Li, 2014), we may still have fallen short on the amount of training required for older adults to successfully comprehend and use GMT. Indeed, post-hoc analyses suggested participants in the GMT condition were more likely to experience confusion during intervention delivery or the post-intervention PM task, suggesting GMT hindered rather than helped some of this group. Our participants were comparable to normative groups on overall cognitive function, retrospective memory and executive function, suggesting our results are not due to recruiting an atypical sample of older adults whose confusion was due to cognitive decline. When analyses were limited to those who comprehended the interventions, there was some evidence for GMT’s efficacy, in comparison to no intervention. GMT was associated with improved PM specifically amongst participants with relatively poor baseline PM ability, if they understood the instructions (whilst regression to the mean may produce a similar pattern, arguments against regression to the mean are presented below). Thus, whilst GMT can potentially improve PM in some older adults, when delivered in a single brief session, older adults in general may struggle effectively to use the strategy to improve their PM accuracy, and indeed may be confused by the training. Perhaps, extending treatment time over multiple sessions or incorporating errorless learning principles when teaching GMT to older adults (as implemented for individuals with brain injury (Bertens, Kessels, Fiorenzato, Boelen, &
Fasotti, 2015) can increase GMT efficacy. Indeed, a recent meta-analysis evaluating the effectiveness of multi-session GMT reported a relationship between improvement in executive function and number of GMT treatment hours (Stamenova & Levine, 2018). Whilst our research is a first step towards determining the appropriate ‘dose’ of GMT to improve PM, future studies are required to tease apart the contributions of training length and additional intervention elements in determining the most beneficial delivery of GMT to improve older adults’ PM.

Our results also demonstrate that baseline PM ability affects response to compensatory intervention. In the implementation intentions condition, response to intervention was greatest for older adults with poorer baseline PM ability. Indeed, these participants improved by 18% following intervention, compared to the average improvement of 3%. With regards to previous findings, no studies have yet evaluated whether baseline PM predicts response to implementation intentions. Some researchers have considered baseline ability in general cognition or domains other than PM with some (Brom & Kliegel, 2014; Brom et al., 2014; Insel, Einstein, Morrow, Koerner, & Hepworth, 2016), but not all (Burkard, Rochat, Juillerat Van der Linden, Gold, & Van der Linden, 2014; McFarland & Glisky, 2011; Schnitzspahn & Kliegel, 2009; Shelton et al., 2016), reporting a greater benefit to participants of lower ability, consistent with our results. Evaluating baseline PM directly, rather than related cognitive abilities, is particularly important because PM requires the dynamic integration of multiple domains and is, thus, more than the sum of its constituent parts.

In the sensitivity analysis excluding participants who experienced confusion, baseline PM ability also moderated the effect of GMT on PM accuracy in comparison to no intervention, again driven by those with below average baseline PM. This finding may help explain why GMT was not effective, overall, at improving PM. That is, whilst compensatory interventions may be most useful for older adults with poorer PM, the
same poorer ability might affect this group’s ability to learn and apply a multi-step strategy like GMT in a single session. In contrast, implementation intentions – which has only two simple steps – is likely to be more easily learnt in a single session, and could thus be more likely to result in improved PM for those with poorer baseline cognitive ability.

A potential criticism of these results could be that statistical phenomena rather than clinically important baseline characteristics might account for the moderating role of baseline PM. However, we believe these explanations do not best account for the pattern of results. Firstly, it could be argued that ceiling effects in our measure may have suppressed potential improvement following intervention in participants with higher baseline PM. However, the lack of improvement in those with average baseline PM, and the tendency for those with highest baseline PM to show poorer post-intervention PM, are not consistent with this account. However, this pattern of improvement in those with lower baseline PM, and decline of those with higher baseline PM is consistent with regression to the mean. While regression to the mean may explain the trends seen in the no intervention condition, the degree of improvement shown by participants with poorer baseline ability was significantly greater for those who received intervention (Figure 4.3), giving us confidence that this increased accuracy reflects a true benefit of the interventions. Put another way, if regression to the mean were the main explanatory factor, then all three groups would show the same pattern, regardless of intervention, which was not observed.

The lack of significant overall effect for GMT in the present study is also notable given the literature supporting the efficacy of GMT when combined with augmented interventions such as content-free cueing. It could be argued that certain factors in our study reduced the potential potency of GMT. Perhaps stronger effects of GMT would be apparent for a harder PM task (e.g. time-based or more naturalistic) or
for participants who did not have relatively high executive function, indexed in our study by TMT A-B difference scores. However, the integration of findings from each of our analyses is important here. Whilst effects were indeed apparent only for participants with poorer baseline PM, this was true only for those displaying good comprehension of the GMT intervention. We found that a substantial minority (21.8%) showed confusion in learning the GMT steps, even in our well-educated, cognitively healthy sample. Thus, while future research should seek to increase the potential improvement that participants can show by using a harder task or participants of lower ability, this will likely need to be balanced with an increased dose of GMT. Our results also highlight that future research should continuously assess participants’ comprehension of GMT throughout the task, and intervene when confusion is apparent.

In conclusion, this study supports the efficacy of implementation intentions in improving PM performance, providing evidence that response to intervention is greatest in those with poorer baseline PM. However, GMT – as implemented in this study – was overall not successful at improving PM in older adults. Our results highlight the need, when designing interventions, to strike a balance between adhering to solid theoretical considerations of underlying deficits (e.g. GMT to target the executive dysfunction that underlies PM deficits), and providing simple, user-friendly strategies that can be quickly and effectively delivered to people with compromised cognition.
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CHAPTER 5

General Discussion
Thesis rationale and overall aims

The existence of prospective memory (PM) deficits amongst older adults is well-established (Kliegel et al., 2016). However, research evaluating ways to improve older adults’ PM is still in its infancy (Hering, Rendell, Rose, Schnitzspahn, & Kliegel, 2014). This thesis aimed to explore two new approaches for improving PM in older adults. First, the role of sleep disruption in older adults’ PM was examined (Chapter 2), with the goal of evaluating whether improving sleep could improve PM (Chapter 3). These studies are among the first in the literature to consider the relationship between habitual sleep and PM performance in older adults, based on known links between sleep and cognition in older adults (Miyata, Noda, Iwamoto, & Kawano, 2013; Song et al., 2015; Spira et al., 2017), and between sleep and PM in younger adults (Leong, Cheng, Chee, & Lo, 2019). Second, this thesis evaluated for the first time an executive function intervention for PM in older adults: goal management training. Whilst executive dysfunction is often emphasised as a driver of age-related PM decline (Azzopardi, Juhel, & Auffray, 2015; Kliegel, Martin, McDaniel, & Einstein, 2002; Mattli, Schnitzspahn, Studerus-Germann, Brehmer, & Zöllig, 2014), the majority of existing PM interventions for older adults have focused on strategies for the retrospective component of PM.

The results of the three empirical studies included in this thesis are summarised below. Findings are then integrated to present the overall contributions made by this thesis to improving PM in older adults. Limitations and future directions are summarised.

Overview of study findings

Chapter 2 of this thesis assessed the relationship between sleep disruption and poorer PM in older adults. Community-dwelling older adults underwent assessment of habitual sleep using actigraphy, and then completed a laboratory event-based PM task. It was hypothesised that less sleep (decreased total sleep time, sleep efficiency) and
more wakefulness (increased wake after sleep onset, awakening length, awakening number) would be associated with poorer PM.

As expected, sleep disruption (in the form of longer awakenings) was indeed associated with poorer PM. Furthermore, longer awakenings fully mediated the relationship between older age and poorer PM, suggesting the increasing sleep disruption experienced by older adults contributes to age-related PM deficits. The mediating effect of awakening length was partially a function of poorer executive function, but not retrospective memory. This finding is consistent with research highlighting the vulnerability of executive functions to sleep disruption (Holanda Júnior & de Almondes, 2016), and implies potential flow-on effects for PM. The particular significance of awakening length (in contrast to other metrics of sleep disruption, which were not related to PM) follows other research emphasizing that it is the fragmentation of sleep that is particularly detrimental to cognition in older adults (Blackwell et al., 2014).

Taking cues from the literature on executive function and sleep, we can speculate on the neural underpinnings of the relationship between sleep disruption and poorer PM. For example, Anderson and Horne (2003) and Lafortune and colleagues (2014) report that older adults’ performance on executive tasks is related to markers of the restorative action of sleep. A role for sleep dependent neuro-restoration may also be indicated by the particular significance of awakening length in Chapter 2. We have suggested that increased awakening length may index significant disruption to restorative slow-wave and REM sleep, due to repeated long awakenings interfering with the cyclical nature of sleep. Interruption to slow-wave and REM sleep and hence to proposed neuro-restoration could then impact the micro and macro neural structures that support PM. Indeed, poor sleep in older adults has been linked to reduced white matter integrity (Sexton et al., 2017), cortical thinning (Lim et al., 2016) and reduced glial and
neuronal integrity (Cross et al., 2013). Future research should aim to include measures of sleep physiology (e.g. PSG) and neural health (e.g. imaging) to test these hypotheses about links between sleep stages, sleep dependent neuro-restoration, neural health and PM.

Building on the findings of Chapter 2, Chapter 3 presented a pilot study examining whether an intervention to improve older adults’ sleep (cognitive behavioural therapy for insomnia: CBT-I) could also improve PM. Older adults with poor sleep were allocated to either a CBT-I group or waitlist control. Pre and post-intervention, participants underwent assessment of PM with a comprehensive laboratory-based task, and sleep with actigraphy and questionnaires. It was hypothesised that CBT-I would be associated with better post-intervention PM, via the beneficial impact of CBT-I on reduced average awakening length and increased sleep efficiency.

Results suggested an improvement in self-reported insomnia symptoms following CBT-I. However, there was no improvement in objective sleep or PM. This suggests subjective improvement in sleep does not necessarily equate to objective improvement in sleep, and is not sufficient to improve PM. Some previous studies have also failed to find consistent effects of CBT-I on measures of objective sleep, especially in older adults, despite self-reported sleep typically improving (Irwin et al., 2014; Lovato, Lack, Wright, & Kennaway, 2014). If older adults’ sleep disruption is more resistant to psychological treatment, this could imply a more stable, neurological basis for poor sleep. However, the non-significant findings in regards to PM do not discount the possibility that improving sleep is still a viable pathway to improve PM, especially considering the small sample size.

Moving to an alternative avenue to intervention, Chapter 4 evaluated a compensatory executive function strategy to improve PM in older adults: goal management training. Older adults were allocated to receive either goal management
training, an established intervention targeting the retrospective component of PM (implementation intentions) or no intervention. Event-based PM was assessed before and after the intervention. It was hypothesised that both implementation intentions and goal management training would result in better post-intervention PM, in comparison to no intervention. Additionally, goal management training was expected to have an advantage over implementation intentions in improving PM. This was based on research suggesting executive dysfunction (supported by goal management training; Stamenova & Levine, 2018) is a greater contributor to age-related PM decline than retrospective memory (supported by implementation intentions; Azzopardi et al., 2015; Kliegel et al., 2002; Mattli et al., 2014; McFarland & Glisky, 2011).

This study found implementation intentions, relative to no intervention, improved PM for participants with poorer baseline PM. However, in the full sample, goal management training was not associated with improved PM, relative to no intervention or implementation intentions. In fact, in post-hoc analyses, participants who received goal management training were more likely to have difficulty comprehending the intervention. Follow-up analyses including only those participants who comprehended the intervention found that, in this group, goal management training was associated with better PM compared to no intervention.

However, similar to implementation intentions, this was only for participants with poorer baseline PM. Overall, these results suggest baseline PM ability affects response to compensatory intervention. Whilst goal management training can potentially improve PM, older adults may have difficulty effectively using this strategy delivered in a single session to improve PM. Thus, whilst compensatory interventions may be most useful for older adults with poorer PM, potential underlying cognitive deficits may interfere with this group’s ability to learn a complex, multi-step strategy like goal management training without significant effort. Implementation intentions may
be pitched more appropriately to be understood in a single session and thus result in improved PM for those who need the most support.

**Integration of findings**

Overall, the findings of the studies included in this thesis suggest possible opportunities to improve older adults’ PM through sleep and compensatory executive function strategies. Importantly, results suggest intervention is likely to be specifically effective for older adults with poorer PM.

This thesis demonstrates that sleep disruption and PM ability are linked amongst older adults, and provides some important insights about this relationship. First, there was full statistical mediation of the relationship between older age and poorer PM by sleep disruption. Similar findings were recently reported in another study using different sleep measures (Scullin et al., 2019). However, attempts to provide further evidence for a causal relationship by manipulating sleep with CBT-I in Chapter 3 weren’t successful. These latter results do not discount the possibility raised in Chapter 2 that improving sleep can improve PM, especially considering this study’s small sample size. However, Chapter 3 also raised the possibility that this approach to improving PM might be hampered by difficulty improving older adults’ sleep, as explained below.

A second insight into the relationship between sleep and PM comes from an integration of the results of Chapter 3 with previous research. The link between sleep and cognition in older adults has potential neural underpinnings, as suggested by associations between older adults’ poor sleep and reduced white matter integrity (Sexton et al., 2017), cortical thinning (Lim et al., 2016) and reduced glial and neuronal integrity (Cross et al., 2013). These findings highlight the importance of establishing whether neural changes are a consequence of poor sleep, or if neural changes lead to poor sleep, with potential future research directions discussed below. The former calls for urgency in delivering intervention to improve sleep. The latter suggests intervention
to improve sleep might be less effective for older adults. Indeed, this was a finding in Chapter 3. If older adults’ sleep disruption is more resistant to treatment, there may be a more stable, neurological basis for poor sleep in some older adults, e.g. atrophy in brain regions presumed responsible for maintaining sleep (Lim et al., 2016; Mander et al., 2013). This possibility has further implications for the nature of the relationship between sleep and cognition. Neurological brain changes that affect sleep might also affect networks supporting cognition (Mander, 2013; Yaffe, Falvey, & Hoang, 2014). Cognition could also be affected secondary to interruption of the restorative action of sleep (Anderson & Horne, 2003; Lafortune et al., 2014; Mendelsohn & Larrick, 2013). Both processes could also occur at once. Thus, whilst the findings of Chapter 2 suggest sleep as a potential avenue to improve PM, Chapter 3 makes clear that further research is required on the relationships between neural changes, sleep disruption and PM, discussed below. If future research suggests a stable, neurological basis for poor sleep in some older adults, discounting the possibility of ameliorating PM deficits by improving sleep, there are still avenues to intervention through compensatory strategies, as explored in Chapter 4.

The findings presented in Chapter 4 of this thesis provides further support for implementation intentions as a compensatory strategy for PM in older adults. Additionally, there was some evidence for the effectiveness of goal management training to improve PM in older adults. However, results also suggest goal management training delivered in a single session may be difficult for older adults to comprehend and use. Overall, delivering compensatory PM interventions requires striking a balance between adhering to solid theoretical considerations of underlying deficits (e.g. goal management training to target the executive dysfunction that underlies PM deficits), and providing simple, user-friendly strategies that can be quickly and effectively delivered.
to people with compromised cognition. Future directions for executive function strategies to improve PM are discussed below.

**Limitations**

A potential limitation of this thesis was the use of different measures of PM across studies. Chapter 2 used a computerised event based PM task with non-focal cues. Chapter 3 used a laboratory based measure designed to mimic real-world PM tasks using both time and event based PM cues. Chapter 4 again used a computerised event based PM task, but with categorical cues. The use of non-focal cues (Chapter 2) is more clearly linked to high executive demands than the use of categorical cues (Chapter 4) or a variety of time and event-based cues (Chapter 3) (McDaniel & Einstein, 2000). The use of tasks with greater executive demands is important because of their sensitivity to age-related PM decline (Kliegel et al., 2016). We also found, in Chapter 2, that poorer executive functioning partially accounted for the relationship between disturbed sleep and poorer PM, underscoring the importance of using PM tasks with high executive demands when evaluating links between age, sleep and PM. Differences in the PM tasks used thus might partially account for the disparity between results of Chapters 2 and 3, with a link between sleep and PM demonstrated in the former (which used a more executive dependent task) but not the latter. The use of a PM measure that was not strictly non-focal might also have limited our ability to tap into the functioning of the executive processes we sought to support with goal management training in Chapter 4. Overall, whilst we explored PM in many forms by using a variety of PM tasks, future research examining PM, ageing and sleep (which crossover in their links with executive processes) should ensure to use PM tasks that are executive dependent, e.g. non-focal or time based.

Another potential limitation of this thesis is the lack of real-world measures of PM. Previous research has made clear that poor performance on laboratory PM tasks
does not necessarily equate to poor performance on real-world PM tasks, especially for older adults (Henry, MacLeod, Phillips, & Crawford, 2004). However, this may be due to other factors that influence real-world PM task performance, beyond objective PM ability. For example, the nature of ongoing activities and routines, use of compensatory strategies, and motivation, all play a role in determining real-world PM performance (Cavuoto, Ong, Pike, Nicholas, & Kinsella, 2017; Schnitzspahn, Ihle, Henry, Rendell, & Kliegel, 2011). Laboratory-based PM tasks, in contrast, attempt to remove or control these factors. Furthermore, performance on laboratory-based PM tasks is related to measures of real-world functioning, such as activities of daily living, quality of life, and medication adherence (Woods, Weinborn, Velnoweth, Rooney, & Bucks, 2012; Zogg, Woods, Sauceda, Wiebe, & Simoni, 2012).

Another potential limitation, although one by no means limited to this thesis alone, is the use of a sample recruited through a university. By virtue of their links with the university and their motivation to be engaged in research, this group might be expected to have more education and be functioning at a higher level than typical older adults. This may have implications for the generalizability of these results. However, many of the findings emerged specifically amongst participants who showed less of the advantages in health and cognition that this context might be associated with, e.g. participants with poorer PM or more sleep disturbance. Thus, it is possible our findings would be stronger in a more typical community sample.

**Future research**

The studies presented in this thesis inform future research in several ways. Chapters 2 and 3 highlight that more research is needed exploring the links between sleep disruption, ageing, and PM. While the findings presented in this thesis suggest an association between sleep disruption and age-related PM decline, the nature of this association is not clear. Thus, future research should evaluate whether this relationship
is causal by using longitudinal designs. By observing changes in sleep and PM performance over time, it may become clear whether increasing sleep disruption leads to reduction in PM performance. It would also be ideal if future research included measures of brain health, using neuroimaging or biomarkers, and sleep physiology, e.g. with PSG. This would help clarify the role of neurological changes in the relationship between sleep disruption and cognition, including PM. Specifically, future research should test whether long-term sleep disruption leads to declining brain health through interruption of sleep-dependent restorative processes, with subsequent effects on cognition. An alternative hypothesis for future research to consider is that age-related neurological changes leads to both increased sleep disruption (by disrupting networks that help maintain sleep) and cognitive decline. By investigating these two possibilities, future research could make clear whether it is indeed a worthwhile venture to use psychological sleep interventions, such as CBT-I, to improve cognitive function, such as PM performance.

Another direction for future research is to consider other interventions to improve PM by improving sleep. Whilst CBT-I is the gold-standard treatment for insomnia, sleep could also be improved by exercise programs or continuous positive airway pressure for OSA (CPAP). The potential flow-on effects of these treatments for PM performance (and possibly other cognitive functions) is worth investigating.

Regarding compensatory executive interventions, it will be important for future research to build on the potential benefits of goal management training explored in this thesis. In particular, future research should evaluate whether goal management training shows greater effectiveness for improving PM in older adults when delivered over multiple training sessions, or with other strategies such as errorless learning. Future research might also consider other executive function strategies to improve PM in older adults. For example, the Cognitive Orientation to Occupational Performance program
has been piloted with older adults to improve goal directed behaviour, and could also be adapted for PM (Dawson et al., 2014).

In terms of methodological considerations, particular strategies around recruitment and intervention delivery might be recommended. Firstly, specifically recruiting participants with poorer PM might allow clearer findings to emerge about the benefits of PM intervention. Secondly, when delivering interventions, continuously assessing participants’ comprehension of training, and acting promptly when confusion is apparent could help ensure participants correctly apply intervention strategies. Thirdly, for evaluation of sleep interventions specifically, using objective sleep parameters when recruiting participants could help clarify exactly how the sleep of older adults with objectively poor sleep changes with intervention.

Conclusion

Overall, this thesis provides further support for implementation intentions as a compensatory strategy for older adults with poor PM. Goal management training is suggested as an additional helpful compensation strategy, but researchers and clinicians will need to be mindful of the complexity of the strategy and thus the amount of training older adults require. Improving sleep was also explored as an avenue to improve PM. However, more research is needed as to how neural changes relate to sleep changes and PM ability, and affect older adults’ potential to benefit from sleep intervention. If poor sleep in some older adults is a consequence of neural changes, and thus not amenable to psychological intervention, then compensatory interventions for PM deficits might be a more fruitful path to improve PM.
References


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https://doi.org/10.1007/s10865-011-9341-9
Supplementary materials

General methods

Participants included in the research presented in this thesis were drawn from the Healthy Ageing Research Program (HARP) at the University of Western Australia, and the Western Australia Memory Study (WAMS) conducted at the Australian Alzheimer’s Research Foundation. Both these research programs are ongoing studies of older adults that collect a range of measures, as detailed below in Table S.1. As a supplement to the information provided in the methods’ sections within the body of the thesis, further information on recruitment, assessment procedures and instruments are provided here.

Recruitment

Methods for recruitment used in HARP and WAMS are ongoing advertising in the community, public information sessions on healthy ageing and dementia, and through connections with community groups that are likely to have appropriate people for the research in their membership base.

Assessment of Mild Cognitive Impairment and Dementia

As outlined in the body of the thesis, the McKhann et al. (2011) criteria are used in HARP and WAMS to define MCI and dementia. To paraphrase from these criteria, a diagnosis of dementia is made in the presence of cognitive or behavioural symptoms that, a) interfere with function, b) represent a decline from previous for that individual, and c) are not explained by delirium or major psychiatric disorder. The cognitive or behavioural symptoms should be evidenced through a combination of history-taking from the person and a knowledgeable informant, and objective cognitive assessment. The cognitive or behavioural symptoms should involve at least two of the following domains: memory, reasoning, visuospatial function, language, and personality/behaviour/social functioning. A diagnosis of MCI is made based on the same criteria,
but where the level of interference with functioning is not considered significant, based on clinical judgement.

In HARP and WAMS, these data are collected as part of normal research proceedings. The criteria are applied by consensus in regular clinical meeting where participant files are reviewed.

**Assessment of Sleep: Actigraphy**

Chapters 2 and 3 of this thesis involved the use of actigraphs to measure sleep. Actigraphs were worn full-time by the participants (except for activities involving water). Naps were not included in the analysis of sleep, since the focus was on nighttime sleep, or the main sleep period. We aimed to have participants wear the actigraphs for 7 days, thus including one weekend in the measure of habitual sleep. All but seven participants adhered to this, with these seven wearing the actigraph for between 6 and 11 days. Our analysis of actigraphy data utilised 30-second epochs and a sample rate of 30 – 100 hertz. When tested against polysomnography, actigraphy is reported to have a sensitivity for detecting sleep of .97 and accuracy of .86, whereas specificity for detecting wake was .33 (Marino et al., 2013). The implications of this lower sensitivity is discussed within the chapters.
References


Table S.1

**Measures used in HARP and WAMS.**

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<tr>
<th>Test</th>
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<tr>
<td>National Adult Reading Test (NART)</td>
<td>Premorbid IQ</td>
<td>Cronbach’s α = .93.</td>
<td>No significant difference in NART scores between controls and patients with cerebral atrophy (atrophy group - mean error score = 23.9, s.d. = 11.2; control group - mean error score = 22.4, s.d. = 10.1; t = 0.6; p &gt; 0.05). N Nelson, H. E., &amp; Willison, J. (1991). National adult reading test (NART). Windsor: Nfer-Nelson.</td>
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<td>METAPROM (novel)</td>
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<td>Klaren, R., &amp; Motl, R. W.</td>
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<tr>
<td></td>
<td></td>
<td>(2013). Psychometric</td>
<td></td>
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<td></td>
<td></td>
<td>properties of the fatigue</td>
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<td></td>
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<td>severity scale and the modified</td>
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<td>fatigue impact scale. Journal</td>
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<td>of the neurological sciences, 331(1-2), 102-107.</td>
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<td></td>
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<td></td>
<td>clinically diagnosed OSA with a sensitivity of 77%, a specificity of 39%, a positive predictive value of 63% and a negative predictive value of 55%. Sforza, E., Chouchou, F., Pichot, V., Herrmann, F., Barthélémy, J. C., &amp; Roche, F. (2011). Is the Berlin questionnaire a useful tool to diagnose obstructive sleep apnea in the elderly? Sleep medicine, 12(2), 142-146.</td>
<td>WAMS</td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Domain assessed</td>
<td>Reliability *</td>
<td>Validity *</td>
<td>Reference</td>
<td>Study</td>
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<td>----------------------------------------------</td>
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<tr>
<td>Test</td>
<td>Domain assessed</td>
<td>Reliability *</td>
<td>Validity *</td>
<td>Reference</td>
<td>Study</td>
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<td>-----------------------------------------------------------------------------------------------------</td>
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</tr>
<tr>
<td>Patient Health Questionnaire 9 (PHQ-9)</td>
<td>Depression</td>
<td>Cronbach’s $\alpha$ = 0.83 – .92.</td>
<td>Demonstrates an area under the curve</td>
<td>Cameron, I. M., Crawford, J. R., Lawton, K., &amp; Reid, I. C. (2008). Psychometric comparison of PHQ-9 and HADS for measuring depression severity in primary care. <em>Br J Gen Prim Care</em>, 58(546), 32-36.</td>
<td>HARP</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder Screener (GAD-7)</td>
<td>Anxiety</td>
<td>Cronbach’s $\alpha$ = .92.</td>
<td>Correlations with the Beck Anxiety Inventory ($r = .72$) and the anxiety subscale of the Symptom Checklist-90 ($r = .74$).</td>
<td>Spitzer, R. L., Kroenke, K., Williams, J. B., &amp; Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: the GAD-7. <em>Archives of internal medicine</em>, 166(10), 1092-1097.</td>
<td>HARP</td>
</tr>
</tbody>
</table>
### Activities of Daily Living Questionnaire (ADLQ)

**Self-report and informant-report daily functioning**

**Test-retest concordance coefficients of .86 or higher.**

**Significant correlation with MMSE ($r = -.42$) and clinical dementia rating scale ($r = .50$).**


### Medication use and adherence questionnaire (novel)

**Self-report and informant report of medication use and adherence.**

**n/a**

**n/a**

**n/a**


### Physical Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>HARP</th>
<th>WAMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Neck circumference</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Near vision</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Zeo Sleep stages</td>
<td>Inter-rater reliability of 81.1%. Agreement with polysomnography for sleep staging 75.8 - 74.7%.</td>
<td>HARP</td>
</tr>
</tbody>
</table>

185
<table>
<thead>
<tr>
<th>Test</th>
<th>Domain assessed</th>
<th>Reliability *</th>
<th>Validity *</th>
<th>Reference</th>
<th>Study</th>
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</thead>
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<tr>
<td>Test</td>
<td>Domain assessed</td>
<td>Reliability *</td>
<td>Validity *</td>
<td>Reference</td>
<td>Study</td>
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</tbody>
</table>

Notes: *all reported effects are significant at \( p < 0.05 \) unless indicated. Reported effects come from original article as listed in reference column, unless indicated. AD: Alzheimer’s disease; TBI: traumatic brain injury; HARP: Healthy Ageing Research Program; WAMS: Western Australian Memory Study.
Supplementary materials for Chapter 2.

Table S.2.5.

Mediation models for predicted relationships between age, sleep disruption, and prospective memory in older adults: sleep efficiency and WASO.

<table>
<thead>
<tr>
<th>Model</th>
<th>Predictor</th>
<th>Mediator</th>
<th>Outcome</th>
<th>Covariate</th>
<th>a</th>
<th>b</th>
<th>e</th>
<th>c</th>
<th>c'</th>
</tr>
</thead>
<tbody>
<tr>
<td>i.</td>
<td>Age</td>
<td>Sleep</td>
<td>PM</td>
<td>Education</td>
<td>0.07‡</td>
<td>-5.88‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Efficiency</td>
<td>Accuracy</td>
<td></td>
<td>0.09</td>
<td>-0.87</td>
<td>1.88‡</td>
<td>-0.62†</td>
<td>-0.23</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TST</td>
<td></td>
<td></td>
<td>0.80</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii.</td>
<td>Age</td>
<td>WASO</td>
<td>PM</td>
<td>Education</td>
<td>-0.31</td>
<td>0.18</td>
<td>1.58‡</td>
<td>-0.62†</td>
<td>-0.19</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Accuracy</td>
<td></td>
<td>0.80</td>
<td>-0.004</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. †: *p* < 0.05; ‡: *p* < 0.01; a: path between predictor and mediator; b: path between mediator and outcome variable, controlling for predictor; d: path between mediators; c: total effect between predictor and outcome variable; c': direct effect between predictor and outcome variable, after taking account of indirect effect(s); e: path between covariate and outcome variable/mediator. PM: prospective memory; TST: Total Sleep Time; WASO: Wake after sleep onset.
Supplementary materials for Chapter 3.

Generating WAProm residual scores from a study-based normative sample of community dwelling older adults.

In order to control for the potential effects of demographic factors in the current study, WAProm scores in a separate dataset were analysed to account for the effects of age, gender and education. The regression coefficients resulting from this analysis were then used to calculate WAProm residual scores in the current study. Here, we describes the participants, methods and results of the analysis conducted with this study-based normative sample.

Methods

Two-hundred and forty-three community-dwelling healthy older adults (aged over 50 years) participating for the first time in the Western Australian Memory Study were assessed for inclusion in the normative sample. WAMS is a longitudinal study of neuropsychological and biological markers of Alzheimer’s disease, led by Dr Hamid Sohrabi, and conducted through Edith Cowan University and the Australian Alzheimer Research Foundation. Participants complete the WAProm (described in the main paper) as part of the broader neuropsychological assessment. Exclusion criteria for the normative sample were: serious neurological conditions (e.g. stroke, epilepsy, traumatic brain injury with loss of consciousness > 30 mins, brain tumour), untreated severe psychiatric conditions, score below age and education defined cut-offs (1.5 standard deviations below means reported in Rossetti, Lacritz, Munro Cullum, & Weiner, 2011) on the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005), or missing data required for calculation of residual scores. Seventeen participants were excluded due to medical conditions, and two were excluded due to MoCA score. Twelve participants were excluded
due to missing data for the time-based PM regression, and 13 for the event-based PM regression, leaving a total sample of 212 participants for time-based PM and 211 for event-based PM.

**Results**

Descriptive statistics for the sample are shown in Table S.3.7.

Table S.3.7.

*Descriptive statistics for demographic variables, MoCA scores and WAProm scores.*

<table>
<thead>
<tr>
<th></th>
<th>Range</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>50 – 89</td>
<td>69.45</td>
<td>7.14</td>
</tr>
<tr>
<td>Education (years)</td>
<td>7 – 20</td>
<td>13.92</td>
<td>2.58</td>
</tr>
<tr>
<td>MoCA (raw Score)</td>
<td>15 – 30 (max: 30)</td>
<td>26.26</td>
<td>2.86</td>
</tr>
<tr>
<td>Event-based PM</td>
<td>0 – 8 (max: 8)</td>
<td>5.74</td>
<td>1.52</td>
</tr>
<tr>
<td>Time-based PM</td>
<td>0 – 8 (max: 8)</td>
<td>3.88</td>
<td>1.97</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Percentage Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>31.6</td>
</tr>
</tbody>
</table>

*Note: MoCA: Montreal Cognitive Assessment (Nasreddine et al., 2005). PM: prospective memory, measured with the Western Australia Prospective Memory Test (WAProm).*

A linear regression was conducted, predicting WAProm event-based and time-based scores from age, education and gender entered simultaneously. The model predicted 11% of the variance in event-based PM, $F (3, 207) = 8.57, p < .000$, and 19% of the variance in time-based PM, $F (3, 208) = 16.29, p < .000$. Coefficients used to generate predicted values and residuals for the main study are shown in Table S.3.8. Descriptive statistics and
percentiles for the resulting residuals in the standardisation sample are shown in Table S.3.9.
Table S.3.8.

*Regression coefficients from model predicting WAProm scores from age, education and gender.*

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Predictor</th>
<th>B</th>
<th>b</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event-based PM</td>
<td>Constant</td>
<td>8.52</td>
<td></td>
<td>.000†</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>-.29</td>
<td>-0.06</td>
<td>.000‡</td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>.10</td>
<td>0.06</td>
<td>.128</td>
</tr>
<tr>
<td></td>
<td>Gender</td>
<td>.12</td>
<td>0.38</td>
<td>.079</td>
</tr>
<tr>
<td>Time-based PM</td>
<td>Constant</td>
<td>9.81</td>
<td></td>
<td>.000‡</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>-.40</td>
<td>-0.11</td>
<td>.000‡</td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>.16</td>
<td>0.12</td>
<td>.014†</td>
</tr>
<tr>
<td></td>
<td>Gender</td>
<td>.01</td>
<td>0.05</td>
<td>.861</td>
</tr>
</tbody>
</table>

*Note:* †: $p < 0.05$; ‡: $p < 0.01$; $B$: standardized coefficient; $b$: unstandardized coefficient; PM: prospective memory, measured with the Western Australia Prospective Memory Test (WAProm).
<table>
<thead>
<tr>
<th>Percentiles</th>
<th>Event-based PM</th>
<th>Time-based PM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>1.43</td>
<td>1.77</td>
</tr>
<tr>
<td>10</td>
<td>-1.69</td>
<td>-2.41</td>
</tr>
<tr>
<td>20</td>
<td>-0.94</td>
<td>-1.58</td>
</tr>
<tr>
<td>30</td>
<td>-0.60</td>
<td>-0.95</td>
</tr>
<tr>
<td>40</td>
<td>-0.21</td>
<td>-0.27</td>
</tr>
<tr>
<td>50</td>
<td>0.12</td>
<td>0.16</td>
</tr>
<tr>
<td>60</td>
<td>0.58</td>
<td>0.43</td>
</tr>
<tr>
<td>70</td>
<td>0.87</td>
<td>1.03</td>
</tr>
<tr>
<td>80</td>
<td>1.27</td>
<td>1.50</td>
</tr>
<tr>
<td>90</td>
<td>1.59</td>
<td>2.19</td>
</tr>
</tbody>
</table>

PM: prospective memory, measured with the Western Australia Prospective Memory Test (WAProm).
Analysis of group level changes

As a supplement to our analysis of individual differences in change using regression, we also analysed group-level changes in PM, objective sleep, and insomnia symptom severity. The effect of time (pre-intervention vs post-intervention) and condition (control vs CBT-I) on time and event-based PM performance, actigraphy sleep variables, and ISI scores was examined using repeated measures analysis of variance (ANOVA). We expected a significant interaction between time and condition, such that participants in the CBT-I group would show better PM, better objective sleep and reduced insomnia symptoms at post-intervention.

We also conducted repeated measures ANOVA (time (pre-intervention vs post-intervention) x condition (control vs CBT-I)) to explore potential changes in aspects of cognition measured pre-intervention and post-intervention alongside PM. These were the cognitive domains hypothesized to underlie PM, namely retrospective memory and executive function (Kliegel et al., 2016), measured with the CVLT long-delay free recall standardized score and the standardized difference score for Trail Making Test A versus Trail Making Test B, respectively.

For PM, there was no significant effect of time on event-based scores, $F(1, 19) = 1.04, p = .320$, or interaction with condition, $F(1, 19) = 1.97, p = .177$. For time-based scores, there was a significant effect of time with higher scores at post-intervention, $F(1, 19) = 7.47, p = .013$, however, this was equivalent across conditions, $F(1, 19) = 0.22, p = .644$.

In regards to objective sleep, there was a significant effect of time on TST, $F(1, 19) = 7.51, p = .013$, which increased post-intervention, equivalent across conditions, $F(1, 19) = 2.07, p = .167$. There were no other significant effects of time, or interactions with
condition, for the other sleep variables (sleep efficiency: time $F(1, 19) = 0.30, p = .589$; condition, $F(1, 19) = 0.12, p = .734$; awakening length: time $F(1, 19) = 1.13, p = .302$; condition $F(1, 19) = 0.48, p = .498$; WASO: time $F(1, 19) = 0.37, p = .551$; condition $F(1, 19) = 0.14, p = .716$).

For insomnia severity, there was a significant effect of time on ISI scores, $F(1, 19) = 5.21, p = .034$, and an interaction with condition, $F(1, 19) = 5.73, p = .027$, such that insomnia symptoms were reduced post-intervention in the CBT-I, but not control, condition.

In regards to other cognitive domains, there was a significant effect of time on retrospective memory, $F(1, 19) = 21.98, p < .001$, which improved significantly post-intervention. There was also a significant interaction with condition, $F(1, 19) = 5.81, p = .026$, such that the control group improved more than the CBT-I group (see Table 3.2, main text). Executive function did not show significant effects of time, $F(1, 19) = 0.12, p = .723$, or interaction with condition, $F(1, 19) = 0.36, p = .554$. 

Supplementary materials for Chapter 4.

Prospective memory costs

No previous studies have examined ongoing task costs following PM intervention in older adults. Thus, we calculated PM costs to allow examination of potential difference in cognitive demand placed by implementation intentions or GMT. These were calculated as the difference between average ongoing task RT for word trials in block 1 compared to block 2 (pre-intervention PM cost), and block 1 compared to block 3 (post-intervention PM cost).

There were significant differences in pre-intervention PM cost (Table S.4.4), such that participants who went on to receive implementation intentions showed less cost to ongoing task RT when the PM task (pre-intervention) was added, $F(2, 154) = 6.775, p = .002$. Thus, PM costs were adjusted by dividing by baseline ongoing task RT (word trials only). Adjusted PM cost, reflecting change in RT when the PM task (pre-intervention) is added as a proportion of average baseline ongoing task RT, remained significantly lower in the implementation intentions group, $F(2, 154) = 7.551, p = .001$.

When pre-intervention adjusted costs were controlled for, there were no differences between the intervention groups (GMT, implementation intentions, no intervention) in post-intervention adjusted PM costs, $F(2, 156) = 1.004, p = .369$. Additionally, the lack of increase in PM costs following the intervention suggests implementation intentions is able to improve older adults PM without significant costs to the ongoing task, consistent with some but not all results in young adults (Chang et al., 2014).
Table S.4.4.

Prospective memory costs.

<table>
<thead>
<tr>
<th></th>
<th>Full sample (N = 157) mean (SD)</th>
<th>No intervention condition (N = 53) mean (SD)</th>
<th>II condition (N = 49) mean (SD)</th>
<th>GMT condition (N = 55) mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimum - maximum</td>
<td>-305.13 – 256.90</td>
<td>73.30 (64.57)</td>
<td>99.91 (58.50)†</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>75.21 (72.32)</td>
<td>73.03 (85.18)†</td>
<td></td>
</tr>
<tr>
<td>Pre-intervention</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM cost (ms)</td>
<td>-365.17 – 273.83</td>
<td>61.03 (90.37)</td>
<td>28.73 (114.88)</td>
<td></td>
</tr>
<tr>
<td>Post-intervention</td>
<td></td>
<td>56.97 (71.63)</td>
<td>93.72 (69.78)</td>
<td></td>
</tr>
<tr>
<td>PM cost (ms)</td>
<td>Adjusted* Pre-intervention PM cost</td>
<td>56.97 (71.63)</td>
<td>93.72 (69.78)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.26 – 34</td>
<td>.10 (.08)</td>
<td>.07 (.10)†</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Adjusted* Post-intervention PM cost</td>
<td>-.32 – 52</td>
<td>.08 (.09)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-.32 – 52</td>
<td>.09 (.11)</td>
<td>.05 (.13)</td>
<td></td>
</tr>
</tbody>
</table>

Note: * expressed as a proportion of ongoing task response time; PM: prospective memory; II: implementation intentions; GMT: goal management training; † p < .05. Pre-intervention/ post-intervention PM cost: difference between baseline OT RT and pre-intervention/ post-intervention OT RT for word trials only; Pre-intervention/ post-intervention adjusted PM cost: difference between baseline OT RT and pre-intervention/ post-intervention OT RT for word trials only, divided by baseline OT RT for word trials only.
**Implementation intentions script:**

For the implementation intentions intervention, participants were read the following script:

“To assist you with the task, we are now going to provide you with some strategies. Press any key to proceed to these strategies.”

*Computer screen shows a summary of information read in script.*

“Now, please take a few moments to imagine yourself responding to a word representing [a fruit/ an animal]. Please visualise yourself making a word-nonword judgment when encountering [a fruit/ an animal] word. Then imagine yourself pressing the ‘X’ key in response to seeing this word. Take a few moments, close your eyes and imagine seeing [a fruit/ an animal] word after which you will press the ‘X’ key. Press any key to practice the above exercise for 30 seconds.”

*Computer screen shows prompt to practice implementation intentions, on 30 second timer.*

“Now, please turn around and say to me three times "when I see [a fruit/ an animal] word, I will press the ‘X’ key".

*Task continues.*

**GMT script:**

For the GMT intervention, participants were read the following script:

“To help you with the tasks, we are now going to provide you with a set of problem solving strategies involving 5 steps. Press any key to beginning reviewing the steps.”

*Participant given laminated GMT flowchart. Computer screen shows five steps.*
“For now, take a few moments to briefly review the 5 steps and the flowchart given to you. I will shortly elaborate each step with you.”

Examiner gestures to “Stop” on the flowchart.

“STOP. In this step, you say “STOP” to yourself to make sure you are on track. Think about what you are supposed to be doing and ensure you are not distracted or off task. For example, let’s say you are watching TV and you remember you need to go see the physio in 30 minutes. You don’t need to physically stop everything you are doing, but just think for a moment “STOP – what am I doing?” The purpose of this step is that you pause and think about what you are doing, so that you don’t get distracted or carried away by other thoughts. In this word-nonword task, you can say “STOP” to yourself and think “what am I doing?” to check whether you are on-task.”

Examiner gestures to “Define” on the flowchart.

“DEFINE. In this step, after briefly pausing and asking “STOP, what am I doing?”, we define the main task or goal. So, we simply ask “what am I supposed to be doing here?” or “what am I trying to achieve?”. So for the current task, you would define the main activity as “make the word – nonword judgments as well as remember to press the ‘X’ key when needed.”

Examiner gestures to “List” on the flowchart.

“LIST. In this step, after defining the main goal, we list the subgoals to achieve the main goal. So for the current task, you would review in your mind the subgoals or substeps. Sub-step 1 is to ask yourself “Is the letter string [a fruit/ an animal]?” if yes, press the ‘X’ key. If no, go to the next substep. Sub-step 2 is to ask yourself “Is the letter string a real word?” If yes, press the ‘Yes’ key and if not, press the ‘No’ key.”

Examiner gestures to “Learn” on the flowchart.
“LEARN. In this step, let’s try to review and consolidate what we have been taught in the first 3 steps.”

*Participant is prompted to verbally recount the steps with assistance or correction if necessary, e.g.*

“Can you tell me what step 1 is?”

“And can you tell me what you must do at that step?”

*Once all steps have been recounted, examiner gestures to “Check” on the flowchart.*

“CHECK. In this step, we want you to check to see if you are doing what you planned to do. If you are on task (for example, making the word-nonword judgments and remembering to press the ‘X’ key), then great, keep going. However, if you have become distracted (for example, thinking of plans for dinner), use this time to get yourself back on task.”

“Every now and then, a "STOP" sign will appear on the screen for 15 seconds during the word-nonword task to remind you to review the 5 steps we have learned. When you see the "STOP" sign, please quickly review the steps learned from the beginning to the end. Press any key to see the sign.”

*Stop sign appears on computer screen.*

“Remember, please review the five steps when you see the "STOP" sign.

“Press any key to continue.”

“To test your understanding of the problem solving strategies, you will now be administered a brief quiz. Press any key to begin the quiz.”

*Participant completes the quiz.*
“To further familiarise you with the strategies, you will now be given some practice trials. As before, your task is do the word-nonword task as well as remember to press the ‘X’ key whenever [a fruit/ an animal] appears. Please remember to quickly review the steps when you see a "STOP" sign. Press the spacebar to proceed to practice trials.”

Participant completes GMT practice trials.

Task continues.
Figure S.4.4

Goal management training flowchart.

Adapted from Levine et al. (2000)