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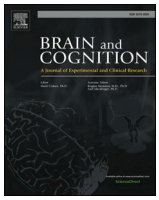

Age-related differences in neural integrity are unrelated to prospective memory age effects

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ABSTRACT

Prospective memory refers to memory for future intentions. In general, prospective memory appears to decline with age when tested in laboratory settings but is preserved or enhanced when tasks need to be completed in daily life. No study to date has tested whether age-related differences in specific brain structures and networks mediate prospective memory age effects in both settings. Here, measures of regional brain volume (anterior prefrontal cortex, frontoparietal networks, and temporal lobes), white matter integrity (prefrontal white matter hypointensities) and prospective memory were obtained from 41 younger and 41 older adults. The results showed that, as expected, older age was associated with smaller regional brain volumes, as well as poorer prefrontal white matter integrity. In addition, age was negatively associated with prospective memory function in the laboratory-based assessment, but positively associated with performance on the task completed in daily life. However, none of these behavioural effects were mediated by age-related differences in neural integrity. These data show that, in contrast to literature focused on neurodegenerative disease in which neural losses have been shown to be predictive of PM impairment, age-related differences in brain integrity may not be the best indicator of *normal* variation in prospective memory function.

1. Introduction

Prospective memory (PM) refers to the process of forming a future intention and remembering to execute that intention. Given the critical importance of PM for functional independence in older adulthood (Hering et al., 2018; Sheppard et al., 2020), it is unsurprising that hundreds of studies have now been conducted focused on trying to establish when and why PM is affected by normal adult ageing. However, this literature reveals substantial heterogeneity, with not all types of PM affected equivalently (Laera et al., 2023), and at least some types of PM remaining relatively intact (Craik and Henry, 2023).

Because PM is a complex multicomponent process that places demands on a wide range of neural structures and networks and their connectivity (Henry, 2021), and brain changes do not occur to the same extent in all brain regions as we age (Zhao et al., 2019), at least some of this age-related variance in PM function might be linked to age-differences in the integrity of neural structures. However, to date, while many studies have now been published that provide evidence for functional age differences in the brain systems that subserve PM function (e.g., Peira et al., 2016; Gonneaud et al., 2017; Lamichhane et al., 2018), few of the studies that have investigated structural correlates of PM in older adulthood have included a younger comparison group (Morand et al., 2020); most have either focused on healthy older adults in isolation or compared older adults to a clinical group with actual or suspected pathology.

In one study that focused solely on healthy older adults, Scullin et al. (2013) reported data from cognitively normal older adults and found that those with a history of hypertension exhibited poorer PM function and had reduced prefrontal white matter relative to those with no such history. Collapsed across groups, there was an association between PM function and white matter volume in the anterior prefrontal cortex (aPFC), but not in the medial temporal lobe (MTL), dorsolateral

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prefrontal cortex (DLPFC), or parietal regions; there were also no associations between PM accuracy and gray matter volume. This study therefore provided only limited evidence for a relationship between normal age-related brain changes and PM function, and suggests that, if there is one, it might be driven by hypertension and not age *per se*.

In a later study, Scullin (2020) investigated the structural correlates of commission errors, a specific type of error in which a PM task is erroneously completed (as in accidental medication double-dosing). The results revealed that, in healthy older adults, higher commission error risk was associated with reduced lateral orbitofrontal cortex (OFC) but somewhat counterintuitively, *increased* MTL/hippocampal and gray matter volume, highlighting a complex brain-behaviour relationship. However, because a measure of commission errors was used, and not a measure of PM function, this study did not speak to the structural correlates of PM function more broadly.

In a later study that directly compared healthy older adults to younger adults, Morand et al. (2020) compared 23 older and 22 younger adults on a laboratory-based measure of time-based PM. Although this study identified patterns of age-related gray matter atrophy that were consistent with broader ageing literature, and that included several regions crucial to PM, the volume of these brain regions was unrelated to PM function in the older group. However, fractional anisotropy values in several white-matter tracts connecting frontal and occipital regions did correlate with PM function in the older, but not the younger group.

Taken together, these studies do not provide clear or consistent support for a structural neural basis in understanding normal age-related *variation* in PM performance. Importantly however, this contrasts with studies that have assessed older cohorts with actual or suspected pathology, which collectively provide much stronger evidence for a neural structural basis for *impairment* (Dermody et al., 2016; Gordon et al., 2011; Hsu et al., 2019; Liu et al., 2021; Nurdal et al., 2020; Scullin et al., 2020; Scullin et al., 2013). Further limiting our understanding of the structural correlates of PM in older adulthood, is that to date, all studies have been confined to the laboratory, with no study assessing PM functioning in an everyday setting. This is an important omission given that PM difficulties observed in laboratory-based tasks do not necessarily generalise to impairment in everyday life, a phenomenon that in the context of normal adult ageing is referred to as 'the age PM paradox' (Aberle et al., 2010; Peter and Kliegel, 2018; Phillips et al., 2008; Rendell and Craik, 2000, Schnitzspahn et al., 2020, although see Koo et al., 2021).

The present pre-registered study was therefore designed to extend prior literature focused on the neural structural correlates of PM age effects by including both lab-based and naturalistic measures of PM to provide the first test of whether age effects in PM function on these different types of test might, in part, be explained by age-differences in neural structural integrity. In addition to testing whether age-related volumetric differences in the grey matter structures and systems thought to particularly subserve PM function explain age-related differences in PM function, following on from Scullin et al. (2013), we also sought to test whether the white matter integrity of the aPFC might be important here. For each of our analyses it was anticipated that, for lab-based PM performance, stronger structural integrity in the brain region of interest would be associated with better lab-based performance for older adults. However, given that older adults sometimes outperform younger adults on PM measures completed in daily life, it was difficult to predict whether stronger – or poorer – structural integrity in the regions of interest would be associated with better daily PM performance for older adults. Consequently, although we predicted that neural structural integrity would mediate age differences on both types of tasks, only for lab-based tasks did we have *a priori* predictions in relation to the directionality of these effects. Detailed in the following sections are overviews of literature that speak to why each specific brain structures and networks may mediate the prospective memory age effects.

1.1. Gray-matter integrity

Two distinct frontoparietal networks are thought to play a key role in PM. The dorsal frontoparietal network is believed to mediate the engagement of strategic monitoring resources, while the ventral frontoparietal network is thought to support more spontaneous retrieval processes (Cona et al., 2015). The distinction between strategic monitoring and spontaneous retrieval processes is also particularly important in relation to age effects given that dual process models of ageing predict that, while controlled processes are subject to age-related decline, more automatic processes are relatively immune to age effects (Craik and Henry, 2023).

With respect to PM specifically, a central tenet of prominent frameworks (e.g., Scullin et al., 2013), is that automatic *or* attention-driven processes can support PM, but their relative importance varies. However, while the distinction between strategic demands is most often discussed in relation to task parameters such as cue focality or salience, a more fundamental distinction may be between tasks completed in the laboratory versus those completed in everyday life.

As mentioned previously, a robust finding is that older adults perform more poorly than younger adults on laboratory PM tasks with high attention demands but are superior on most naturalistic tasks—a pattern known as the age-PM paradox (Phillips et al., 2008; Rendell and Craik, 2000; Menéndez-Granda et al., 2025). Here, differences in the availability of environmental support are believed to play a key role (Craik and Henry, 2023; Haines et al., 2020). Environmental support is typically absent in the laboratory, but plentiful in daily life and potentially 'triggers' the to-be-remembered event, thereby supporting more spontaneous retrieval processes – and reducing demands on more effortful ones. Additionally, whereas in the lab, multiple PM tasks typically need to be completed within a brief period, reinforcing the need for controlled, effortful monitoring, in real life, they are typically spaced out and completed over more extended temporal periods. The results of a recent meta-analysis also revealed that the level of abstraction of a task, as well as the familiarity of the environment in which the task needs to be executed, can both explain some of the differences in younger and older adults' performance (Menéndez-Granda et al., 2025).

Considering the different demands PM tasks completed in the lab relative to those completed in daily life likely place on strategic processing, our first hypothesis was that older adults' poorer performance on lab-based measures might, in part, be explained by greater age-related differences in the structural integrity of the dorsal frontoparietal network. By contrast, age-effects on a PM measure completed in daily life were predicted to be explained, at least in part, by age-related differences in the gray matter integrity of the ventral frontoparietal network, as this network supports spontaneous processing.

Another critical brain region implicated in PM function is the aPFC. Neuroimaging studies reveal distinct patterns of haemodynamic changes in the lateral and medial portions (activation and deactivation, respectively) of the aPFC during PM task performance, and these have been argued to provide a 'gateway' mechanism that mediates the capacity to engage in internal thought while concurrently attending to external stimuli (Burgess et al., 2003). Because there is greater age-related decline in prefrontal structures relative to many other brain regions (Bartzokis et al., 2001; Gunning-Dixon et al., 2009; Raz et al., 2005), the second hypothesis was that age-related differences in the volumetric integrity of the aPFC might also, in part explain age-effects in PM function.

Medial temporal lobe structures also play a key role in episodic memory and should theoretically also be linked to PM. Although functional neuroimaging support for their involvement is mixed (Cona et al., 2015), structural MRI data from clinical populations more consistently indicates that the integrity of these regions is related to PM performance. For instance, hippocampal atrophy strongly correlates with PM function across different dementia syndromes (Dermody et al., 2016), and cortical thickness in hippocampal subfields correlates with performance

on the retrospective component of PM in prodromal Alzheimer's disease (Nurdal et al., 2020). Our third hypothesis was therefore that gray matter integrity in medial temporal lobe structures might also, at least in part, explain PM age effects.

1.2. Prefrontal white matter integrity

PM difficulties can also arise owing to disruption in the connectivity within and between neural networks (Henry, 2021). White matter lesions become more common with age, and while on computed tomography and T1-weighted MRI, these appear dark and are termed white matter hypointensities, and on a fluid-sensitive MRI protocol they appear bright and are termed white matter hyperintensities, they provide equivalent markers of white matter damage in normal ageing. For instance, white matter hypointensities and hyperintensities demonstrate equivalent correlations with age and CSF β-amyloid in cognitively intact older adults (Wei et al., 2019). In cognitively healthy older adults, poorer PM performance has been linked to reduced prefrontal white matter volume in cognitively healthy older adults (Scullin et al., 2013), and lower fractional anisotropy values in anterior tracts (Morand et al., 2020), as well as to an increased burden of white matter hyperintensities in people diagnosed with amnestic mild cognitive impairment (Yoon et al., 2018). Here, we therefore also provided the first test of whether age effects on PM measures might, in part, be explained by age differences in the burden of prefrontal white matter hypointensities. Hypointensities were used to quantify white matter lesions because of the structural imaging data acquisition approach used.

1.3. Aims and hypotheses

The overall aim of the study was to provide the first test of whether age-related differences in specific brain structures and networks mediate prospective memory age effects across tests completed in the lab, as well as in naturalistic settings. The primary hypotheses were that, for lab-based PM performance, stronger structural integrity in the brain region of interest would be associated with better lab-based performance for older adults. However, our secondary hypotheses were simply that neural structural integrity would mediate age differences on naturalistic tasks, i.e., we did not have *a priori* predictions in relation to the directionality of these latter effects owing to the fact older adults sometimes outperform younger adults on PM measures completed in daily life.

2. Methods

2.1. Participants

Prior research on PM typically reveals moderate-to-large sized age-related losses in lab-based settings, and equivalent sized age-related improvements in naturalistic settings (Henry et al., 2004). A power analysis using G*Power 3.1 software revealed the minimum number of participants required to detect this size of group difference (*t*-test) effect ($d = 0.70$) with adequate power (80 %) is 68 participants. G*Power 3.1 also revealed that, to detect moderate-large multiple mediation effects (for an $F2 = 0.20$) with the same level of power, the minimum number of participants required is 59). A total of 82 participants were therefore recruited from community settings and received $60 for their participation or course credits (41 younger adults aged between 18–31 years, and 41 older adults aged between 60 and 83). All older adults completed a cognitive screen (the Mini-Mental State Exam; MMSE) and were required to score above the recommended cut-off (≥ 27). Exclusionary criteria for all participants included the presence or history of serious psychiatric or neurological illness. Three older adults were excluded due to low scores on the MMSE, so the final sample comprised 41 younger adults (*Mage* = 22.98, *SD* = 4.19) and 38 older adults (*Mage* = 70.37, *SD* = 5.62). Seven participants (four younger, three older) were excluded from the naturalistic PM analyses due to task incompleteness.[1] The full demographic breakdown is displayed in Table 1. The study was pre-registered on the Open Science Framework (https://osf.io/27adq/?view_only=9216671299d54c4d80e4e84046665cc5).

2.2. Material and procedure

Ethics approval was provided by (details omitted to preserve bind review). Participants were tested individually in a 2–3-hour laboratory testing session in which they completed the measures of PM as well as a broader behavioural assessment. This session also included the structural neuroimaging component. All participants provided written informed consent, then completed a real-life measure of PM in their everyday lives (MEMO). Specifically, after providing demographic information and completing a brain scan session, participants were taken to a separate room in which they completed all the lab-based behavioural tests reported in this study, which were presented in counterbalanced order to minimise potential order effects. All participants were then provided with standardized instructions on how to complete the measure of PM that needed to be completed in their everyday lives (MEMO), which they were asked to complete over the next week.

2.2.1. Background assessments

To broadly characterize cognitive function, all participants completed a range of validated measures. These were measures of executive functioning (Go-No Go), verbal learning and memory (Hopkins Verbal Learning Test), working memory (N-back), and processing speed (Choice Reaction Time). All participants also completed a questionnaire pack that included measures of mental health (the Hospital Anxiety and Depression Scale and the State Trait Anxiety Inventory), quality of life (the 12-item Short Form Survey), self-reported memory (the Multifactorial Memory Questionnaire) and a measure of the tendency for an individual to engage in and enjoy thinking (The Need for Cognition Scale). Performance on these background measures is reported in Supplementary Table 1.

2.2.2. Laboratory measures of prospective memory

Three validated laboratory measures of PM were administered: The Cambridge Prospective Memory Test (CAMPROMPT; Wilson et al., 2004), Memory for Intentions Screening Test (MIST; Raskin, 2009), and Virtual Week (Rendell and Craik, 2000). The CAMPROMPT includes six PM tasks with variable delay intervals, completed while engaged in a range of ongoing distractor tasks. The total in-session assessment lasted ~25 min. The MIST is another widely used and validated behavioural assessment with a total in-session administration time of ~30 min. In total, eight PM tasks are presented, which vary according to cue type (time versus event), delay interval (2 min, 15 min or 24 h delay), and response type (verbal versus action), with a word search puzzle being used as the ongoing task. Virtual Week is a multi-intention paradigm that requires the planning and execution of multiple PM tasks while engaged in a board game setting. As participants move around the board, they are required to make choices about plausible daily activities and remember to carry out PM tasks (regular/irregular event-based, time-of-day, and time-interval tasks). A correct response was defined as one where the participant completed the appropriate PM task in response to the relevant cue. More specifically, responses were categorized as correct if they were performed when the token arrived at (or just passed) the target position on the board, and before the next die roll. In regard to time-interval tasks, responses were categorized as correct if the

[1] Lab-based analyses were completed with and without these seven participants. As the pattern of results did not change, they were retained.

Table 1
Demographic breakdown separated by younger and older adult participants and prospective memory task-type analyses.

	Lab-Based PM Tasks			Naturalistic PM Task		
	Younger Adults $n = 41$	Older Adults $n = 38$	Group difference	Younger Adults $n = 37$	Older Adults $n = 35$	Group difference
Age *M* (SD)	22.98 (4.19)	70.37 (5.62)	$t(77) = 42.69$***	23.16 (4.32)	70.14 (5.76)	$t(70) = 39.28$***
Gender(F:M)	25:16	27:11	$X^2(1) = 0.89$	23:14	24:11	$X^2(1) = 0.33$
Education *M* (SD)	15.54 (2.33)	17.09 (3.99)	$t(77) = 2.14$*	15.54 (2.39)	17.39 (3.98)	$t(70) = 2.40$*
MMSE *M* (SD)	29.34 (0.83)	29.00 (0.90)	$t(77) = 1.76$	29.35 (0.75)	29.09 (0.89)	$t(70) = 1.37$

Note. PM = prospective memory. *$p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

task was completed within 10 s of the target time.

For each of these measures, the percentage of correct responses was used as an indicator of PM performance. Because performance across these three measures of PM was significantly correlated ($rs = 0.45$, 0.50 and 0.36 between Virtual Week and MIST, Virtual WEEK and CAMPROMPT, and MIST and CAMPROMPT, respectively), to provide a more stable measure of laboratory-based PM, a single composite score was calculated that was the average percentage of correct scores of the three constituent PM tasks.

2.2.3. Naturalistic measure of prospective memory

MEMO (Haines et al., 2020) involved participants carrying a dedicated smartphone so that they can use a customised application and the smartphones' camera function to index PM performance in their actual daily lives. For time-based tasks, each morning participants are told that they need to complete quiz activities at two specific pre-determined times (over which they are given some choice, to help ensure that they can complete the quiz at allocated times), and two at random points throughout the day (i.e., participants will be asked if they can complete a quiz in 10, 15, or 20 min time). At these times participants are required to open the application to initiate the time-based PM task. Each morning MEMO also provides a list of four events participants need to remember to photograph that day. The act of photographing and uploading these events serves as the event-based PM tasks. Half of these events are selected by the participant at an earlier point in time from a list of common activities and events. Only events identified as "extremely likely to occur during the testing days" are selected; the remainder are events experienced by most people daily, such as brushing teeth. In total, participants completed two full days of MEMO. Due to low engagement in the time interval task ($n = 15$; e.g., participants not looking at the phone frequently enough to see the notification), only the time and event-based tasks were included (maximum score of 12). To allow for direct comparison with the lab-based measure, data were converted to proportion correct.

2.3. Structural imaging data acquisition

Data were collected on a MAGNETOM Skyra 3T MR scanner (Siemens Healthcare, Erlangen, Germany) equipped with a 32-channel headcoil. Whole-brain anatomical images were acquired using an MP RAGE sequence (Brant-Zawadzki et al., 1992) with an isotropic voxel size of 1.0 mm and the following sequence parameters: TE of 2.07 ms, TR of 2300 ms, flip angle of 9 degrees, TI of 900 ms. To take advantage of parallel imaging capabilities, GRAPPA (Griswold et al., 2002) was used with an iPAT acceleration factor of 3. Regions of interest were extracted using a standard brain atlas (The Deskian-Killiany Atlas, Desikan et al., 2006).

2.4. Structural imaging data processing

The anatomical images were processed using FreeSurfer to automatically segment and classify various tissue types (Dale et al., 1999). The automatic tissue segmentations were then assessed for quality control and manually corrected when necessary[2] (Guenette et al., 2018; McCarthy et al., 2015). The quality control and correction procedures were guided by Qoala-T – a supervised learning tool that automatically assesses the quality of the FreeSurfer-processed scans (Klapwijk et al., 2019). Grey matter volume (mm^3) and white matter hypointensities (μl) were extracted from our pre-defined regions of interest (ROIs) as per the predictions. Brain corrections were made for volume in relation to estimated intracranial volume, to account for sex differences and the cross-sectional nature of this study (Backhausen et al., 2022).

2.5. Statistical analyses

A series of multiple mediated regressions with age as the independent variable, and PM performance (separately for lab-based and naturalistic) as the outcome variable were performed in SPSS with the use of Hayes' PROCESS macro (Model 4) with 5000 bootstrap samples (Hayes et al., 2017). As per the pre-registration, each of these analyses were conducted separately for raw and normalized brain data. As the two approaches yielded equivalent results, only the normalized results are reported here. There were three cases of missing data that were inputted with the group mean (three younger adults within the time-based MEMO task).

To test Hypothesis 1, two models with three potential mediators were completed. In the first, the superior parietal lobe, superior frontal lobe, and precuneus were included as the regions of interest comprising the dorsal frontoparietal network. For the second model, the supramarginal gyrus, inferior parietal lobe, and the rostral middle frontal were included to capture the ventral frontoparietal network. To test Hypothesis 2, a model with two potential mediators (lateral and medial aPFC gray matter volume) was assessed. To test Hypothesis 3, a model with two potential mediators (hippocampal and parahippocampal gray matter volumes) was followed. Finally, to test Hypothesis 4, a simple mediation was performed with prefrontal white matter hypointensities included as the potential mediator.

Finally, Supplementary Table 1 provides comparisons of younger and older participants' performance on all background measures (cognitive and self-report), conducted using independent sample *t*-tests; Supplementary Table 2 reports means, standard deviations and bivariate correlations of normalized brain data with age and prospective memory task type; Supplementary Tables 3a to 3e report all pre-registered multiple mediated regressions described above, using the raw brain data.

[2] Eight participants (two younger, six older) were flagged during the manual correction. However, as the pattern of results did not change with and without their exclusion, these participants were retained for all analyses.

3. Results

3.1. Age-related differences in dorsal and ventral frontoparietal networks and PM function

As shown in Table 2, age differences on neither the lab-based nor naturalistic PM measures were explained by age-related gray matter differences in the dorsal or ventral frontoparietal networks.

For the lab-based analyses, age predicted lower gray matter volume within the dorsal and the ventral frontoparietal networks. However, support for Path b was not found, as gray matter integrity within the dorsal and ventral frontoparietal networks did not predict PM performance. Age remained a significant negative predictor of lab-based PM performance after controlling for the potential mediators. The total indirect effects were non-significant, (dorsal frontoparietal network: $B = 0.08$, $SE = 0.05$, 95 % CI[-0.01, 0.18], $\beta = 0.14$; ventral frontoparietal network: $B = 0.09$, $SE = 0.06$, 95 % CI = [-0.02, 0.21], $\beta = 0.15$).

For the naturalistic task analyses, age predicted lower gray matter volume within the dorsal and the ventral frontoparietal networks. However, support for Path b was not found, with gray matter integrity within the dorsal and ventral frontoparietal networks not predictive of PM performance. Age remained a significant positive predictor of PM performance after controlling for the potential mediators. The total indirect effects were non-significant, (dorsal frontoparietal network: $B = -0.06$, $SE = 0.11$, 95 % CI[-0.27, 0.16], $\beta = -0.07$; ventral frontoparietal network: $B = 0.09$, $SE = 0.12$, 95 % CI[-0.12, 0.34], $\beta = 0.10$).

3.2. Age-related differences in the aPFC and PM function

As displayed in Table 2, age-related differences in PM function were not explained by age-related gray matter integrity in either the lateral or medial aPFC. Specifically, while age was a predictor of lower gray

Table 2
Neural integrity and prospective memory functioning: multiple mediation results.

	Lab Based PM Tasks ($n = 79$)			Naturalistic PM Tasks ($n = 72$)		
	B	*SE*	*β*	*B*	*SE*	*β*
Dorsal frontoparietal networks						
Path a						
Age – Superior Parietal Lobe	-3.84×10^{-5}	7.53×10^{-6}	−0.50***	-3.72×10^{-5}	8.19×10^{-6}	−0.48***
Age – Superior Frontal Lobe	-9.37×10^{-5}	1.01×10^{-5}	−0.73***	-9.77×10^{-5}	1.08×10^{-5}	−0.73***
Age – Precuneus	-3.84×10^{-5}	5.20×10^{-6}	−0.64***	-3.95×10^{-5}	5.49×10^{-6}	−0.65***
Path b						
Superior Parietal Lobe – PM	100.65	810.29	0.01	1975.67	1649.63	0.17
Superior Frontal Lobe – PM	−385.18	501.29	−0.08	−769.95	1038.65	−0.11
Precuneus – PM	−1267.43	1158.12	−0.13	1545.94	2427.45	0.10
Path c'	−0.53	0.68	−0.91***	0.53	0.15	0.59***
Ventral frontoparietal networks						
Path a						
Age – Supramarginal Gyrus	-3.25×10^{-5}	6.87×10^{-6}	−0.47***	-3.28×10^{-5}	6.9×10^{-6}	−0.49***
Age – Inferior Parietal Lobe	-6.67×10^{-5}	8.29×10^{-6}	−0.68***	-6.76×10^{-5}	8.86×10^{-6}	−0.67***
Age – Rostral Middle Frontal	-7.27×10^{-5}	9.33×10^{-6}	−0.66***	-7.92×10^{-5}	9.53×10^{-6}	−0.70***
Path b						
Supramarginal Gyrus – PM	−940.23	712.92	−0.11	1594.93	1611.48	0.12
Inferior Parietal Lobe – PM	120.44	597.37	0.02	−1126.17	1265.41	−0.13
Rostral Middle Frontal – PM	−930.39	523.07	−0.17	−786.55	1158.44	−0.10
Path c'	−0.54	0.07	−0.92***	0.38	0.15	0.43*

	Lab Based PM Tasks ($n = 79$)			Naturalistic PM Tasks ($n = 72$)		
	B	*SE*	*β*	*B*	*SE*	*β*
Anterior prefrontal cortex (aPFC)						
Path a						
Age – Lateral	-2.29×10^{-5}	4.03×10^{-6}	−0.54***	-2.39×10^{-5}	4.07×10^{-6}	−0.57***
Age – Medial	-1.62×10^{-5}	2.39×10^{-6}	−0.61***	-1.70×10^{-5}	2.47×10^{-6}	−0.64***
Path b						
Lateral – PM	−195.55	1435.16	−0.01	−3130.71	3064.35	−0.15
Medial – PM	2883.46	2416.58	0.13	−2969.23	5060.46	−0.09
Path c'	−0.41	0.06	−0.70***	0.34	0.12	0.39***
Medial temporal lobe						
Path a						
Age – Hippocampus	-1.19×10^{-5}	2.16×10^{-6}	−0.53***	-1.25×10^{-5}	2.27×10^{-6}	−0.55***
Age – Parahippocampus	-4.06×10^{-6}	1.57×10^{-6}	−0.28*	-4.22×10^{-6}	1.52×10^{-6}	−0.31**
Path b						
Hippocampus – PM	−84.27	2561.15	0.003	−1611.55	5372.47	−0.04
Parahippocampus – PM	−439.36	3531.91	−0.01	−1732.09	8010.10	−0.03
Path c'	−0.45	0.05	−0.77***	0.44	0.11	0.50***
White matter hypointensities						
Path a	4.50×10^{-5}	1.09×10^{-5}	0.43***	4.64×10^{-5}	1.19×10^{-5}	0.42***
Path b	−252.83	448.44	−0.05	82.63	919.10	0.01
Path c'	−0.44	0.05	−0.75***	0.47	0.10	0.52***

PM = prospective memory. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

matter in both the lateral and medial aPFC, the two aPFC ROI's were not predictive of PM performance on either the composite lab-based or naturalistic task. Age remained a significant negative predictor of lab-based and positive predictor of naturalistic PM performance after controlling for the potential mediators. The total indirect effect was non-significant (lab based: $B = -0.04$, $SE = 0.04$, 95 % CI[-0.12, 0.03], $\beta = -.07$; naturalistic: $B = 0.13$, $SE = 0.08$, 95 % CI[-0.02, 0.29], $\beta = 0.14$.

3.3. Age-related medial temporal lobe integrity and PM function

As can be seen in Table 2, age-related differences in medial temporal lobe gray matter did not explain age effects in PM function. Specifically, while age was a predictor of gray matter atrophy in both the hippocampus and parahippocampus, the two medial temporal lobe ROI's were not predictive of PM performance on either the composite lab-based or naturalistic task. Age remained a significant negative predictor of lab-based and positive predictor of naturalistic PM performance after controlling for the potential mediators. The total indirect effect was non-significant (lab based: $B = 0.003$, $SE = 0.03$, 95 % CI[-0.06, 0.07], $\beta = 0.01$; naturalistic: $B = 0.03$, $SE = 0.07$, 95 % CI[-0.09, 0.15], $\beta = 0.03$).

3.4. Age-related prefrontal white matter hypointensities and PM function

The data reported in Table 2 also shows that prefrontal white matter integrity (as indexed by white matter hypointensities) does not explain significant variance in age differences in PM function. While analyses revealed age to be a significant positive predictor of prefrontal white matter hypointensities, white matter hypointensities did not predict PM performance. Age remained a significant negative predictor of lab based and positive predictor of naturalistic PM performance after controlling for the potential mediator. The indirect effect was non-significant (lab based: $B = -0.01$, $SE = 0.02$, 95 % CI[-0.05, 0.05], $\beta = -0.02$; naturalistic: $B = 0.004$, $SE = 0.04$, 95 % CI[-0.12, 0.09], $\beta = 0.004$).

4. Discussion

The present study provided the first test of whether age-related differences in specific brain structures and networks might help to explain age effects in PM function in a normal aging population, when these assessments are completed both in the lab as well as in daily life. The results are therefore important in identifying a typical profile of age-related differences on tests of PM function, but no mediating role of age-related structural brain differences.

As anticipated, age emerged as a significant predictor of lower gray matter in the lateral and medial aPFC, dorsal and ventral frontoparietal networks as well as increased prefrontal white matter hypointensities. Also as predicted, age emerged as a significant negative predictor of lab-based and positive predictor of naturalistic PM. However, none of these brain differences mediated any of the observed age effects in PM performance. These data therefore meaningfully extend prior studies that speak to whether there might be a relationship between normal age-related structural brain differences and PM function (Morand et al., 2020; Scullin et al. 2013; Scullin et al. 2020) and suggest that age-related differences in brain integrity may not be the best indicator of normal variation in prospective memory function. Although the brain is ultimately the seat of PM functioning, collectively such findings are consistent with there being compensatory mechanisms at the neural and/or behavioural level. For instance, function may be preserved despite volumetric losses if connectivity is intact, and normal aging is associated not only with decreases but also with some increases in white matter structural connectivity (Coelho et al. 2022). Interestingly too, although there are typically differences in the age effects identified for laboratory versus more naturalistic type tasks (Menéndez-Granda et al., 2025), here we are seeing a uniform pattern, whereby brain structure does not explain the relationship between age and performance for either type of task.

As noted, other studies to investigate the structural correlates of PM function in late adulthood have focused on older adults with actual or suspected pathology (Dermody et al., 2016; Hsu et al., 2019; Liu et al., 2021; Nurdal et al., 2020). Yet even here, while these studies have consistently revealed moderate to strong brain-behaviour relations (particularly in relation to hippocampal involvement), they also highlight how the precise nature of this relationship may differ depending both on the type of pathology, as well as the type of PM measure used in the assessment.

For instance, Nurdal et al. (2020) found that in a heterogeneous group of older adults with suspected, actual, or no cognitive impairment, poorer PM performance was associated with a reduction in the thickness of bilateral frontal-temporal-parietal cortex, as well as volumetric losses in a specific hippocampal subfield. Dermody et al. (2016) also found that hippocampal atrophy correlated strongly with PM integrity in Alzheimer's disease and behavioural-variant frontotemporal dementia but noted that dissociable neural systems also contributed to PM dysfunction in each group. Gordon et al. (2011) examined the structural correlates of PM accuracy in an older cohort, approximately half of whom had mild dementia. Focusing on four distinct ROIs and two distinct types of PM task (focal and non-focal), the results revealed a positive association between MTL volume and performance on the focal PM task, with this relationship strongest for the hippocampus. However, no associations were identified between structural integrity and performance on the non-focal PM task. Contrasting with these three studies, Liu et al (2021) identified a particularly strong role for prefrontal regions in supporting PM function across various types of dementia. However, PM was indexed solely via subjective ratings which is problematic in older cohorts (Thompson et al., 2015). Finally, Hsu et al. (2019) compared older adults with actual or suspected cognitive impairment and found that PM function was associated with the microstructural integrity of major cerebral pathways connecting the frontal lobe with posterior regions.

When considered together, while previous research focused on normal ageing fails to provide clear or consistent support for a neural basis in understanding normal age-related *variation* in PM performance, prior studies focused on clinical populations provide stronger evidence for a neural basis for impairment. Most evidence from these studies points to a key role for the hippocampus, which may be important in understanding the excellent clinical sensitivity that PM shows to MCI and dementia, even prospectively. For instance, PM function has been shown to predict future cognitive decline and incident dementia even after controlling for broader cognitive function (Browning et al., 2023), and a longitudinal study of 511 people aged 60–90 revealed that hippocampal atrophy predicted dementia during a 6-year follow-up period (den Heijer et al., 2006).

4.1. Limitations and future directions

A few limitations to this study should also be noted. First, the cross-sectional nature of the current results limits the scope of interpretation of our findings. However, although statistical mediation analysis cannot confirm a causal relationship when identified on cross-sectional data, it provides an important first step into plausible underlying biological mechanisms that allow refinement of further hypotheses which can be tested in future studies (Salthouse, 2011). Second, our sample size is relatively modest when compared with some other cohorts. However, it is comparable, and in some cases substantially larger, than almost all prior studies to investigate the structural correlates of PM, including some that have revealed structural losses are related to PM deficits in the context of hypertension or neurological illness (Dermody et al., 2016; Hsu et al., 2019; Nurdal et al., 2020; Scullin et al., 2013). Moreover, the study was sufficiently powered to detect direct effects (i.e., Paths A, B and C′). Although the study had less power to detect mediation (i.e., indirect effect) than direct effects, critically, evidence for Path B was

consistently not found (i.e., no brain region predicted PM performance). To demonstrate mediation *both* Paths A and B need to be present (Kenny, 2023). The absence of a direct effect for Path B therefore means that the absence of mediation cannot be attributed to a lack of power. Instead, these data suggest that the significant bivariate correlations found between the potential mediating brain regions and PM performance, were simply due to age.

It is also important to note that, although the MEMO was designed to be highly accessible, the proficiency of the older participants in using the MEMO application in their everyday lives could indicate a sample of high functioning older adults, and this may limit the generalizability of this study's findings to participants with lower levels of digital proficiency or cognitive functioning. Given that previous research also suggests that any relationship between normal age-related brain changes and PM function might be driven by hypertension (Scullin et al., 2013), it was unfortunate that we did not have access to participants' hypertension status to provide a further test of this idea. In addition, as is often the case in between-group designs, the two groups were found to differ on some of the background assessments. This means that it is important to acknowledge that factors other than age may potentially have contributed to the between-group effects identified here. Indeed, while this study identified significant age effects on neural structures, it is possible that these may have been smaller if we had used longitudinal research methods. Broader literature shows how in cognitive domains, age-effects are typically larger in cross-sectional relative to longitudinal designs, and recent studies suggest that this also extends to PM (Zuber et al., 2025). Neural changes within participants may similarly be smaller than cross-sectional comparisons suggest, and this highlights the need for longitudinal examinations that capture within-subject variation.

It is also important to acknowledge that the metrics used to quantify age-related differences in brain morphology may not have been sufficient to appropriately characterize the mediating role of age-related atrophy in these regions. For instance, because fractal dimensionality captures not only age-related changes in gray matter volume but also brain shape complexity, such as degree of cortical folding (Madanm, 2021), it may provide a more sensitive indicator of age-related brain differences that is better able to capture any mediating role of age-related brain atrophy in PM age effects. It is also of course possible that specific sub-regions of the brain areas examined could mediate this relationship, but this was masked by taking the volume of the entire networks. Future research is therefore also needed that considers each of the brain regions considered here, but at this more nuanced, subregional level. Because cognitive function does not exist in a vacuum, but is shaped by a lifetime's experience and set against a rich social environment, it is also important to continue to consider other non-brain based factors that may contribute to age-differences in PM function, including (but not limited to) age-based stereotype threat, cognitive offloading, motivational shifts, experience and expertise (Henry, Grainger & von Hippel., 2023).

Finally, the regions selected for the dorsal frontoparietal network (superior parietal lobe, superior frontal lobe, precuneus) and ventral frontoparietal network (supramarginal gyrus, inferior parietal lobule, rostral middle frontal gyrus) were chosen based on their established roles in top-down and bottom-up attentional control, respectively, which are critical for PM. However, the specific regions in our study do not perfectly align with those identified by others, such as Corbetta and Shulman (2002) and Cona et al. (2015). While the functional roles of these regions in attentional control and executive function (which are essential for PM, see Henry, 2021), provide a strong rationale for their inclusion in the dorsal and ventral frontoparietal networks, we have also made our data freely available to others so that other researchers are able to reanalyse this data with the same or different ROIs (https://osf.io/m5gcp).

4.2. Conclusions

Many daily PM tasks are critical for the maintenance of independence, such as remembering to take medication, to check food cooking, to turn off appliances or to pay bills, and this means that it is difficult to overstate the fundamental role of PM in older adults' everyday lives. In contrast to literature focused on neurodegenerative disease, in which volumetric losses (particularly in the hippocampus) have been shown to be strongly predictive of behavioural PM losses, this study shows that that normal age-related differences in prospective memory function are not explained by age-related differences in brain structure. Such findings therefore suggest that normal age-related differences in neural structural integrity may not be the best indicator for prospective memory function in older cohorts.

CRediT authorship contribution statement

Julie D. Henry: Writing – original draft, Supervision, Resources, Investigation, Funding acquisition, Conceptualization. **Sarah P. Coundouris:** Writing – review & editing, Methodology, Investigation, Formal analysis. **Izelle Labushagne:** Writing – review & editing, Methodology, Investigation. **Kirra Liu:** Writing – review & editing, Project administration, Methodology. **Simon Haines:** Writing – review & editing, Project administration, Methodology. **Sarah A. Grainger:** Writing – review & editing, Methodology. **Juan F. Domínguez:** Methodology. **Alex Puckett:** Writing – review & editing, Methodology, Investigation. **Peter G. Rendell:** Writing – review & editing, Supervision, Resources, Methodology, Investigation, Conceptualization. **Jessica Taubert:** Writing – review & editing, Methodology, Investigation, Formal analysis.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bandc.2025.106301.

Data availability

This study was preregistered on the Open Science Framework (https://osf.io/27adq/?view_only=9216671299d54c4d80e4e84046665cc5). Extensive supplementary materials are also available: Supplementary Table 1 provides younger and older participants' performance on all background measures. Supplementary Table 2 reports descriptive statistics and bivariate correlations of normalized brain data with age and prospective memory

task type. Supplementary Tables 3a to 3e report precise statistics for the raw brain data mediation analyses, separately for the dorsal frontoparietal network, ventral frontoparietal network, aPFC, medial temporal lobe and white matter hypointensities. We have also made our data freely available to others (https://osf.io/m5gcp).

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