Procrastination and Dementia

Cormac Monaghan[1,2], Rafael de Andrade Moral[3], and Joanna McHugh Power[2]

[1]Hamilton Institute, Maynooth University, Ireland

[2]Department of Psychology, Maynooth University, Ireland

[3]Department of Mathematics and Statistics, Maynooth University, Ireland

Author Note

Cormac Monaghan https://orcid.org/https://orcid.org/0000-0001-9012-3060

Rafael de Andrade Moral https://orcid.org/https://orcid.org/0000-0002-0875-3563

Joanna McHugh Power https://orcid.org/https://orcid.org/0000-0002-7387-3107

Correspondence concerning this article should be addressed to Cormac Monaghan, Email: cormacmonaghan@protonmail.com

Procrastination and Dementia

Dementia is a syndrome characterized initially by the progressive and typically irreversible decline of cognitive function, leading to memory loss, impaired reasoning, and difficulties with activities of daily living (Prince et al., 2013; **?**). It arises from various conditions, including Alzheimer's disease, vascular dementia, and Lewy body dementia (Cao et al., 2020). The global burden of dementia is substantial, with projections suggesting that the number of affected individuals will rise from 57.4 million in 2019 to 152.8 million by 2050 (Nichols et al., 2022). Given this projection, identifying and addressing modifiable risk factors is crucial to mitigate the growing prevalence difficulties with activities of daily living of dementia worldwide. In fact, targeting these factors could prevent up to 50% of dementia cases worldwide (Livingston et al., 2024). The *Lancet* Commission highlights 14 key modifiable risks, emphasizing that dementia risk can be reduced at any age. Nevertheless, age remains the strongest non-modifiable risk factor, accounting for the largest proportion of dementia cases.

Mild cognitive impairment (MCI) is a condition characterized by cognitive changes — such as memory lapses or difficulty making decisions — that exceed typical age-related decline (Gauthier et al., 2006). Prior studies suggest that 46% of people with MCI transition to a dementia state within three years and 80% within six years (Cooper et al., 2015; Shigemizu et al., 2020; Tschanz et al., 2006). However, progression is neither uniform nor inevitable. Heterogeneity in outcomes underscores the importance of distinguishing between two classes of predictors: causal risk factors (e.g. smoking, hypertension, or diabetes) which may accelerate dementia onset and are potential targets for intervention (Livingston et al., 2024), and prodromal markers (e.g. memory loss, apathy, or struggles in daily living) that reflect early disease processes and may signal limited opportunities for prevention. While causal factors offer avenues for mitigating risk, prodromal features often emerge closer to irreversible pathological changes, highlighting the need for early detection and tailored interventions.

Apathy, defined as a significant loss of motivation, distinct from depression and cognitive impairment (Fresnais et al., 2023; Richard et al., 2012) is among the most prevalent

neuropsychiatric symptoms in both MCI and dementia (Donovan et al., 2014; Fresnais et al., 2023; Salem et al., 2023; **?**). Individuals with apathy exhibit reduced goal-directed behavior and a diminished desire to pursue rewards or pleasure (Fresnais et al., 2023). Importantly, apathy has been identified as a significant predictor for the transition from MCI to dementia (Ruthirakuhan et al., 2019; Salem et al., 2023; **?**). For instance, a meta-analysis by (**?**) found that apathy almost doubles the risk of transitioning to dementia. Additionally, levels of apathy measured using both the *"Neuropsychiatric Inventory Questionnaire"* (Kaufer et al., 2000) and *"Apathy Evaluation Scale"* (Marin et al., 1991) have shown to be correlated with higher levels of amyloid burden and neurofibrillary tangles in individuals with dementia, suggesting a potential connection to underlying neuropathology (Donovan et al., 2014; Marshall et al., 2013).

While apathy primarily reflects a lack of motivation, procrastination involves a delay in action despite an intention to complete such action (Steel, 2007). Although both behaviours indicate disruptions in goal-directed activity, they differ in their phenomenology: apathy is marked by diminished drive or emotional engagement, whereas procrastination typically involves intact intention but impaired follow-through. These differences suggest that procrastination may capture aspects of executive dysfunction and emotional regulation not fully accounted for by apathy alone.

Both behaviours have been linked with impaired executive function, particularly in goal-oriented behaviours, decision-making, and self-regulation (Gustavson et al., 2015; Kawagoe et al., 2017; Steel, 2010), domains which are frequently compromised in individuals with MCI and dementia (Kirova et al., 2015; Stopford et al., 2012). While apathy has been well-established as a prodromal marker and risk factor for dementia progression (Ruthirakuhan et al., 2019; Salem et al., 2023; **?**), emerging evidence also suggests a potential link between procrastination and early cognitive decline. For instance, Fridén (2020) and S. Zhang et al. (2019) highlight that chronic procrastination is associated with dysfunction in both the dorsolateral and ventromedial prefrontal cortices, regions of the brain known to not only show early degeneration in dementia (Joseph et al., 2021), but also be linked to higher levels of apathy (Fahed & Steffens, 2021; S. Zhang et al., 2019).

Furthermore, procrastination may exacerbate existing cognitive decline by reinforcing patterns of inaction and passivity. Individuals who chronically delay tasks may inadvertently engage in fewer cognitively stimulating activities, such as physical activity, problem-solving, decision-making, and goal-setting. Such activities are known to build cognitive resilience and reduce dementia risk (Chowdhary et al., 2022; Livingston et al., 2024). By limiting engagement in such activities, procrastination may contribute to a self-reinforcing cycle of cognitive disengagement, potentially accelerating decline over time.

Given these behavioural and neuro-cognitive parallels with apathy, it is worth exploring whether chronic procrastination could serve as an early behavioural marker for cognitive impairment, or even a risk factor for dementia, especially in older adults. By examining procrastination alongside apathy, we may uncover a broader behavioural phenotype indicative of early neuropathological changes. Crucially, procrastination may identify individuals with motivational or executive dysfunction who do not yet meet the threshold for apathy, offering a potentially valuable and earlier behavioural marker for neurodegenerative risk.

Moreover, since age remains the strongest predictor of MCI and dementia, it is important to consider how behavioural predictors such as procrastination operate across the lifespan. Many established modifiable risk factors such as, hypertension, hearing loss, smoking, and social isolation, exert age-dependent effects, with certain factors carrying more weight at midlife than in late life (Livingston et al., 2024; **?**). In light of this, we sought to explore whether procrastination also demonstrates an age-dependent risk profile. Understanding how procrastination interacts with age may help clarify its role in the aetiology of cognitive decline and identify windows of opportunity for targeted intervention.

Although current research on procrastination in relation to dementia is non-existent, this possible association warrants exploration. Identifying procrastination as a potential risk factor could expand the scope of early interventions aimed at preventing or slowing the progression of dementia. As such, the purpose of this study was to (a) assess group differences in procrastination levels across the normal cognition, MCI, and dementia groups, and b) test the hypotheses that

higher levels of procrastination would be associated with an increased probability of transitioning from normal cognitive function or MCI to dementia.

Method

Data and study population

Analyses were conducted using a secondary data source; a multi-wave prospective cohort study called the Health and Retirement Study (HRS; (Fisher & Ryan, 2018), which tracks the health, economic, and social well-being of over 18,000 American adults primarily aged > 50. The HRS is managed by the Institute for Social Research at the University of Michigan, with data collected every two years. Initial data collection of a participant is conducted through a face-to-face interview, with follow-up biennial interviews conducted either by phone or face-to-face. The average re-interview response rate ranges from 68.8% to 92.3% (Health and Retirement Study, 2017). At the time of writing, fifteen years of HRS data are currently archived.

For this study, we focused on four waves of HRS data (from 2016 to 2022). Specifically our study sample consisted of respondents who participated in an experimental module assessing procrastination during the 2020 wave. These experimental modules, administered at the end of the core HRS interview, consist of concise questionnaires designed to explore new topics or supplement existing core survey data. Each respondent receives only one experimental module, with sample sizes for each module constituting approximately 10% of the core sample. As a result, while the core HRS sample includes approximately 18,000 respondents, our initial sample of interest consisted of 1,368 respondents. We excluded respondents with missing cognitive assessment data for any wave ($n = 419$), those under 60 years of age ($n = 398$), and those with complete missing values across the procrastination measure ($n = 2$). This resulted in a final analytic sample of 549 respondents.

Measures

Outcome: Cognitive Function and Cognitive Category

Cognitive function in the HRS is assessed using a series of tests adapted from the Telephone Interview for Cognitive Status (TICS; (Brandt et al., 1988; Fong et al., 2009). These tests include an immediate and delayed 10-noun free recall test, a serial seven subtraction test, and a backward count from 20 test. Based on these assessments, Crimmins et al. (2011) developed both a 27-point cognitive scale and validated cut-off points to assess and classify cognitive status. Using these points, respondents who scored $12 - 27$ were classified as having normal cognition, $7 - 11$ as having MCI, and $0 - 6$ as having dementia.

Predictor: Procrastination

Procrastination was measured using the Pure Procrastination Scale (Steel, 2010), which consists of 12 items rated on a Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). The total procrastination score ranges from 12 to 60, with higher scores indicating greater procrastination. The Pure Procrastination Scale conducted in 2020 (wave 3) had a Cronbach's α score of 0.92 in this sample, indicating high internal consistency. An example of a question from the scale includes "I delay making decisions until it's too late".

Confounders

To account for potential confounding, we controlled for variables withestablished associations with cognitive function and procrastination. These included measures of of age, sex, educational attainment, and depressive symptoms (Abner et al., 2012; Freedman & Cornman, 2024; Monaghan et al., 2024; Yu et al., 2013). Educationalattainment was classified into three categories: no formal education, GED (General Educational Development)/high school diploma, and college/further education. Depressive symptoms were measured using an eight-item version of the Center for Epidemiological Studies Depression (CES-D) scale (Briggs et al., 2018), with scores ranging from 0 to 8, demonstrating good internal consistency ($\alpha = 0.60$).

Data Analysis

All data analysis was carried out in R (R Core Team, 2025). Within the final dataset, missing data accounted for only 0.3%, all of which was contained within the educational attainment variable. To model transitions in cognitive states over time, we employed a first order discrete-time Markov model, a class of stochastic processes that satisfy the Markov property (Y. F. Zhang et al., 2010), which can be formally expressed as:

$$P(X_{t+1} = j | X_t = i, X_{t-1} = i_{t-1}, \ldots X_0 = i_0) = P(X_{t+1} = j | X_t = i)$$

This property asserts that the probability of transitioning to a future state X_{t+1} depends only on the current state X_t, and not on the full history of preceding states.

Unlike continuous-time models, discrete-time Markov models are not readily supported by a dedicated R package for deriving transition probabilities. Therefore, we implemented the model manually using multinomial logistic regression from the `nnet` package (Venables & Ripley, 2002). This approach estimates the log-odds of transitioning to each non-reference state as a linear function of covariates, relative to a chosen reference category. For a system with K cognitive states (with state K as the reference), the model takes the form:

$$log\left(\frac{P(Y = j|x)}{P(Y = k|x)}\right) = \alpha + \beta_j^T x \qquad \text{for } j = 1, \ldots K-1$$

From these equations, the predicted transition probabilities for the non-reference states $j = 1, \ldots, K-1$ are derived as:

$$P(Y = j|x) = \frac{e^{\alpha_j + \beta_j^T x}}{1 + \sum_{k=1}^{k-1} e^{\alpha_k + \beta_k^T x}}$$

and for the reference state K as:

$$P(Y = k|x) = \frac{1}{1 + \sum_{k=1}^{k-1} e^{\alpha_k + \beta_j^T x}}$$

Results

As mentioned, our initial sample of interest consisted of 1, 368 respondents. However, after excluding 819 respondents our final analytic sample comprised 549 respondents with the following age distribution: 60 - 70 (n = 186), 71 - 80 (n = 203), 81 - 90 (n = 142), and 90+ (n = 18). Descriptive statistics for the full sample, as well as stratified by cognitive status (normal cognition, MCI, and dementia), are presented in Table [table:1]. Additionally, Table 1 presents both the frequencies and estimated transition probabilities of cognitive status changes across waves. Specifically, it captures unconditional transitions between wave one and two (first transition), wave two and three (second transition), and wave three and four (third transition), yielding a total of 1,647 observed transitions over time.

Note. Descriptives for continuous and categorical variables are represented using means ± standard

* deviations and percentages and frequencies respectively. NC = Normal cognition; MCI = Mild

* cognitive impairment; GED = General Educational Development.

Note. NC = Normal cognition; MCI = Mild cognitive impairment

Initially, a Kruskal-Wallis test was conducted to examine differences in procrastination scores (measured in 2020) across three cognitive status groups after Levene's test indicated violation of homogeneity of variance (p = 0.039). The analysis revealed a statistically significant effect of cognitive status, ($\chi^2(2) = 17.54, p < .001$), indicating that procrastination levels differed significantly between at least two of the groups. Post-hoc analysis using a pairwise Wilcoxon rank-sum test with a Benjamini-Hochberg correction showed that participants with normal cognition ($M = 27.7, SD = 11.7$) reported significantly lower levels of procrastination than those with both MCI ($M = 32.1; SD = 11.3; p = 0.004$) and dementia ($M = 36.2; SD = 14.8; p = 0.005$). No significance difference was found between those with MCI and dementia (p = 0.334). Figure 1 displays the distribution of procrastination scores across groups, with both boxplots and dotplots showing median values and individual data points.

Significance bars indicate the pairwise differences described above.

Group differences in 2020 procrastination scores according to cognitive status.

Markov analysis

Results from the discrete-time Markov analysis, showed that all covariates significantly influenced the likelihood of transitioning between cognitive states (see Table 34). Notably, procrastination interacted significantly with age to affect two key transitions: increasing the likelihood of transitioning from normal cognition to MCI (OR = 1.001; $p < 0.001$) and decreasing the likelihood of reverting from MCI to normal cognition (OR = 0.999; $p < 0.001$).

In addition women were significantly less likely than men to transition from both normal cognition to dementia (OR = 0.068; $p < 0.001$) and from MCI to dementia (OR = 0.70; $p < 0.001$). Educational attainment was another strong predictor of cognitive transitions. Individuals with a GED were less likely to transition from normal cognition to either MCI (OR = 0.49; $p < 0.001$) or dementia (OR = 0.29; $p < 0.001$) and from MCI to dementia (OR = 0.64; $p < 0.001$). They were also more likely to back transition from MCI to normal cognition (OR = 2.18; $p < 0.001$).

Furthermore, those with a college level education or higher demonstrated a significantly reduced likelihood of transitioning from normal cognition to either MCI (OR = 0.32; $p < 0.001$) or dementia (OR = 0.07; $p < 0.001$) and from MCI to dementia (OR = 0.22; $p < 0.001$). They were also more likely to back transition from MCI to normal cognition (OR = 3.13; $p < 0.001$).

Finally, higher levels of depressive symptomatology were associated with an increased likelihood of transitioning from normal cognition to both MCI (OR = 1.13; $p = 0.002$) and dementia (OR = 1.18; $p = 0.046$) and and a decreased likelihood of transitioning from MCI back to normal cognition (OR = 0.89; $p = 0.001$).

Finally, Figure á2 presents the predicted transition probabilities across varying levels of age and procrastination. These estimates illustrate how the interaction between age and procrastination influences the likelihood of progressing between cognitive states over time. Notably, while transition probabilities remain relatively stable at very low levels of

procrastination, substantial shifts emerge as both age and procrastination increase. In particular, older individuals with higher procrastination scores show an elevated probability of cognitive decline and a reduced likelihood of cognitive recovery, highlighting the compounded risk posed by these two variables in later life.

Note. Each curve represents a different age profile, ranging from the minimum (62 years) to the maximum (97 years) of the dataset. Specific ages of interest (70, 80, and 90) are highlighted.

Predicted transition probabilities by procrastination and age.

References

Abner, E. L., Kryscio, R. J., Cooper, G. E., Fardo, D. W., Jicha, G. A., Mendiondo, M. S., Van Eldik, L. J., Wan, L., & Schmitt, F. A. (2012). Mild cognitive impairment: Statistical models of transition using longitudinal clinical data. *International Journal of Alzheimer's Disease*, *2012*(1), 291920. https://doi.org/10.1155/2012/291920

Brandt, J., Spencer, M., & Folstein, Marshal. (1988). The telephone interview for cognitive status. *Neuropsychiatry Neuropsychol Behav Neurol*, *1*(2), 111–117.

Briggs, R., Carey, D., O'Halloran, A. M., Kenny, R. A., & Kennelly, S. P. (2018). Validation of the 8-item centre for epidemiological studies depression scale in a cohort of community-dwelling older people: Data from the irish longitudinal study on ageing (TILDA). *European Geriatric Medicine*, *9*, 121–126. https://doi.org/10.1007/s41999-017-0016-0

Cao, Q., Tan, C.-C., Xu, W., Hu, H., Cao, X.-P., Dong, Q., & Lan Tan, and J.-T. Y. (2020). The prevalence of dementia: A systematic review and meta-analysis. *Journal of Alzheimer's Disease*, *73*(3), 1157–1166. https://doi.org/10.3233/JAD-191092

Chowdhary, N., Barbui, C., Anstey, K. J., Kivipelto, M., Barbera, M., Peters, R., Zheng, L., Kulmala, J., Stephen, R., Ferri, C. P., et al. (2022). Reducing the risk of cognitive decline and dementia: WHO recommendations. *Frontiers in Neurology*, *12*, 765584. https://doi.org/10.3389/fneur.2021.765584

Cooper, C., Sommerlad, A., Lyketsos, C. G., & Livingston, G. (2015). Modifiable predictors of

dementia in mild cognitive impairment: A systematic review and meta-analysis. *American Journal of Psychiatry*, *172*(4), 323–334. https://doi.org/10.1176/appi.ajp.2014.14070878

Crimmins, E. M., Kim, J. K., Langa, K. M., & Weir, D. R. (2011). Assessment of cognition using surveys and neuropsychological assessment: The health and retirement study and the aging, demographics, and memory study. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *66*(suppl_1), i162–i171. https://doi.org/10.1093/geronb/gbr048

Donovan, N. J., Wadsworth, L. P., Lorius, N., Locascio, J. J., Rentz, D. M., Johnson, K. A., Sperling, R. A., Marshall, G. A., Initiative, A. D. N., et al. (2014). Regional cortical thinning predicts worsening apathy and hallucinations across the alzheimer disease spectrum. *The American Journal of Geriatric Psychiatry*, *22*(11), 1168–1179. https://doi.org/10.1016/j.jagp.2013.03.006

Fahed, M., & Steffens, D. C. (2021). Apathy: Neurobiology, assessment and treatment. *Clinical Psychopharmacology and Neuroscience*, *19*(2), 181. https://doi.org/10.9758/cpn.2021.19.2.181

Fisher, G. G., & Ryan, L. H. (2018). Overview of the health and retirement study and introduction to the special issue. *Work, Aging and Retirement*, *4*(1), 1–9. https://doi.org/10.1093/workar/wax032

Fong, T. G., Fearing, M. A., Jones, R. N., Shi, P., Marcantonio, E. R., Rudolph, J. L., Yang, F. M., Kiely, D. K., & Inouye, S. K. (2009). Telephone interview for cognitive status: Creating a crosswalk with the mini-mental state examination. *Alzheimer's & Dementia*, *5*(6), 492–497. https://doi.org/10.1016/j.jalz.2009.02.007

Freedman, V. A., & Cornman, J. C. (2024). Dementia prevalence, incidence, and mortality trends among US adults ages 72 and older, 2011–2021. *The Journals of Gerontology, Series A: Biological Sciences and Medical Sciences*, *79*(Supplement_1), S22–S31. https://doi.org/10.1093/gerona/glae105

Fresnais, D., Humble, M. B., Bejerot, S., Meehan, A. D., & Fure, B. (2023). Apathy as a predictor for conversion from mild cognitive impairment to dementia: A systematic review and

meta-analysis of longitudinal studies. *Journal of Geriatric Psychiatry and Neurology*, *36*(1), 3–17. https://doi.org/10.1177/08919887221093361

Fridén, I. (2020). *Procrastination as a form of self-regulation failure: A review of the cognitive and neural underpinnings*.

Gauthier, S., Reisberg, B., Zaudig, M., Petersen, R. C., Ritchie, K., Broich, K., Belleville, S., Brodaty, H., Bennett, D., Chertkow, H., Cummings, J. L., de Leon, M., Feldman, H., Ganguli, M., Hampel, H., Scheltens, P., Tierney, M. C., Whitehouse, P., & Winblad, B. (2006). Mild cognitive impairment. *The Lancet*, *367*(9518), 1262–1270. https://doi.org/10.1016/S0140-6736(06)68542-5

Gustavson, D. E., Miyake, A., Hewitt, J. K., & Friedman, N. P. (2015). Understanding the cognitive and genetic underpinnings of procrastination: Evidence for shared genetic influences with goal management and executive function abilities. *Journal of Experimental Psychology: General*, *144*(6), 1063. https://doi.org/10.1037/xge0000110

Health and Retirement Study. (2017). *Sample sizes and response rates*. https://hrs.isr.umich.edu/sites/default/files/biblio/ResponseRates_2017.pdf

Joseph, S., Knezevic, D., Zomorrodi, R., Blumberger, D. M., Daskalakis, Z. J., Mulsant, B. H., Pollock, B. G., Voineskos, A., Wang, W., Rajji, T. K., et al. (2021). Dorsolateral prefrontal cortex excitability abnormalities in alzheimer's dementia: Findings from transcranial magnetic stimulation and electroencephalography study. *International Journal of Psychophysiology*, *169*, 55–62. https://doi.org/10.1016/j.ijpsycho.2021.08.008

Kaufer, D. I., Cummings, J. L., Ketchel, P., Smith, V., MacMillan, A., Shelley, T., Lopez, O. L., & DeKosky, S. T. (2000). Validation of the NPI-q, a brief clinical form of the neuropsychiatric inventory. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *12*(2), 233–239. https://doi.org/10.1176/jnp.12.2.233

Kawagoe, T., Onoda, K., & Yamaguchi, S. (2017). Apathy and executive function in healthy elderly—resting state fMRI study. *Frontiers in Aging Neuroscience*, *9*, 124. https://doi.org/10.3389/fnagi.2017.00124

Kirova, A. M., Bays, R. B., & Lagalwar, S. (2015). Working memory and executive function decline across normal aging, mild cognitive impairment, and alzheimer's disease. *BioMed Research International*, *2015*(1), 748212. https://doi.org/10.1155/2015/748212

Livingston, G., Huntley, J., Liu, K. Y., Costafreda, S. G., Selbæk, G., Alladi, S., Ames, D., Banerjee, S., Burns, A., Brayne, C., Fox, N. C., Ferri, C. P., Gitlin, L. N., Howard, R., Kales, H. C., Kivimäki, M., Larson, E. B., Nakasujja, N., Rockwood, K., … Mukadam, N. (2024). Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. *The Lancet*, *404*(10452), 572–628. https://doi.org/10.1016/S0140-6736(24)01296-0

Marin, R. S., Biedrzycki, R. C., & Firinciogullari, S. (1991). Reliability and validity of the apathy evaluation scale. *Psychiatry Research*, *38*(2), 143–162. https://doi.org/10.1016/0165-1781(91)90040-V

Marshall, G. A., Donovan, N. J., Lorius, N., Gidicsin, C. M., Maye, J., Pepin, L. C., Becker, J. A., Amariglio, R. E., Rentz, D. M., Sperling, R. A., et al. (2013). Apathy is associated with increased amyloid burden in mild cognitive impairment. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *25*(4), 302–307. https://doi.org/10.1176/appi.neuropsych.12060156

Monaghan, C., Avila-Palencia, I., Han, S. D., & McHugh Power, J. (2024). Procrastination, depressive symptomatology, and loneliness in later life. *Aging & Mental Health*, 1–8. https://doi.org/10.1080/13607863.2024.2345781

Nichols, E., Steinmetz, J. D., Vollset, S. E., Fukutaki, K., Chalek, J., Abd-Allah, F., Abdoli, A., Abualhasan, A., Abu-Gharbieh, E., Akram, T. T., et al. (2022). Estimation of the global prevalence of dementia in 2019 and forecasted prevalence in 2050: An analysis for the global burden of disease study 2019. *The Lancet Public Health*, *7*(2), e105–e125. https://doi.org/10.1016/S2468-2667(21)00249-8

Prince, M., Bryce, R., Albanese, E., Wimo, A., Ribeiro, W., & Ferri, C. P. (2013). The global prevalence of dementia: A systematic review and meta analysis. *Alzheimer's & Dementia*, *9*(1), 63–75. https://doi.org/10.1016/j.jalz.2012.11.007

R Core Team. (2025). *R: A language and environment for statistical computing*. R Foundation for

Statistical Computing. https://www.R-project.org/

Richard, E., Schmand, B., Eikelenboom, P., Yang, S., Ligthart, S., Moll van Charante, E., Van Gool, W., & Initiative, A. D. N. (2012). Symptoms of apathy are associated with progression from mild cognitive impairment to alzheimer's disease in non-depressed subjects. *Dementia and Geriatric Cognitive Disorders*, *33*(2-3), 204–209. https://doi.org/10.1159/000338239

Ruthirakuhan, M., Herrmann, N., Vieira, D., Gallagher, D., & Lanctôt, K. L. (2019). The roles of apathy and depression in predicting alzheimer disease: A longitudinal analysis in older adults with mild cognitive impairment. *The American Journal of Geriatric Psychiatry*, *27*(8), 873–882. https://doi.org/10.1016/j.jagp.2019.02.003

Salem, H., Suchting, R., Gonzales, M. M., Seshadri, S., & Teixeira, A. L. (2023). Apathy as a predictor of conversion from mild cognitive impairment to alzheimer's disease: A texas alzheimer's research and care consortium (TARCC) cohort-based analysis. *Journal of Alzheimer's Disease*, *92*(1), 129–139. https://doi.org/10.3233/JAD-220826

Shigemizu, D., Akiyama, S., Higaki, S., Sugimoto, T., Sakurai, T., Boroevich, K. A., Sharma, A., Tsunoda, T., Ochiya, T., Niida, S., et al. (2020). Prognosis prediction model for conversion from mild cognitive impairment to alzheimer's disease created by integrative analysis of multi-omics data. *Alzheimer's Research & Therapy*, *12*, 1–12. https://doi.org/10.1186/s13195-020-00716-0

Steel, P. (2007). The nature of procrastination: A meta-analytic and theoretical review of quintessential self-regulatory failure. *Psychological Bulletin*, *3*(1). https://doi.org/10.1037/0033-2909.133.1.65

Steel, P. (2010). Arousal, avoidant and decisional procrastinators: Do they exist? *Personality and Individual Differences*, *48*(8), 926–934. https://doi.org/10.1016/j.paid.2010.02.025

Stopford, C. L., Thompson, J. C., Neary, D., Richardson, A. M., & Snowden, J. S. (2012). Working memory, attention, and executive function in alzheimer's disease and frontotemporal dementia. *Cortex*, *48*(4), 429–446. https://doi.org/10.1016/j.cortex.2010.12.002

Tschanz, J., Welsh-Bohmer, K., Lyketsos, C., Corcoran, C., Green, R. C., Hayden, K., Norton, M.

C., Zandi, P., Toone, L., West, N., et al. (2006). Conversion to dementia from mild cognitive disorder: The cache county study. *Neurology*, *67*(2), 229–234. https://doi.org/10.1212/01.wnl.0000224748.48011.84

Venables, W. N., & Ripley, B. D. (2002). *Modern applied statistics with s-PLUS* (Fourth). Springer. https://www.stats.ox.ac.uk/pub/MASS4/

Yu, H., Yang, S., Gao, J., Zhou, L., Liang, R., & Qu, C. (2013). Multi-state markov model in outcome of mild cognitive impairments among community elderly residents in mainland china. *International Psychogeriatrics*, *25*(5), 797–804. https://doi.org/10.1017/S1041610212002220

Zhang, S., Liu, P., & Feng, T. (2019). To do it now or later: The cognitive mechanisms and neural substrates underlying procrastination. *Wiley Interdisciplinary Reviews: Cognitive Science*, *10*(4), e1492. https://doi.org/10.1002/wcs.1492

Zhang, Y. F., Zhang, Q. F., & Yu, R. H. (2010). Markov property of markov chains and its test. *2010 International Conference on Machine Learning and Cybernetics*, *4*, 1864–1867. https://doi.org/10.1109/ICMLC.2010.5580952

Table 1

Frequencies (and transition probabilities) of cognitive status transitions from HRS waves.

2016			
	NC	MCI	Dementia
NC	(0.75)	(0.07)	(0.001)
MCI	(0.09)	(0.06)	(0.01)
Dementia	(0)	(0)	(0.02)
2018			
	NC	MCI	Dementia
NC	(0.75)	(0.09)	(0)
MCI	(0.07)	(0.03)	(0.02)
Dementia	(0)	(0)	(0.03)
2020			
	NC	MCI	Dementia
NC	(0.68)	(0.13)	(0.02)
MCI	(0.05)	(0.05)	(0.01)
Dementia	(0)	(0)	(0.05)

	Intercept	Gender	Age	GED
NC - MCI	0.114*** (0.113 - 0.114)	0.941 (0.690 - 1.284)	1.008** (1.002 - 1.014)	0.446*** (0.360 - 0.551)
NC - Dementia	0.009*** (0.009 - 0.009)	0.669*** (0.662 - 0.687)	1.023*** (1.010 - 1.035)	0.295*** (0.289 - 0.302)
MCI - NC	9.008*** (8.986 - 9.030)	1.061 (0.789 - 1.482)	0.992** (0.986 - 0.997)	2.46*** (1.829 - 2.759)
MCI - Dementia	0.13*** (0.129 - 0.130)	0.708*** (0.678 - 0.740)	1.008 (0.995 - 1.021)	0.646*** (0.630 - 0.662)
	Further Education	Depression	Procrastination	Procrastination x Age
NC - MCI	0.310*** (0.264 - 0.363)	1.147*** (1.067 - 1.233)	0.921*** (0.882 - 0.962)	1.001*** (1.001 - 1.002)
NC - Dementia	0.068*** (0.068 - 0.068)	1.150 (0.979 - 1.353)	0.981 (0.888 - 1.084)	1.000 (0.888 - 1.084)
MCI - NC	3.240*** (2.789 - 3.762)	0.872*** (0.811 - 0.937)	1.085*** (1.039 - 1.134)	0.999*** (0.998 - 0.999)
MCI - Dementia	0.214*** (0.212 - 0.216)	1.004 (0.852 - 1.184)	1.005 (0.952 - 1.168)	0.999 (0.998 - 1.001)