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RESEARCH ARTICLE

Procrastination as a marker of cognitive decline: Evidence from longitudinal transitions in the older adult population

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Abstract

INTRODUCTION: Cognitive decline is a global health concern, making the identification of early, modifiable risk factors essential. While apathy is a recognized prodromal marker, procrastination may also signal early executive dysfunction.

METHODS: We used longitudinal secondary data from the United States Health and Retirement Study among adults aged 60+ ($n = 549$; $\bar{x} = 69.70$; $s = 7.58$). Cognitive function, procrastination, depression, and a proxy measure of apathy were assessed. Transitions between normative cognitive function, mild cognitive impairment (MCI), and dementia were modeled using a discrete-time first-order Markov model.

RESULTS: Procrastination scores were higher among individuals with MCI or dementia than those with normative cognitive function. Procrastination also interacted with age, disproportionately increasing the risk of decline in the oldest participants.

DISCUSSION: Procrastination was associated with cognitive impairment and predicted transitions to MCI, suggesting it may serve as both an early behavioral marker and compounding risk factor.

KEYWORDS
aging, apathy, dementia risk, executive dysfunction, mild cognitive impairment, procrastination

Highlights

- Procrastination predicts cognitive decline in older adults.
- Effects remain after accounting for apathy.
- Longitudinal study links everyday behavior to dementia risk.
- Procrastination may be a potentially modifiable early behavioral marker.

1 | BACKGROUND

Dementia involves progressive cognitive decline that impairs daily functioning,[1,2] with cases projected to rise to 152.8 million by 2050.[3] Identifying and addressing modifiable risk factors is crucial to mitigate this projected rise in prevalence. Two particularly vulnerable groups are older adults and those with mild cognitive impairment (MCI), of whom up to 80% progress to dementia within 6 years.[4–6]

The Lancet Commission on Dementia identified 14 causal risk factors including smoking, hypertension, and diabetes, which account for ≈ 45% of dementia cases worldwide.[7] Prodromal markers, on the other hand, reflect early disease processes and appear closer to the onset of the disease.[8] One such prodromal marker is apathy, or the significant loss of motivation, as distinct from depression and cognitive impairment.[9,10] Apathy is prevalent in both MCI and dementia subgroups[9,11–13] and is a robust predictor of progression from MCI to dementia.[12–14] While apathy has been well established as a prodromal marker for dementia progression, emerging evidence suggests that procrastination may also relate to early cognitive decline.

Although apathy and procrastination can appear similar in that both lead to reduced engagement in everyday activities, the underlying behavioral processes are distinct. Apathy reflects reduced internal drive and emotional engagement, impairing initiation of action.[9] In contrast, procrastination is a self-regulatory failure defined by the voluntary delay of an important and intended course of action despite expecting negative consequences.[15,16] This distinction suggests that individuals who procrastinate typically form intentions but fail to execute them in a timely manner. This failure can often be understood through an affective framework, in which delayed action may stem from the avoidance of negative emotions (i.e., stress or anxiety) associated with the task.[16] Furthermore, while also conceptualized as a stable trait linked to specific personality factors,[17] these factors can shift in later life and may interact with emerging cognitive or motivational changes to influence procrastination behavior in older adults.[18,19]

Both apathy and procrastination have been linked to dysfunction in prefrontal regions implicated in dementia.[20,21] Procrastination has been associated with alterations in brain circuitry supporting cognitive control and emotional regulation. Research has identified both structural and functional differences primarily in the prefrontal cortex (PFC) and parahippocampal cortex.[20] Specifically, high procrastination is associated with decreased gray matter volume in the dorsolateral PFC (dlPFC) and increased spontaneous activity in the ventromedial PFC (vmPFC). Additionally, functional connectivity studies indicate altered couplings between both the vmPFC and dlPFC, along with the vmPFC and frontal gyri.[20] Critically, these same networks are affected in neurodegenerative conditions, including Alzheimer's disease.[21]

These parallels raise the possibility that procrastination could serve as an early marker of cognitive impairment or even a modifiable risk factor. Similar to apathy, chronic procrastination may exacerbate decline by limiting engagement in cognitively stimulating activities such as physical exercise, problem solving, and goal setting, which build cognitive resilience and reduce dementia risk.[22,23] Reduced engagement may contribute to a cycle of cognitive disengagement, accelerating decline.

Importantly, procrastination is responsive to intervention through cognitive–behavioral strategies and self-regulation training.[24,25] Identifying procrastination in older adults may therefore offer a dual benefit: it highlights individuals with emerging motivational or executive dysfunction who may not yet meet the threshold for apathy, and it opens a window for targeted intervention at a stage when cognitive decline may still be preventable.

Because age remains the strongest predictor of MCI and dementia, it is important to consider how procrastination operates across the lifespan (i.e., its interaction with age). 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.[7,23] Understanding how procrastination interacts with age may help clarify its role in the etiology of cognitive decline and identify windows of opportunity for targeted intervention.

To our knowledge, no studies have directly examined procrastination as a predictor of cognitive decline or dementia progression. Accordingly, the present study aimed to (1) assess differences in procrastination levels across three groups: individuals with dementia, individuals with MCI, and individuals with neither dementia nor MCI, and (2) test whether higher procrastination scores predict transition from normative cognition function to dementia or MCI to dementia.

2 | METHODS

2.1 | Data and study population

We used data from the Health and Retirement Study (HRS),[26,27] a nationally representative longitudinal survey of US adults aged 50+, focusing on four waves of data from 2016 to 2022. Inclusion criteria for the analysis required participants to have completed the experimental module assessing procrastination during the 2020 wave. This identified an initial sample of 1368 respondents. We then applied the following exclusion criteria: respondents with missing cognitive assessment data for any wave ($n = 419$), those < 60 years of age (as cognitive symptoms typically occur around this age ; $n = 398$), and those with missing values across the procrastination measure ($n = 2$). The application of these exclusion criteria resulted in a final analytic sample of 549 respondents, of whom 61.57% ($n = 338$) were female. Further details on the HRS and experimental modules are provided in the supporting information.

RESEARCH IN CONTEXT

1. **Systematic review**: We reviewed PubMed and longitudinal cohort studies examining early behavioral markers of cognitive decline. While apathy is well documented, the role of procrastination has not been evaluated. Relevant studies linking executive dysfunction to everyday behaviors were identified and cited.
2. **Interpretation**: Our findings indicate that procrastination predicts cognitive decline independently of apathy. This suggests that procrastination may serve as an early behavioral marker of cognitive impairment and highlights a potentially modifiable target for interventions aimed at preserving cognitive health.
3. **Future directions**: Future research should investigate the mechanisms linking procrastination to cognitive decline, explore intervention strategies to reduce procrastination in older adults, and evaluate whether modifying procrastination behaviors can delay or prevent progression to dementia. Longitudinal replication in diverse populations is also warranted.

2.2 | Measures

2.2.1 | 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[28,29] (TICS). These tests include an immediate and delayed 10-noun free recall test (to assess episodic memory), a serial seven subtraction test (to assess working memory), and a backward count from 20 test (to assess mental processing speed). Based on these assessments, Crimmins et al.[30] 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 to 27 were classified as having normative cognitive function, 7 to 11 as having MCI, and 0 to 6 as having dementia.

2.2.2 | Predictor: procrastination

Procrastination was measured using the Pure Procrastination Scale[31] (PPS), which consists of 12 items rated on a Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). An example of a question from the scale includes "I delay making decisions until it's too late." Total procrastination scores range from 12 to 60, with higher scores indicating greater tendency to procrastinate. In the HRS, the PPS was administered only in wave 3 (2020) of the respective waves. As such, we use the wave 3 measure as both a retrospective and prospective proxy for procrastination scores assuming relative temporal stability. In this sample, the PSS had a Cronbach α score of 0.92, indicating high internal consistency.

2.2.3 | Covariate: apathy

While no direct measure of apathy exists within the HRS, we used two questions from the eight-item version of the Center for Epidemiological Studies Depression (CESD-8) scale[32] as proxies for apathy: "You felt that everything you did was an effort" and "You could not get going." Both items capture core features of apathy (behavioral and motivational disengagement) and, while not a comprehensive measure of apathy, provide a valid and pragmatic approximation for modeling purposes. Each item was measured on a binary "yes/no" scale with total scores ranging from 0 to 2.

2.2.4 | Covariate: depression

Depressive symptoms were included in our model based on their associations with both procrastination[15] and cognitive aging.[7] As mentioned, the HRS uses the CESD-8 as a measure of depressive symptomatology. The scale consists of 8 items that are scored as either 0 (no) or 1 (yes), with items between 4 and 6 being reversed scored. Because we used two items from this scale as proxies for apathy, we scored the remaining 6 items, giving a total score ranging from 0 to 6, with higher scores indicating higher levels of depressive symptoms. Across the waves, this 6-item scale showed reliable internal consistency ($\alpha\varepsilon$[0.81 – 0.83]). An example of a question from the scale includes "Much of the time during the past week, your sleep was restless."

2.2.5 | Covariates: demographics

To account for potential confounding, we controlled for demographic variables with established associations with both cognitive function and procrastination.[33] These included measures of age, sex, and education attainment.[7,34] Education attainment was classified into three categories: no formal education, GED (General Educational Development)/high school diploma, and college/further education.

2.3 | Data analysis

All data analysis was carried out in R.[35] We modelled changes in cognitive status across waves using a first-order discrete-time Markov model. Transitions among the three cognitive states were estimated using multinomial logistic regression via the nnet package.[36] Predicted transition probabilities were derived from the fitted multinomial models for each wave-to-wave interval. Full mathematical specifications and derivation of transition probabilities are provided in the supporting information.

TABLE 1 Baseline descriptive for the study sample and stratified by cognitive status category.

	Full sample ($n = 549$)	NC ($n = 452$)	MCI ($n = 86$)	Dementia ($n = 11$)
Age (years)	69.70 ± 7.58	69.70 ± 7.55	69.60 ± 7.55	70.00 ± 9.89
Sex				
Male	38.43% ($n = 211$)	39.82% ($n = 180$)	32.56% ($n = 28$)	27.27% ($n = 3$)
Female	61.57% ($n = 338$)	60.18% ($n = 272$)	67.44% ($n = 58$)	72.73 ($n = 8$)
Education				
No degree	16.24% ($n = 98$)	10.56% ($n = 47$)	39.53% ($n = 34$)	63.54% ($n = 7$)
GED	51.58% ($n = 279$)	51.91% ($n = 231$)	52.33% ($n = 45$)	27.27 ($n = 3$)
Further education	32.29% ($n = 175$)	37.53% ($n = 167$)	8.14% ($n = 7$)	9.09% ($n = 1$)
Apathy	0.37 ± 0.63	0.31 ± 0.59	0.65 ± 0.76	0.36 ± 0.51
Depression	0.98 ± 1.48	0.88 ± 1.41	1.41 ± 1.71	1.09 ± 1.87
Procrastination*	28.60 ± 12.00	27.70 ± 11.30	32.10 ± 13.80	39.00 ± 16.00

Note: Descriptives for continuous and categorical variables are represented using means ± standard deviations and percentages and frequencies respectively.
*Procrastination scores were collected in 2020 (Wave 3) and are presented here for descriptive comparison, although they were not measured at baseline.
Abbreviations: GED, General Educational Development; MCI, mild cognitive impairment; NC, normative cognitive function.

3 | RESULTS

Our final analytic sample comprised 549 respondents with the following age distribution: 60 to 70 (n = 186), 71 to 80 (n = 203), 81 to 90 (n = 142), and 90 + (n = 18). Descriptive statistics for the full sample, as well as data stratified by cognitive status (normative cognitive function, MCI, and dementia) are presented in Table 1. Both Figure 1 and Figure S1 in supporting information capture the unconditional transitions and transition probabilities (respectively) between wave one and two (first transition), wave two and three (second transition), and wave three and four (third transition), yielding a total of 1647 observed transitions over time.

3.1 | Cross-sectional differences

A Kruskal–Wallis test was conducted to examine differences in procrastination scores (measured in 2020) across three cognitive status groups, after the Levene test indicated violation of homogeneity of variance ($P = 0.039$). The analysis revealed a statistically significant effect of cognitive status ($\chi^2[2] = 17.54$, $P < 0.001$), indicating that procrastination scores 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 normative cognitive function (M = 27.7, standard deviation [SD] = 11.7) reported significantly lower procrastination scores 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 significant difference in scores was found between those with MCI and dementia ($P = 0.334$). Figure 2 displays the distribution of procrastination scores across groups, with significance bars indicating the pairwise differences described above.

3.2 | Markov analysis

Results from the discrete-time Markov analysis showed that all covariates significantly influenced the likelihood of transitioning between cognitive states (see Figure 3). Notably, procrastination interacted significantly with age to affect two key transitions: increasing the likelihood of transitioning from normative cognitive function to MCI (odds ratio [OR] = 1.001; $P < 0.001$) and decreasing the likelihood of reverting from MCI to normative cognitive function (OR = 0.999; $P < 0.001$). Women were significantly less likely than men to transition from both normative cognitive function to dementia (OR = 0.67; $P < 0.001$) and from MCI to dementia (OR = 0.71; $P < 0.001$).

For education attainment, individuals with a GED were less likely to transition from normative cognitive function to either MCI (OR = 0.46; $P < 0.001$) or dementia (OR = 0.29; $P < 0.001$) and from MCI to dementia (OR = 0.65; $P < 0.001$). They were also more likely to back transition from MCI to normative cognitive function (OR = 2.24; $P < 0.001$). Those with a college level education or higher demonstrated a significantly reduced likelihood of transitioning from normative cognitive function to either MCI (OR = 0.31; $P < 0.001$) 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 normative cognitive function (OR = 3.23; $P < 0.001$).

Higher levels of depression were associated with an increased likelihood of transitioning from normative cognitive function to MCI (OR = 1.14; $P = 0.013$) and a reduced likelihood of transitioning back from MCI to normal cognition (OR = 0.88; $P = 0.013$). Finally, higher levels of apathy were associated with an increased likelihood of transitioning from normative cognitive function to dementia (OR = 1.20; $P < 0.001$).

Figure 4 presents the predicted transition probabilities across varying levels of age and procrastination. These estimates illustrate how the

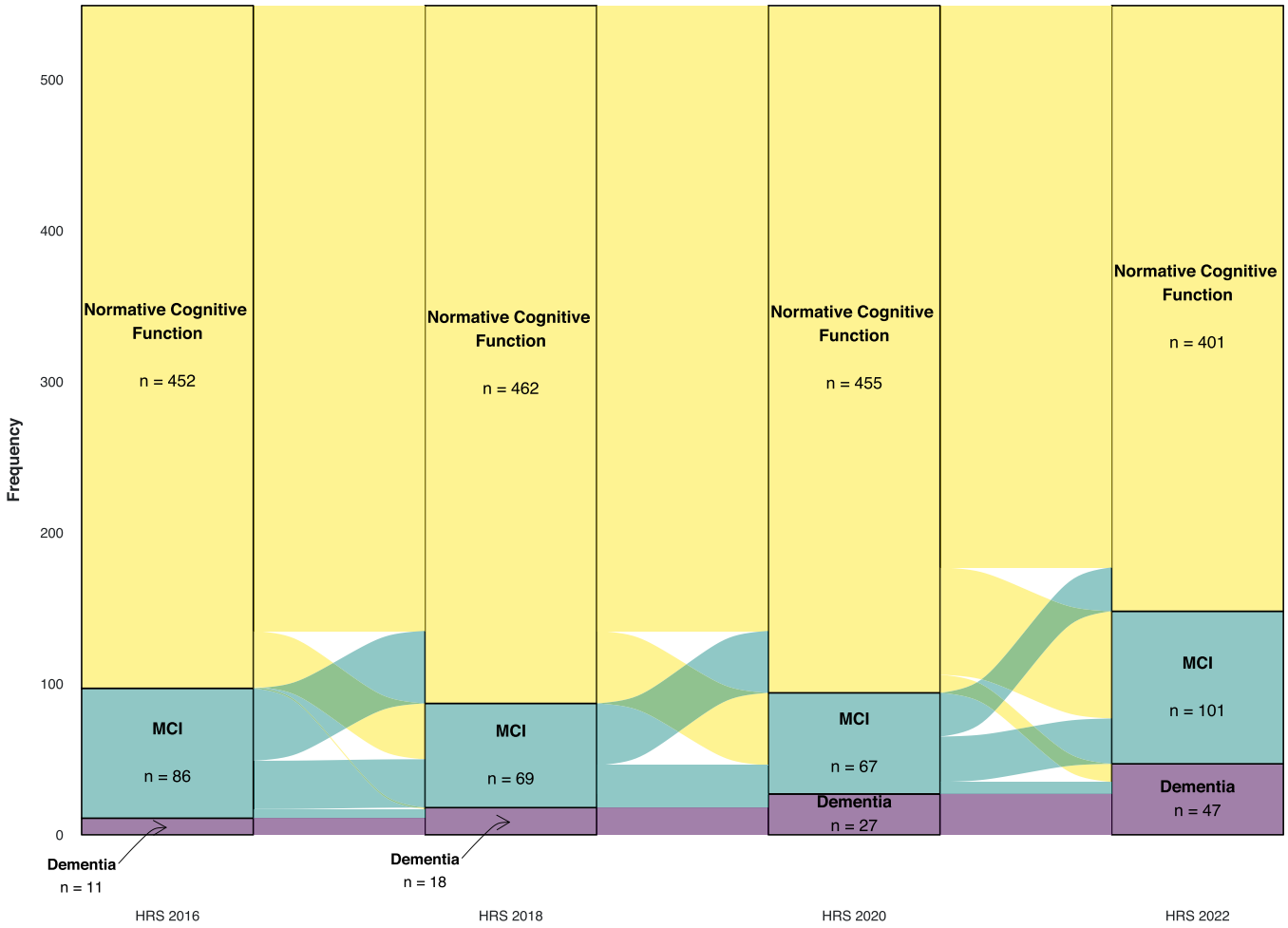

FIGURE 1 Frequency of cognitive status transitions between HRS waves. HRS, Health and Retirement Study; MCI, mild cognitive impairment.

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 transitioning from normative cognitive function to MCI and a reduced likelihood of transitioning back from MCI to normative cognitive function, highlighting the compounded risk posed by these two variables in later life.

4 | DISCUSSION

Dementia poses a growing global health burden, with modifiable risk factors and prodromal markers offering important targets for intervention.[7,23] While apathy is an established prodromal marker,[9,11–13] emerging evidence suggests that procrastination may also signal early cognitive dysfunction.[20,21] Our analysis revealed significant group differences in procrastination, with individuals experiencing cognitive impairment, both MCI and dementia, reporting higher procrastination scores than those with normative cognitive function. These findings support the hypothesis that procrastination may be associated with cognitive decline and aligns with emerging evidence linking procrastination to cognitive dysfunction.[20,21] Interestingly, while both MCI and dementia groups exhibited elevated procrastination scores, no significant difference emerged between these two groups. This suggests that increases in procrastination may occur relatively early in the neurodegenerative process, potentially preceding or paralleling the emergence of more overt cognitive symptoms.

However, it should be noted that the number of participants classified as having MCI ($n = 67$) or dementia ($n = 27$) was relatively small compared to those with normative cognitive function ($n = 455$). This imbalance in group sizes may have limited the sensitivity of our comparisons and raises the possibility that subtle differences between MCI and dementia groups were obscured by statistical power constraints. In addition, our analytic sample contained a higher proportion of women than men (61.57% female), reflecting the demographic composition of the available HRS respondents who met eligibility criteria. Although this imbalance was not the result of a sampling decision, it may influence the generalizability of our findings, as women in older cohorts

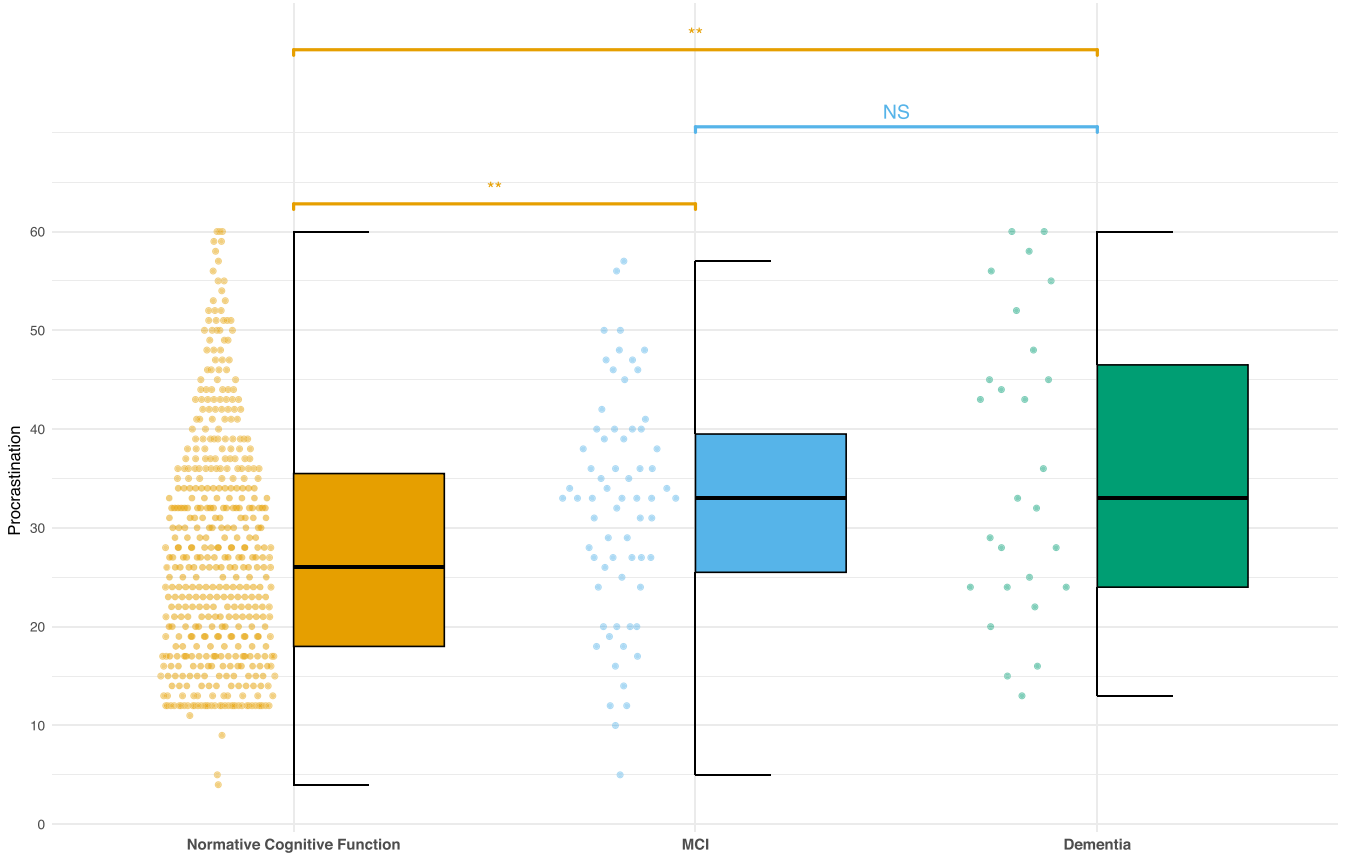

FIGURE 2 Group differences in 2020 procrastination scores according to cognitive status. MCI, mild cognitive impairment.

differ from men in both dementia risk profiles and health-seeking behaviors.[37] Future research should aim to replicate these findings in more evenly distributed samples to better determine whether these results are consistent across men and women and whether procrastination continues to increase across progressive stages of impairment or plateaus after early decline.

Beyond these cross-sectional differences, our discrete-time Markov analysis offered insight into how procrastination, alongside other demographic and psychological factors, influences the likelihood of transitioning between cognitive states over time. Notably, procrastination emerged as a dynamic predictor of cognitive change, interacting with age to significantly increase the odds of transitioning from normative cognitive function to MCI, while simultaneously decreasing the odds of moving from MCI back to normative cognitive function.

These findings imply that procrastination may serve dual roles, both as a marker of early cognitive dysfunction and, potentially, as a behavioral impediment to moving from MCI state to the normative cognitive state. One plausible pathway involves the reinforcement of maladaptive behaviors such as reduced cognitive engagement or reduced participation in cognitively protective activities, such as physical exercise, social interaction, or medical adherence.[22,38–40] In parallel, procrastination has been associated with chronic stress and elevated cortisol levels,[41] which may accelerate hippocampal atrophy, amyloid beta plaque deposition, and brain inflammation,[42,43] further undermining cognitive resilience. These behavioral and biological mechanisms align with self-regulation theories suggesting that procrastination reflects executive dysfunction,[24] an early feature of neurodegenerative progression.[44] At the same time, these results align with both the affective and trait-based perspectives on procrastination. Higher procrastination in individuals with cognitive impairment may partially reflect affective dysregulation (e.g., anxiety or stress-related avoidance)[16] or age-related shifts in stable behavioral tendencies that accompany neurodegenerative change.[18] While our study cannot differentiate between these frameworks, our results highlight that procrastination is a multifaceted construct encompassing motivational, affective, and self-regulatory dimensions.

The observed interaction with age (see Figure 4) suggests that the influence of procrastination on cognitive trajectories may grow stronger with advancing age, a period during which neuro-plasticity diminishes, and behavioral risk factors exert greater influence.[7,23] This effect was particularly pronounced among the oldest participants. As illustrated in Figure 4, the effect of procrastination is relatively modest for younger-older adults (those age 70). However, the slope of these transitions steepens considerably for individuals aged 80 and especially for those age 90. These findings indicate that procrastination is increasingly associated with a higher risk of cognitive decline and a lower likelihood of improvement in cognitive status in late old age.

This pattern underscores the possibility that procrastination functions as a compounding risk factor in older adulthood, particularly among the oldest individuals, by both reflecting emerging cognitive

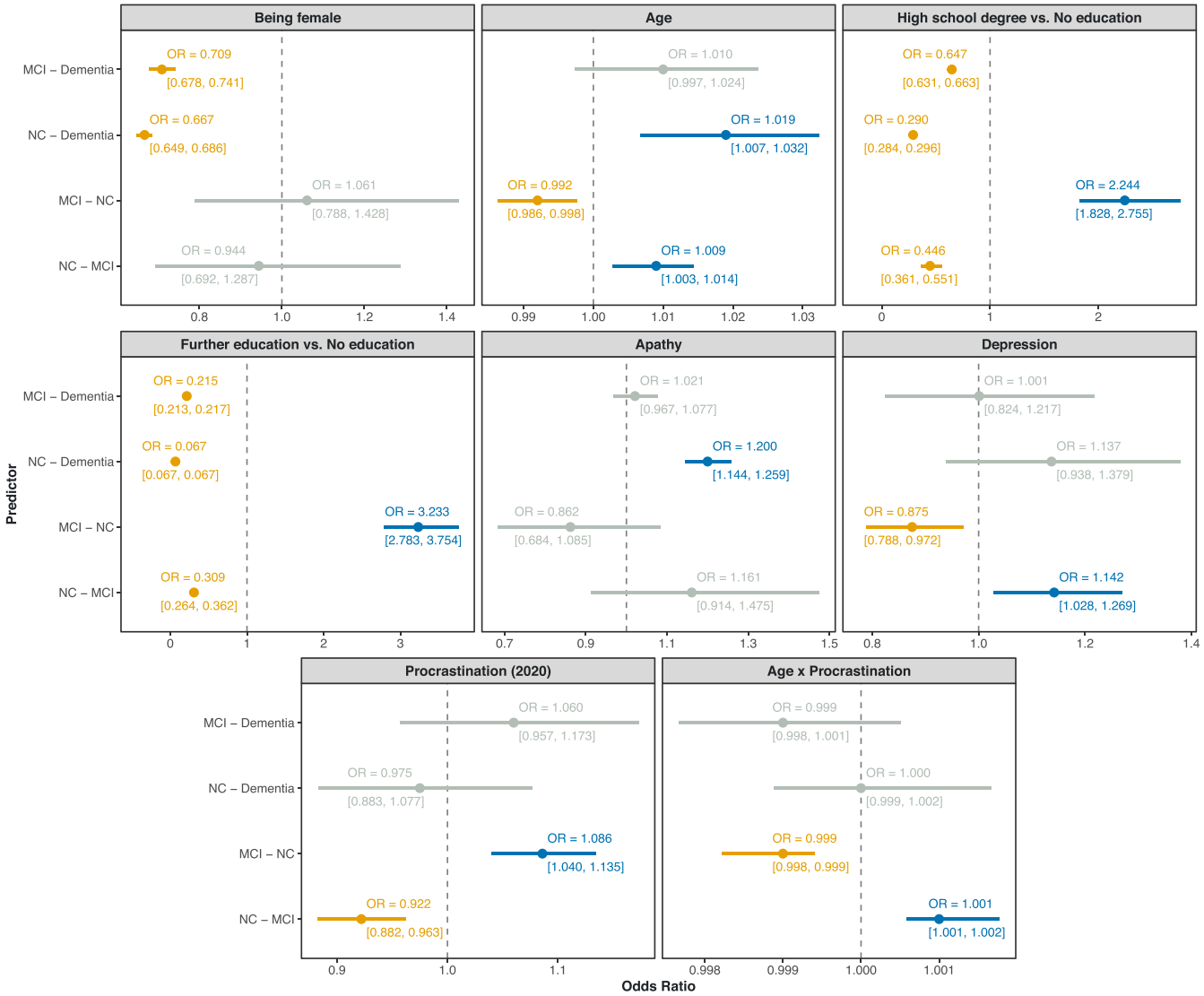

FIGURE 3 Odds ratios (95% confidence intervals) of transitioning from one cognitive state to another calculated using generalized logit models. Note: Odds ratios significantly different from 1 at a 5% significance rate are presented in blue (positive) or orange (negative), otherwise they are presented in gray. MCI, mild cognitive impairment; NC, normative cognitive function; OR, odds ratio.

difficulties and potentially accelerating their progression. In younger-old adults, cognitive reserve and compensatory mechanisms[45] may buffer against the impact of poor self-regulation. However, as individuals reach more advanced ages, these protective systems weaken.[46] Consequently, maladaptive behavioral tendencies like procrastination can exert a disproportionate toll on cognitive function. These findings underscore the potential value of addressing behavioral regulation and self-management in older adults as part of broader dementia risk-reduction strategies, particularly for those of a much older age.

By revealing a significant interaction between age and procrastination, this study highlights the importance of broadening dementia risk models to incorporate dynamic lifestyle and psychological variables. Procrastination, as both a modifiable and measurable behavioral tendency,[25] represents a promising target for low-cost, non-invasive interventions aimed at enhancing cognitive resilience in older populations. Tracking self-regulatory behavioral tendencies such as procrastination may offer an early warning system for emerging cognitive risk, opening avenues for preventative action before irreversible decline takes hold.

4.1 | Limitations

Despite the insights offered by this study, several limitations should be considered. Most notably, although our longitudinal Markov modelling approach captured changes in cognitive status, a key constraint was the measurement of procrastination at only a single time point (2020). As a result, we assumed temporal stability in procrastination scores across time and used this measure as both a retrospective and prospective proxy for procrastination scores. However, this precluded analysis of within-person changes in this behavior over time and whether such changes affect cognitive transitions. It should be noted that this

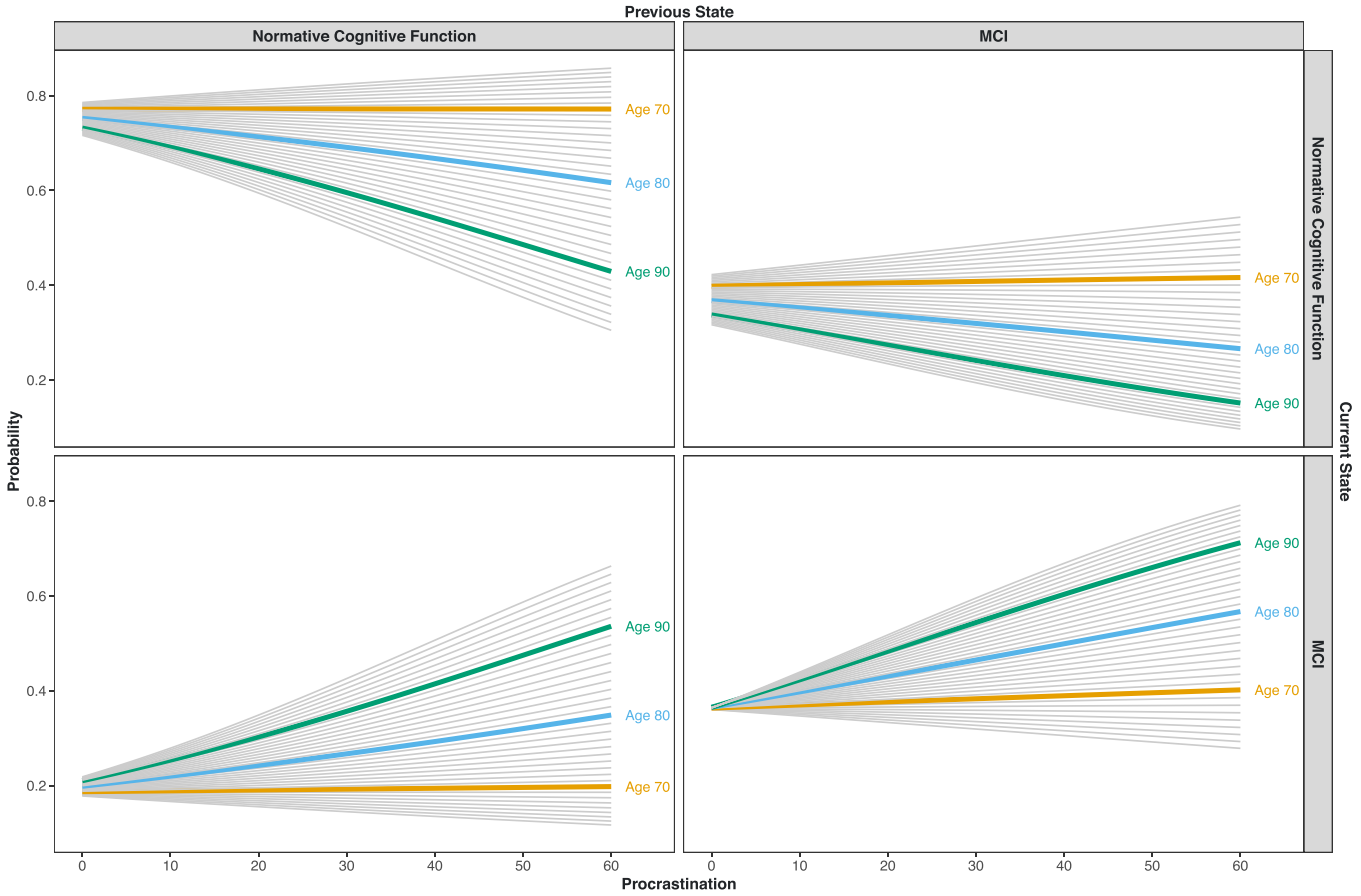

FIGURE 4 Predicted transition probabilities by procrastination and age. Note. Each curve represents a different age profile, ranging from the minimum (62 years) to the maximum (97 years) of the dataset. Specific ages of interest (70, 80, and 90) are highlighted. MCI, mild cognitive impairment.

limitation is inherent to the HRS dataset rather than a methodological oversight.[27] Future studies should prioritize repeated assessments of procrastination to determine whether increases in procrastination precede, accompany, or follow cognitive decline.

A further consideration concerns the classification of cognitive status. As our analyses rely on the HRS, we use its established operational definitions of MCI and dementia,[30] which are based on threshold scores on the TICS.[28,29] Developed as a telephone-based analogue of the Mini-Mental State Examination, the TICS is a widely adopted and well-validated cognitive screener.[29] However, like all brief screening measures, TICS-based classifications may not fully align with more detailed clinical assessments, raising the possibility of diagnostic misclassification. Future research using more comprehensive cognitive batteries or clinical diagnoses would help validate and refine the patterns observed here.

Additionally, negative emotional states (e.g., anxiety) are important psychological factors linked to both cognitive aging and procrastination.[7,16] However, we were unable to robustly adjust for anxiety in our models due to substantial systematic missingness in the HRS anxiety measure (see Figures S2 and S3 in supporting information). Moreover, our use of a proxy measure of apathy may not have fully captured the multidimensional nature of apathy. Additionally, it meant that only six of the original eight CESD-8 items were used for scoring depression, possibly introducing measurement error. Future research should incorporate more validated apathy assessments to strengthen the behavioral inferences drawn.

Finally, while the models adjusted for several key demographic and psychological covariates, unmeasured confounding (undiagnosed medical conditions, medication use, or sleep quality) could also have influenced cognitive outcomes. Future research should seek to replicate and extend these findings using more clinically diverse samples.

5 | CONCLUSIONS

In summary, this study offers preliminary evidence that procrastination may function as an early behavioral tendency marker of cognitive decline, particularly in older age. Individuals with MCI and dementia reported higher procrastination scores, and longitudinal modelling revealed that procrastination, especially when coupled with advancing age, was predictive of increased cognitive decline. These findings underscore the importance of everyday self-regulatory behaviors in dementia risk and resilience. As a modifiable and measurable construct, procrastination holds promise as a target for early detection and

preventative intervention strategies aimed at sustaining cognitive health in aging populations.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest. Author disclosures are available in the supporting information.

CONSENT STATEMENT

Consent was not required as all analyses were conducted on a publicly available secondary source dataset.

DATA AVAILABILITY STATEMENT

Data used in this study were obtained from the HRS public survey database (https://hrsdata.isr.umich.edu/data-products/public-survey-data). Upon making a HRS account all waves of public data from 1992 to 2022 are available for download. In addition, the R code that supports this study can be found on GitHub at https://github.com/C-Monaghan/crisp-clam.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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