# UC San Diego UC San Diego Previously Published Works

# Title

Parental dementia and subjective memory impairment in the health and retirement study

# Permalink

https://escholarship.org/uc/item/2r62j5g5

## **Journal** Aging & Mental Health, 26(5)

**ISSN** 1360-7863

# Authors

Bell, Tyler R Hill, Nikki L Bhargava, Sakshi <u>et al.</u>

# **Publication Date**

2022-05-04

# DOI

10.1080/13607863.2021.1910790

Peer reviewed



# **HHS Public Access**

Author manuscript Aging Ment Health. Author manuscript; available in PMC 2023 May 01.

Published in final edited form as:

Aging Ment Health. 2022 May; 26(5): 992–1000. doi:10.1080/13607863.2021.1910790.

# Parental Dementia and Subjective Memory Impairment in the Health and Retirement Study

Tyler R. Bell, Ph.D.<sup>1</sup>, Nikki L. Hill, Ph.D.<sup>2</sup>, Sakshi Bhargava, Ph.D.<sup>2</sup>, Jacqueline Mogle, Ph.D.<sup>2</sup>

<sup>1</sup>Department of Psychiatry, University of California San Diego, La Jolla, California, 92122

<sup>2</sup>College of Nursing, The Pennsylvania State University, University Park, PA

## Abstract

**Objectives:** To examine relationships between subjective memory impairment (SMI) and parental dementia among in older adults while considering the interactive influence of depressive symptoms, ethnicity, and race.

**Method:** The sample was drawn from the Health and Retirement Study, a nationally representative longitudinal study of aging (n = 3,809;  $M_{age} = 66.09$ ; SD = 1.88; 84.20% White; 12.23% Black; 7.88% Hispanic). Biennial assessments included two measures of SMI (current memory problems and perceived memory decline), depressive symptoms, and parental dementia, over periods of up to sixteen years. Multilevel modeling analyses examined longitudinal relationships between parental dementia and SMI and whether depressive symptoms, ethnicity, and race interactively influenced this association.

**Results:** Results showed that when older adults reported parental dementia, they were more likely to report a decline in memory in the past two years. They also reported poorer current memory problems, especially when they experienced increased depressive symptoms. Associations of parental dementia were consistent across ethnicity and race.

**Conclusions:** Results demonstrate the importance of considering parental dementia as a factor that may contribute to SMI in older adults.

#### Keywords

older adults; psychosocial factors; depressive symptoms; self-reported memory

## Introduction

Over 25% of older adults report subjective memory impairment (SMI), i.e., the belief that memory is poor or declining (Hertzog, Hülür, Gerstorf, & Pearman, 2018). SMI has been associated with accelerated cognitive decline and transitions into impairment. For example, studies demonstrate a two to four-fold increased risk of dementia for individuals who endorse SMI despite normal cognitive function (Benito-León, Mitchell, Vega, &

Correspondence concerning this article should be addressed to Dr. Tyler Bell, Department of Psychiatry, University of California San Diego, 3252 Holiday Court, La Jolla, California, 92122. trbell@health.ucsd.edu.

Bermejo-Pareja, 2010). SMI also leads to increased affective symptoms, lower activity participation, and lower wellbeing (Montejo, Montenegro, Fernandez, & Maestu, 2012; Wion, Hill, DePasquale, Mogle, & Bratlee-Whitaker, 2019), possibly explaining some changes in cognition. Understanding what leads to SMI for many might help explain increased dementia risk and other adverse outcomes in later life.

One factor of interest is the role of history of familial dementia, as it might confer greater genetic risk for dementia in oneself. Hypothetically, such individuals would report SMI as they experience subtle memory decline due to early neurodegeneration from dementia (e.g., Hausmann et al., 2014). However, non-genetic factors might exert interactive influences, such as emotional reactions to learning about parental dementia bolstering SMI reporting. For example, having a close relative with dementia might make one feel more vulnerable to memory decline or dysfunction, affecting perceptions of their own memory ability (Kinzer & Suhr, 2016). Also, observing a parental with dementia might make an individual more conscious of dire consequences, including functional decline, emotional distress, morbidity, and death related to memory loss – hence one would be more likely to report SMI due to greater concern. This would be aligned with studies showing that familial dementia is related to greater perceived threat of dementia, subsequent worries about their own memory, and help-seeking for memory problems (Ramakers et al., 2009; Suhr & Kinkela et al., 2007). Parental dementia might be a salient factor affecting SMI reports due to genetic and interactive influences.

Generally, studies on familial dementia and SMI differences are mixed. Initially, Rue et al. (1996) found that persons with a relative with Alzheimer's disease (AD) (n = 61) reported greater SMI reports than people without a relative with AD (n = 41). However, other studies found no differences. When looking at people with first-degree relative with AD (n = 25) or not (n = 26), McPherson et al. (1995) found comparable SMI reports. This was also shown by Heun et al. (2003) who found no significant differences in SMI between older adults who had first-degree relatives with AD (n = 146) or not (n = 136). Such null differences were also shown between children who had a parent with AD (n = 25) and did not (n = 25) (Cutler & Hodgson, 2001). Because small-sample designs might contribute to mixed findings, however, larger studies might provide more certain findings via adequate power.

As one of the first large scale studies, Tsai, Green, Benke, Silliman, and Farrer (2006) found that older adults with a first-degree relative with AD (n = 1,499) were nearly twice as likely to report SMI than those with spouses with AD (OR = 1.9). However, it is possible that asking about parents might be even more salient to examine as they might provide greater genetic predisposition for memory problems than other first-degree family members (e.g., siblings). Still, studies with greater sample size and a focus on more proximate family members might be limited by cross-sectional design. Specifically, the influence of parental dementia might be greatest on SMI reports after learning of parental dementia, especially due to interactive influences of emotional reactions on genetic risk. As such, we aimed to conduct a large longitudinal data analysis examine what happens to SMI once one reports parental dementia as a time-varying predictor.

One interactive influence may change in depressive symptoms after learning of parental dementia. Studies show depressive symptoms to be related to higher SMI reports, both within- and between-persons (Hill et al., 2019; Hülür, Hertzog, Pearman, Ram, & Gerstorf, 2014). This may suggest the role of attribution error: Described by the Hopelessness Theory of Depression (Abramson, Metalsky, & Alloy, 1989), depressive symptoms increase the perception that adverse events like dementia are highly occurrent and unavoidable. Hence, when a parent develops dementia, these individuals may be more likely to report SMI due to greater concerns about dementia development. Understanding this interactive influence on genetic risk might help more accurately identify at-risk groups to decelerate dementia progression. Specifically, increased depressive symptoms might need to be considered when determining levels of SMI involved with parental dementia.

Finally, ethnic and racial identities are other important interactive influences to consider for generalizability. Although scant, some studies suggest that memory beliefs are socioculturally informed, which could affect how one interprets their own abilities after learning about parental dementia. For example, Hispanic and Black adults have been reported as more likely to endorse dementia as a normal process of aging than non-Hispanic White adults (Cahill et al., 2015; Gray et al., 2009). This might also explain why Hispanic older adults report significantly more cases of SMI than Non-Hispanic older adults (Harwood et al., 1998), as they might see memory lapses as more indicative of dementia and therefore more concerning. It could also explain why Black older adults report slightly more SMI than White older adults (Taylor et al., 2018). As such, differential tendencies to report SMI might attenuate the association between dementia and SMI reports. Our study will thus examine associations between parental dementia and SMI across ethnic and racial identities.

Using a large random sample of older adults in the U.S., the current study investigated the influence of parental dementia on reports of SMI. Furthermore, we aimed to address previous limitations in the literature by: 1) examining relationships between longitudinal reports of parental dementia and SMI ; 2) considering how the association might differ by SMI question type; 3) exploring the potential interactive influence of depressive symptoms, and, 4) exploring the interactive influence of ethnic and racial identities. Regarding specific hypothesis, we predicted that parental dementia would be related to greater current memory problems and a higher likelihood to report perceived memory decline. We also hypothesized that due to greater beliefs about the inescapable nature of dementia, depressive symptoms, Hispanic ethnicity, and Black racial identity would exacerbate the association between parental dementia and SMI reports.

#### Methods

Participants were selected from four cohorts of the Health and Retirement Study (HRS), a nationally representative longitudinal study of aging aimed to understand contextual factors that impact older adult well-being. Led by the Institute for Social Research at the University of Michigan (Health and Retirement Study, n.d.; Sonnega et al., 2014), HRS conducted stratified random sampling to derive a nationally representative group of adults ages 50 years and over. Cohorts sampled included people born before 1924 (Cohort 1), 1924 to 1930 (Cohort 2), 1931 to 1942 (Cohort 3), and 1942 to 1947 (Cohort 4). More extensive

Page 4

information about the protocol, instruments, and sampling strategy is provided from the University of Michigan's Institute for Social Research (Wallace & Herzog, 1995). HRS collected data biennially starting in 1992. For the current study, data were included from 1998 to 2014, when questions on the parental history of dementia were introduced. For this study, data were restructured such that the wave at which participant data was first available was assigned as wave 1, i.e., baseline. Overall, this baseline included a potential sample of 4,316 participants who completed self-reports of SMI (i.e., did not use a proxy) and provided information on parental dementia. For our analysis, we additionally removed individuals with a diagnosis of AD or probable cognitive impairment (n = 495; estimated from performing two standard deviations below normal in two or more cognitive tests; see Kasper & Freedman, 2018) and individuals for whom imputation was performed (n = 12). This left a final analytical sample of 3,809 older (aged 65 and above, see Supplemental Table 1 for wave-specific sample sizes) adults (58.86% females;  $M_{age} = 66.09$ ; SD = 1.88, range from 65 to 84) from diverse ethnic (92.12% non-Hispanic, 7.88% Hispanic) and racial backgrounds (84.20% White; 12.23% Black; 3.57% Other). Sex and race proportions align with recent estimates in the United States (U.S. Census Bureau, 2018). Average years of education was 13.01 (SD = 2.85), ranging from 0 to 17 years. Average income was \$77,463 (SD = \$203,551), ranging from 0 to \$7,307,860. Approximately 2% (n = 66) of participants came from HRS's Cohort 1, 3.49% (n = 133) from Cohort 2, 68.13% (n = 2595) from Cohort 3, and 26.65% (n = 1015) from Cohort 4. This analytical sample provided up to nine biennial waves (i.e., 16 years of follow-up;  $M_{\text{waves}} = 3.19$ , SD = 2.00, range = 1 to 9). Supplementary Table 1 shows sample characteristics across waves.

#### Measures

**SMI reports.**—SMI was measured using reports on two questions: current memory problems and perceived memory decline. Current memory problems assessed with the item "How would you rate your memory at the present time?" Participants responded on a five-point Likert-type scale: 1 = excellent, 2 = very good, 3 = good, 4 = fair, 5 = poor. Perceived memory decline was assessed with the item "Compared with (previous wave/two years ago), would you say your memory is better now, about the same, or worse than it was then?" Participants responded on a three-point Likert-type scale: 1 = better, 2 = same, 3 = worse. Due to low variability (only 3% reported "better"), responses were recoded into a binary variable where 0 = better/same and 1 = worse.

**Parental dementia.**—Self-reported history of parental dementia was used as a measure of parental dementia. From years 1998 to 2008, it was assessed with the following item: "Has a doctor ever said that your mother/father has a memory-related disease?" Participants provided dichotomous responses (0 = No; 1 = Yes). From year 2010 (wave 10) to 2014 (wave 12), the following item was used: "Has a doctor ever told your mother/father that she/he has Alzheimer's disease?" or "Has a doctor ever told your mother/father that she/he has dementia, senility or any other serious memory impairment?" Participants provided dichotomous responses (0 = No; 1 = Yes) for both questions. Responses from these two questions were combined to create two variables: "mother dementia" and "father dementia." Next, sensitivity analyses showed that questions on mother/father dementia asked before 2010 and after 2010 produced similar responses and therefore were combined to use as

a longitudinal measure of mother and father dementia. Next, responses from mother and father dementia variables were combined to measure parental dementia. This new variable was assigned a score of 1 if the participant reported that either their mother or father had dementia, and 0 if neither had dementia. The missing data were imputed such that once a score of 1 was assigned to at any given wave, the score remained 1 for all subsequent waves. A score of 0 was assigned to waves prior to the wave in which participants first reported a score of 1 for parental dementia.

**Demographic factors.**—At each assessment, participants provided demographic information on age (years), education level (years), and household income (US \$). These demographics were included as continuous variables in later analyses.

**Depressive symptoms.**—Depressive symptoms were measured using the Center of Epidemiological Studies scale (CES-D, Radloff, 1977). Participants were asked whether they experienced eight depressive symptoms in the past week including "was depressed," "everything was an effort," "sleep was restless," "felt lonely," "felt sad," and "could not get going." Two other reverse-coded items were "was happy" and "enjoyed life." Participants responded as either "no" (0 = not experiencing symptom) or "yes" (1 = experiencing symptom). A composite score was created, and scores ranged from 0 to 8 with higher scores indicating greater depressive symptoms. Reliability at baseline was acceptable (Cronbach's a = .79).

#### **Data Analysis**

Prior to examining longitudinal associations, descriptive analyses were performed to examine any mean differences in the proportion of persons with parental dementia and perceived memory decline by sex, race, education level, income level, and cohort. Next, mean differences in current memory problems by participants' sex, race, education level, and income level were examined. Additionally, intercorrelations were examined among key study variables.

Next, multilevel modeling (MLM) analyses were performed to examine longitudinal associations between exposure to parental dementia and SMI symptoms (i.e., current memory rating and perceived memory decline). Specifically, MLM examined the time-varying associations of parental dementia with SMI symptoms across waves. Additionally, we examined whether depressive symptoms interactively influenced this association as they changed at each wave (i.e., within-person effects), after adjusting for baseline differences (i.e., between-person effects). Ethnicity and race were also assessed as potential interactive influences. Current memory rating was treated as an ordinal outcome and modeled using SAS proc glimmix using a multinomial distribution with a cumulative logit link. Perceived memory decline was treated as a dichotomous categorical variable and was modeled with SAS proc glimmix using a binary distribution with a logit link.

First, two unconditional (i.e., not adjusting for variables of interest) multilevel models examined the trajectories of current memory problems and perceived memory decline over time (see Table 1, Models 1). Time was the only predictor included in these models. Then, two conditional (i.e., adjusting for variables of interest) multilevel models examined the

longitudinal time-varying associations of parental dementia with SMI reports and whether depressive symptoms, ethnicity, and race interactively influenced (i.e., moderated) these associations (see Table 1, Models 2). Both models included parental dementia as a predictor. Depressive symptoms, ethnicity, and race were included as interaction terms with parental dementia. Nonsignificant interactions were trimmed from the final models and main effects were retained as covariates. Statistical codes for these models are provided online (https://github.com/trbellucsd/HRSFamilyHistory).

Regarding variable coding, parental dementia was included as a raw time-varying predictor, where 0 indicated no parental dementia reported and 1 indicates parental dementia reported. To look at changes in depressive symptoms (especially when parental dementia occurs), we calculated within-person effects of depressive symptoms by centering time-varying values by the person's value at their first wave as done in prior work (e.g., Stawski et al., 2013). We adjust for between-person effects of depressive symptoms by including centering baseline values around the grand-mean of the sample at the first wave. Ethnicity was coded as 0 for non-Hispanic (reference group) and 1 for Hispanic. Race was coded as 1 for White (reference group), 2 for Black, and 3 for Other. Time (waves), sex (1 = male, 2 = female; male chosen as reference group), age, education years, income (\$), and cohort (4-level categorical variable; Cohort 3 was selected as reference group due to large membership) were included as covariates. Multilevel model equations are provided in Table 1.

#### Results

#### **Preliminary Analysis**

Preliminary analyses showed that approximately 31.42% of participants reported having a parent with dementia at their first wave, and 14.20% of those who did not have a parent with dementia at the first wave reported having a parent with dementia at a later wave (see Supplementary Table 1 for wave-specific details). At baseline, some demographic differences were observed in current memory problems and parental dementia (see Table 2). Intercorrelations among key study variables showed significant but weak associations of depressive symptoms with current memory problems (r=.20, p < .001) and perceived memory decline (r=.16, p < .001). Additionally, current memory problems were significantly associated with perceived memory decline (r=.30, p < .001). See Table 3 for intercorrelations among the study variables.

#### **Substantive Analysis**

**Unconditional multilevel models.**—Modeling trajectories of SMI reports, over time, older adults were more likely to report higher levels of current memory problems (OR = 1.16; 95% CI[1.14, 1.19]) and to perceive a decline in their memory (OR = 1.12; 95% CI[1.09, 1.15]).

#### Conditional multilevel models.

<u>Current memory problems.</u>: After accounting for covariates, there was a significant interaction between parental dementia and within-person changes in depressive symptoms on current memory problems. Specifically, parental dementia with increased depressive

symptoms related to greater likelihood of reporting worse levels of current memory problems (OR = 1.10, 95% CI[1.03, 1.17]; see Table 4, Model 1). In Figure 1, this interaction is plotted by showing the association of parental dementia when people show mean and ±1 *SD* change in depressive symptoms within person. The mean change in depressive symptoms at any wave was an increase of 1.32 units; this corresponded to increased odds of reporting a higher level of current memory problems when reporting parental dementia (OR = 1.22, 95% CI[1.01 to 1.48]). When depressive symptoms decreased by .42 units at any wave (-1 *SD*), individuals did not show a significant change in the odds of reporting higher levels of current memory problems when reporting parental dementia (OR = 0.96, 95% CI[0.81, 1.13]). When depressive symptoms increased by 3.06 units at any wave (+1 *SD*), individuals were more likely to report higher levels of current memory problems when reporting parental dementia (OR = 1.56, 95% CI[1.24 to 1.98]). Meanwhile, parental dementia associations with current memory problems did not significantly differ by Hispanic (p = .197) or racial identity (ps > .72).

Regarding covariates, greater depressive symptoms between persons related to greater likelihood of reporting worse levels of current memory problems (OR = 1.44, 95% CI[1.37, 1.51]). Hispanic older adults were more likely to report higher levels of current memory problems (OR = 1.66, 95% CI[1.18, 2.32]). Black older adults were more likely to report higher levels of current memory problems than White older adults (OR = 2.26, 95% CI[1.74, 2.94]). Higher education related less likelihood of reporting higher levels of current memory problems (OR = .73, 95% CI[.69, .76]).

**Perceived memory decline.:** Results showed that parental dementia was related to perceived memory decline (OR = 1.21, 95% CI[1.03, 1.42]; see Table 4, Model 2, and Figure 1), such that once participants reported having a parent with dementia, they were more likely to perceive a memory decline. Depressive symptoms did not interactively influence this association (p = .420). Parental dementia associations were not interactively influenced by Hispanic (p = .956) or racial identity (p = .656) either.

Regarding covariates, higher depressive symptoms related to higher likelihood of report perceived memory decline between persons (OR = 1.52, 95% CI[1.44, 1.60]) and within persons over time (OR = 1.30, 95% CI[1.25, 1.35]). Hispanic older adults were less likely to report perceived memory decline than non-Hispanic older adults (OR = 0.59, 95% CI[0.42, 0.83]). Higher education also related to increased likelihood of reporting perceived memory decline (OR = 1.07, 95% CI[1.02, 1.13])

#### Discussion

The current study examined whether parental dementia impacted SMI reports in cognitively intact older adults (specifically, current memory problems and perceived memory decline) and the interactive influences of depressive symptoms, ethnicity, and race. Generally, parental dementia was positively associated with SMI over time. Below we describe these associations by SMI report type while discussing interactive influences.

As our major finding, we found a positive association between parental dementia and perceived memory decline, independent of depressive symptoms and consistent across ethnic and racial backgrounds. Such a finding extends previous work in other large datasets (Mogle et al., 2020; Tsai et al., 2006) and might suggest general familial dementia effect for two reasons: First, because dementia is a partly inherited disease (Gatz et al., 1997; Green et al., 2002), individuals with familial dementia might be more likely to notice subclinical manifestations of dementia in themselves (discerned as perceived memory decline). Second, familial dementia might also lead to increased symptom awareness. After becoming aware of their increased inherited risk, signs of memory decline might be more salient due to worries about dementia (Cutler & Hodgson, 2001; Hodgson & Cutler, 2003; Suhr & Kinkela, 2007). Identifying genetic and non-genetic (e.g., symptom awareness) influences from parental dementia to SMI emerges as logical next steps for the field.

We also found a positive association between parental dementia and current memory problems, significant across ethnic and racial identities. However, in contrast to perceived memory decline, greater depressive symptoms increased reporting of current memory problems after parental dementia. In fact, the effect of parental dementia on current memory problems appeared nil when no change in depressive symptoms occurred (i.e., no significant main effect). This suggests an interactive influence such that emotional reactions to parental dementia bolsters SMI reporting above genetic risk. As described by the Hopelessness Theory of Depression (Abramson et al., 1989), depressive symptoms may prime one to perceive adverse events as inevitable, which might increase their concern and SMI reporting - especially as memory lapses are seen as signs of inevitable dementia. A lack of an interactive influence on perceived memory decline might have resulted from discordant timeframes. Specifically, current memory problems and depressive symptoms asked about recent experiences, whereas perceived memory decline asked individuals to think about experiences across two years. It could also be that perceived memory decline maps more onto a direct genetic risk due to this broader timeframe referenced, lowering an interactive influence. Differential effects of timeframes queried should be an improtant consideration in future study designs.

Counter to hypotheses, associations between dementia and SMI reports were similar across ethnic and racial identities, i.e., no interactive influences. Still, we did note significant ethnic and racial differences on SMI reports. Hispanic older adults rated greater current memory problems than non-Hispanic persons, aligned with previous works (Harwood et al., 1998). However, they did report fewer cases of perceived memory decline when non-Hispanic persons, suggesting potential differences by SMI item used. Harwood et al. (1998) used a multi-item measure of memory complaints, which might align more with current memory problems rather than perceived memory decline. Black older adults also reported higher current memory problems than White older adults, consistent with previous (Taylor et al., 2018) and recent studies (Hill et al., 2019). This suggests that while cultural and racial experiences might differentially shape evaluation of one's memory, parental dementia seems to exert a common effect on SMI reports. Indeed, this would be consistent with a shared genetic risk. It also suggests that ethnicity and race exert different effect depending on the SMI item types.

Future studies should parcel out genetic and interactive influences on the association between parental dementia and SMI reports through leveraging available genetic data or strategic recruitment methodology (i.e., twin design). Investigations might also benefit from assessing associations with multiple sources of dementia, including people with various genetic and non-genetic links (e.g., sibling and cousin relative versus spouses and friends). If the importance of interactive non-genetic factors on SMI arises alongside genetic attributions, then understanding psychological reactions after dementia might improve the specificity of SMI for identifying early cognitive changes in people with predispositions for dementia.

As a note, this work adds previous studies from HRS while delivering additional avenues for future inquiry as data collection ensues. Previous studies have found that within-person declines in objective memory abilities and depressive symptoms increase SMI reports (Hülür et al., 2014). They also showed this association when looking at latent changes in objective memory and SMI across study participation (Hülür et al., 2015). Future work might therefore be worthwhile to consider how parental dementia modifies associations between changes in objective memory and SMI in the HRS sample. Also, more recent waves include the calculation of polygenic risk scores that might elucidate the genetic links between parental dementia and increased memory problems.

#### **Limitations and Strengths**

Our findings should be considered alongside limitations. First, this study did not include information on dementia diagnoses within the full family system (e.g., sibling or spouse). However, a focus on parents is vital as they are more genetically linked than a heterogeneous sample of relatives of various connections, affecting their genetic predisposition for dementia. Also, their parents' aging occurs much further ahead in time which would lead to more concerns about prospective aging, affecting their perceived susceptibility. Adult children are also the second most common caregivers for parents with dementia and acquire a high degree of such (Brodaty & Donkin, 2009), making this a key dyad to consider. Second, HRS changed the wording of the question about parental dementia throughout the study, including the use of "memory-related disease" and not a formal diagnosis of dementia; this leaves room for using more precise clinical indicators in future studies. However, this more open question allows us to capture various forms of memory impairment that would affect individuals' views about their own memory. It also allows us to include a greater number of participants whose family faces disparity or other disadvantages in obtaining a formal diagnosis of dementia. Next, our measurements were limited in the range of SMI reports assessed. Although we were able to examine the impact of parental dementia on current memory problems and perceived memory decline, other methods of measuring SMI reports might be important to consider for future studies. More continuous measures can capture within-person fluctuations in SMI reports (Mogle, Muñoz, Hill, Smyth, & Sliwinski, 2017) and might be more sensitive to the effects of parental dementia on memory perception over time.

Despite these drawbacks, our study had several strengths. First, our study used a large random sample of older adults across the U.S., which improves generalizability. Previous

studies included small samples (Heun et al., 2003) or international samples that might not represent the U.S. (Tsai et al., 2006). Second, this study measured parental dementia using an innovative approach: most previous research only looked at between-group differences in dementia on SMI reports without consideration of time since discovery. Leveraging longitudinal data, we were able to examine the within-person associations of parental dementia on SMI reports. Lastly, although continuous SMI measures may be more likely to capture nuanced relationships, our study took into consideration important recommendations for SMI measurement (Jessen et al., 2014). Specifically, previous studies combined different questions about memory into summary scores that ignore the qualitatively different experiences involved (Rabin et al., 2015); our study considered current memory problems and perceived memory decline as unique symptoms that can provide more precise findings for the field. Furthermore, to address reliability issues with one-item measures, we applied MLM across years, which improves measurement precision. Lastly, MLM included all participants regardless of the number of available follow-ups, which limits the influence of attrition on results.

#### Conclusion

In the U.S., over 5.8 million people live with AD and related dementias (Alzheimer's Association, 2019), prioritizing the need to understand the broader implications of how familial dementia influences older adults. Our results suggest parental dementia might characterize a group of older adults with SMI, and these might represent an important group for preemptive intervention to reduce dementia risk. For example, older adults reporting SMI tend to engage in fewer positive health behaviors (i.e., physical activity, socialization, and intellectual tasks; Ha & Pai, 2018; Lee, 2014) and also experience more health conditions than non-reporters (Jacob et al., 2019). Studies suggest that modification of such health risk factors is feasible (Ngandu et al., 2015) and may offset millions of AD cases in the population (Livingston et al., 2017). Those with parental dementia represent a crucial target due to enhanced genetic risk.

#### Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

### Acknowledgements

The authors thank the research team and study participants from the Health and Retirement study (HRS). HRS data is publicly available and can be accessed by registering here: https://hrs.isr.umich.edu/data-products/access-to-public-data. Request for additional information regarding the specific use of data and data analytic methods may be sent to the first author.

#### Funding

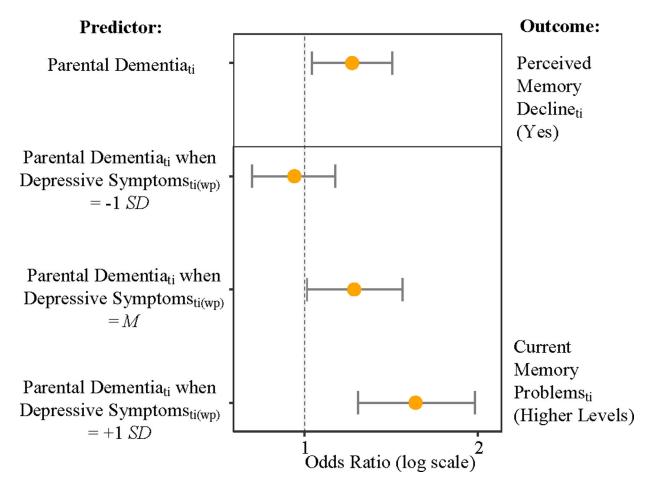
This work was supported by the National Institute on Aging of the National Institutes of Health (grant number NIA R01AG055398). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. This study used data from the Health and Retirement Study (HRS), a public use dataset produced and distributed by the University of Michigan with funding from the National Institute on Aging [grant number NIA U01AG009740].

#### References

- Abramson LY, Metalsky GI, & Alloy LB (1989). Hopelessness depression: A theory-based subtype of depression. Psychological Review, 96(2), 358–372. http://dx.doi.org.ezaccess.libraries.psu.edu/ 10.1037/0033-295X.96.2.358
- Alzheimer's Association. (2019). 2019 Alzheimer's disease facts and figures. Alzheimer's & Dementia, 15(3), 321–387. 10.1016/j.jalz.2019.01.010
- Benito-León J, Mitchell AJ, Vega S, & Bermejo-Pareja F (2010). A population-based study of cognitive function in older people with subjective memory complaints. Journal of Alzheimer's Disease, 22(1), 159–170. 10.3233/JAD-2010-100972
- Brodaty H, & Donkin M (2009). Family caregivers of people with dementia. Dialogues in Clinical Neuroscience, 11(2), 217. [PubMed: 19585957]
- Cutler SJ (2015). Worries about getting Alzheimer's: Who's concerned? American Journal of Alzheimer's Disease & Other Dementias, 30(6), 591–598. 10.1177/1533317514568889
- Cutler SJ, & Hodgson LG (1996). Anticipatory dementia: A link between memory appraisals and concerns about developing Alzheimer's disease. The Gerontologist, 36(5), 657–664. 10.1093/ geront/36.5.657 [PubMed: 8942109]
- Cutler SJ, & Hodgson LG (2001). Correlates of personal concerns about developing Alzheimer's disease among middle-aged persons. American Journal of Alzheimer's Disease & Other Dementias, 16(6), 335–343. 10.1177/153331750101600604
- Gatz M, Pedersen NL, Berg S, Johansson B, Johansson K, Mortimer JA, ... Ahlbom A (1997). Heritability for Alzheimer's disease: The study of dementia in Swedish twins. The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 52A(2), M117–M125. 10.1093/ gerona/52A.2.M117
- Gray HL, Jimenez DE, Cucciare MA, Tong HQ, & Gallagher-Thompson D (2009). Ethnic differences in beliefs regarding Alzheimer disease among dementia family caregivers. The American Journal of Geriatric Psychiatry, 17(11), 925–933. 10.1097/JGP.0b013e3181ad4f3c [PubMed: 20104051]
- Green RC, Cupples LA, Go R, Benke KS, Edeki T, Griffith P, ... Mirage Study Group. (2002). Risk of dementia among White and African American relatives of patients with Alzheimer disease. JAMA, 287(3), 329–336. 10.1001/jama.287.3.329 [PubMed: 11790212]
- Ha JH, & Pai M (2018). Subjective memory problems and availability of emotional support. Research on Aging, 40(10), 978–1007. 10.1177/0164027518797622 [PubMed: 30222047]
- Harwood D, Barker W, Ownby R, & Duara R (1998). Memory complaints in the elderly: A comparative analysis of informant and subject reports among Hispanics and White non-Hispanics. Clinical Gerontologist: The Journal of Aging and Mental Health, 18(3), 56–60. 10.1300/ J018v18n03\_06
- Haussmann R, Ganske S, Gruschwitz A, Werner A, Osterrath A, Lange J, ... & Donix M (2018). Family History of Alzheimer's Disease and Subjective Memory Performance. American J ournal of Alzheimer's Disease and Other Dementias, 33(7), 458–462. 10.1177/1533317518775033
- Health and Retirement Study. (n.d.). (Public Survey Data 1998–2014) public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740) Ann Arbor, MI, 1998–2014.
- Hertzog C, Hülür G, Gerstorf D, & Pearman AM (2018). Is subjective memory change in old age based on accurate monitoring of age-related memory change? Evidence from two longitudinal studies. Psychology and Aging, 33(2), 273–287. 10.1037/pag0000232 [PubMed: 29658747]
- Heun R, Kockler M, & Ptok U (2003). Subjective memory complaints of family members of patients with Alzheimer's disease and depression. Dementia and Geriatric Cognitive Disorders, 16(2), 78– 83. 10.1159/000070679 [PubMed: 12784031]
- Hill NL, & Mogle J (2018). Alzheimer's disease risk factors as mediators of subjective memory impairment and objective memory decline: Protocol for a construct-level replication analysis. BMC Geriatrics, 18(1), 260. 10.1186/s12877-018-0954-5 [PubMed: 30373526]
- Hill NL, Mogle J, Bhargava S, Bell TR, Bhang I, Katz M, & Sliwinski MJ (2019). Longitudinal relationships among depressive symptoms and three types of memory self-report in cognitively intact older adults. International Psychogeriatrics, 1–14. 10.1017/S104161021900084X

- Hill NL, Mogle J, Bhargava S, Bell TR, & Wion RK (2019). The influence of personality on memory self-report among black and white older adults. PloS one, 14(7), e0219712. 10.1371/ journal.pone.0219712 [PubMed: 31306444]
- Hill NL, Mogle J, Whitaker EB, Gilmore-Bykovskyi A, Bhargava S, Bhang IY, ... Van Haitsma K (2018). Sources of response bias in cognitive self-report items: "Which memory are you talking about?" The Gerontologist, 59(5), 912–924. 10.1093/geront/gny087
- Hodgson LG, & Cutler SJ (2003). Looking for signs of Alzheimer's disease. International Journal of Aging & Human Development, 56(4), 323–343. 10.2190/E6J1-PUX7-J43C-090B [PubMed: 14738213]
- Hodgson LG, Cutler SJ, & Livingston K (1999). Alzheimer's disease and symptom-seeking. American Journal of Alzheimer's Disease, 14(6), 364–374. 10.1177/153331759901400606
- Hülür G, Hertzog C, Pearman A, Ram N, & Gerstorf D (2014). Longitudinal associations of subjective memory with memory performance and depressive symptoms: Between-person and within-person perspectives. Psychology and Aging, 29(4), 814–827. 10.1037/a0037619 [PubMed: 25244464]
- Hülür G, Hertzog C, Pearman AM, & Gerstorf D (2015). Correlates and moderators of change in subjective memory and memory performance: Findings from the Health and Retirement Study. Gerontology, 61(3), 232–240. 10.1159/000369010 [PubMed: 25790970]
- Jacob L, Haro JM, & Koyanagi A (2019). Physical multimorbidity and subjective cognitive complaints among adults in the United Kingdom: a cross-sectional community-based study. Scientific Reports, 9(1), 1–11. 10.1038/s41598-019-48894-8 [PubMed: 30626917]
- Jessen F, Amariglio RE, van Boxtel M, Breteler M, Ceccaldi M, Chételat G, ... Subjective Cognitive Decline Initiative Working, G. (2014). A conceptual framework for research on subjective cognitive decline in preclinical Alzheimer's disease. Alzheimer's & Dementia, 10(6), 844–852. 10.1016/j.jalz.2014.01.001
- Kasper J, & Freedman V (2018). National Health and Aging trends Study (NHATS) user guide: Rounds 1—7 beta release. Retrieved from www.nhats.org
- Kinzer A, & Suhr JA (2016). Dementia worry and its relationship to dementia, psychological factors, and subjective memory concerns. Applied Neuropsychology: Adult, 23(3), 196–204. 10.1080/23279095.2015.1030669 [PubMed: 26496236]
- Kornadt AE, Voss P, & Rothermund K (2015). Age stereotypes and self-views revisited: Patterns of internalization and projection processes across the life span. The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 72(4), 582–592. 10.1093/geronb/gbv099
- Lee PL (2014). The relationship between memory complaints, activity and perceived health status. Scandinavian Journal of Psychology, 55(2), 136–141. 10.1111/sjop.12107 [PubMed: 24646046]
- Leventhal H, Diefenbach M, & Leventhal EA (1992). Illness cognition: Using common sense to understand treatment adherence and affect cognition interactions. Cognitive Therapy and Research, 16(2), 143–163. 10.1007/BF01173486
- Livingston G, Sommerlad A, Orgeta V, Costafreda SG, Huntley J, Ames D, ... & Mukadam N (2017). Dementia prevention, intervention, and care. The Lancet, 390(10113), 2673–2734. 10.1016/S0140-6736(17)31363-6
- McPherson S, La Rue A, Fitz A, Matsuyama S, Jarvik LF, McPherson, ... Jarvik LF (1995). Self-reports of memory problems in relatives of patients with probable Alzheimer's disease. International Psychogeriatrics, 7(3), 367–376. Retrieved from cin20. 10.1017/ S1041610295002110 [PubMed: 8821344]
- Mogle J, Hill NL, Bell TR, Bhargava S, Bratlee-Whitaker E, Wion RK, & Tiwari PA (2020). Combined influences of dementia and personality on self-reported memory problems. American Journal of Alzheimer's Disease & Other Dementias, 35, 1–10. 10.1177/1533317519899792
- Mogle J, Muñoz E, Hill NL, Smyth JM, & Sliwinski MJ (2017). Daily memory lapses in adults: Characterization and influence on affect. The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences, 74(1), 59–68. 10.1093/geronb/gbx012
- Montejo P, Montenegro M, Fernandez MA, & Maestu F (2012). Memory complaints in the elderly: Quality of life and daily living activities. A population based study. Archives of Gerontology and Geriatrics, 54(2), 298–304. 10.1016/j.archger.2011.05.021 [PubMed: 21764152]

- Ngandu T, Lehtisalo J, Solomon A, Levälahti E, Ahtiluoto S, Antikainen R, ... & Kivipelto M (2015). A 2 year multidomain intervention of diet, exercise, cognitive training, and vascular risk monitoring versus control to prevent cognitive decline in at-risk elderly people (FINGER): a randomised controlled trial. The Lancet, 385(9984), 2255–2263. 10.1016/S0140-6736(15)60461-5
- Rabin LA, Smart CM, Crane PK, Amariglio RE, Berman LM, Boada M, ... Sikkes SA (2015). Subjective cognitive decline in older adults: An overview of self-report measures used across 19 international research studies. Journal of Alzheimer's Disease, 48(1), S63–S86. 10.3233/ JAD-150154
- Radloff LS (1977). The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement, 1, 385–401. 10.1177/014662167700100306
- Ramakers IH, Visser PJ, Bittermann AJ, Ponds RW, van Boxtel MP, & Verhey FR (2009). Characteristics of help-seeking behaviour in subjects with subjective memory complaints at a memory clinic: a case-control study. International Journal of Geriatric Psychiatry, 24(2), 190–196. 10.1002/gps.2092 [PubMed: 18642390]
- Rue AL, Small G, McPherson S, Komo S, Matsuyama SS, & Jarvik LF (1996). Subjective memory loss in age-associated memory impairment: Family history and neuropsychological correlates. Aging, Neuropsychology, and Cognition, 3(2), 132–140. 10.1080/13825589608256618
- Stawski RS, Sliwinski MJ, & Hofer SM (2013). Between-person and within-person associations among processing speed, attention switching, and working memory in younger and older adults. Experimental Aging Research, 39(2), 194–214. 10.1080/0361073X.2013.761556 [PubMed: 23421639]
- Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JW, & Weir DR (2014). Cohort profile: The health and retirement study (HRS). International Journal of Epidemiology, 43(2), 576–585. 10.1093/ije/dyu067 [PubMed: 24671021]
- Suhr JA, & Kinkela JH (2007). Perceived threat of Alzheimer disease (AD): The role of personal experience with AD. Alzheimer Disease & Associated Disorders, 21(3), 225–231. 10.1097/ WAD.0b013e31813e6683 [PubMed: 17804955]
- Taylor CA, Bouldin ED, & McGuire LC (2018). Subjective cognitive decline among adults aged 45 years—United States, 2015–2016. Morbidity and Mortality Weekly Report, 67(27), 753. [PubMed: 30001562]
- Tsai D, Green R, Benke K, Silliman R, & Farrer L (2006). Predictors of subjective memory complaint in cognitively normal relatives of patients with Alzheimer's disease. The Journal of Neuropsychiatry and Clinical Neurosciences, 18(3), 384–388. 10.1001/ jama.1992.03490110111047 [PubMed: 16963588]
- U.S. Census Bureau. (2018, July 1). Quick facts—Race and Hispanic origin. Retrieved from https:// www.census.gov/quickfacts/fact/table/US/PST045218
- Wallace RB, & Herzog AR (1995). Overview of the health measures in the Health and Retirement Study. Journal of Human Resources, 30, S84–S107. 10.2307/146279
- Wion RK, Hill NL, DePasquale N, Mogle J, & Bratlee-Whitaker E (2019). The relationship between subjective cognitive impairment and activity participation: A systematic review. Activities, Adaptation & Aging, 1–21. 10.1080/01924788.2019.1651188



# Figure 1: Associations between Parental Dementia with Current Memory Problems and Perceived Memory Decline

Note: Parental dementia associations with the likelihood of reporting perceived memory decline model the main effect of parental dementia. Parental dementia associations with the likelihood of higher levels of current memory problems model the significant interaction with within-person depressive symptoms when low (-1 SD = -0.42), mean (M = 1.32), and high change (+1 SD = 3.06) occurred. t = observation at each wave, i = observation per each individual participant, wp = within-person centered.

#### Table 1.

#### Equations for multilevel models.

Unconditional <sup>a</sup> multilevel models	$\begin{array}{l} Cumulative \ Logit \ Pr(Current \ Memory \ Problems_{ti} = k + 1) = \beta 0_i + \beta 1_i(wave_{ti}) + e_{ti} \\ Logit \ Pr(Perceived \ Memory \ Decline)_{ti} = \beta 0_i + \beta 1_i(wave_{ti}) + e_{ti} \end{array}$
Conditional <sup>b</sup> multilevel models	$ \begin{array}{l} Cumulative Logit Pr(Current Memory Problems_{ti} = k + 1) = \beta 0_i + \beta 1_i(wave_{ti}) + \beta 2_i(parental dementia_{ti}) + \\ \beta 3_i(depressive symptoms_{ti} - depressive symptoms_{baseline gmc}) + \beta 4_i(parental dementia_{ti} * depressive symptoms_{ti} - \\ depressive symptoms_{baseline gmc}) + \beta 5_i(parental dementia_{ti} * ethnicity_{ti}) + \beta 5_i(parental dementia_{ti} * race_{ti}) + e_{ti} \\ \beta 0_i = \gamma 00 + \gamma 01(age_i baseline) + \gamma 02(sex_i cat) + \gamma 03(education_{i gmc}) + \gamma 04(income_{i gmc}) + \gamma 05(ethncitiy_{i cat}) + \\ \gamma 06(race_{i cat}) + \mu 0_i \\ Logit Pr(Perceived Memory Decline = 1)_{ti} = \beta 0_i + \beta 1_i(wave_{ti}) + \beta 2_i(parental dementia_{ti}) + \beta 3_i(depressive symptoms_{ti} - \\ depressive symptoms_{baseline gmc}) + \beta 4_i(parental dementia_{ti} * depressive symptoms_{ti} - depressive symptoms_{baseline gmc}) + \\ \beta 5_i(parental dementia_{ti} * ethnicity_{ti}) + \beta 5_i(parental dementia_{ti} * race_{ti}) + e_{ti} \\ \beta 0_i = \gamma 00 + \gamma 01(age_i baseline) + \gamma 02(sex_{i cat}) + \gamma 03(education_{i gmc}) + \gamma 04(income_{i gmc}) + \gamma 05(ethncitiy_{i cat}) + \\ \gamma 06(race_{i cat}) + \mu 0_i \end{array}$

*Note.* Random intercepts were specified for all models; Notations: baseline = variable comes from value at first available wave, treated as fixed; cat = variable is categorical; gmc = grand-mean centered; i = observations for each individual (Level 2); k = level of ordinal variable; t = observations for each timepoint (Level 1).

 $^{a}$ Unconditional describes multilevel models where the only predictor is wave.

 ${}^{b}\mathrm{Conditional}$  describes multilevel models where additional predictors were added in addition to wave.

	Age	Sex Difi	Sex Differences	Ethnicity Differences	lifferences	I	Race Differences		Income (\$)	Education (Years)
	[( <i>US</i> ) <i>W</i> ]	Male [%( <i>n</i> )]	Female [% (n)]	Hispanic [% ( <i>n</i> )]	Non-Hispanic [%(n)]	White $[\% (n)]$	Black [% (n)]	Other [% $(n)$ ]	[( <i>QS</i> ) <i>W</i> ]	[( <i>U</i> ( <i>SD</i> )]
Total Sample	66.09 (1.88)	41.14 (1567)	58.86 (2242)	7.88 (300)	92.12 (3509)	84.20 (3207)	12.23 (466)	3.57 (136)	77,463 (203,551)	13.01 (2.85)
Current Memory Problems (Higher is More Problems)										
(1)	66.84 (2.81)	6.89 (108)	3.57 (80)	4.82 (169)	6.33 (19)	4.77 (153)	6.01 (28)	5.15 (7)	91728 (182396)	13.35 (3.23)
(2)	66.11 (1.96)	23.93 (375)	24.49 (549)	25.22 (885)	13.00 (39)	25.44 (816)	16.95 (79)	21.32 (29)	101358 (315100)	13.91 (2.45)
(3)	66.04 (1.76)	44.16 (692)	47.55 (1066)	46.91 (1646)	37.33 (112)	47.55 (1525)	40.13 (187)	33.82 (46)	75497 (173202)	13.15 (2.58)
(4)	66.04 (1.83)	41.61 (342)	58.39 (480)	20.12 (706)	38.67 (116)	19.77 (634)	29.83 (139)	36.03 (49)	56309 (83353)	11.89 (3.16)
(5)	65.92 (1.09)	42.74 (50)	57.26 (67)	2.94 (103)	4.67 (14)	2.46 (79)	7.08 (33)	3.68 (5)	43995 (60858)	11.32 (3.45)
Significance Test	R4, 3804) = 6.36, b < .001, d = .06	$\chi^{2}$ (4) = $p < .001$	$\chi^2$ (4) = 23.25, <i>p</i> < .001, <i>p</i> = .08	$\chi^2$ (4) = 70.54, $p = <.001$ , $\varphi = .14$	:70.54, , φ=.14		$\chi^2$ (8) = 84.52, $p < .001, \ \varphi = .15$		R4, 3804) = 6.50, p < .001, d = .06	R4,3804) = 71.31, p < .001, d = .19
Perceived Memory Decline										
No (1)	66.13 (1.96)	82.71 (1296)	77.92 (1747)	79.45 (2788)	85.00 (255)	79.36 (2545)	82.83 (386)	82.35 (112)	78,420 (216,952)	12.98 (2.90)
Yes (2)	65.95 (1.51)	17.29 (17.29)	22.08 (495)	20.55 (721)	15.00 (45)	20.64 (662)	17.17 (80)	17.65 (24)	73,663 (138,067)	13.15 (2.65)
Significance Test	i(1483.2) = 2.77, p = .005, d = .14	$\chi^2 = 1$ p = .003	$\chi^2 = 13.14,$ $p = .003, \ p = .06$	$\chi^2 (1) = 5.29,$ p = .021, p = .04	= 5.29, φ = .04		$\chi^2$ (2) = 3.59, $p$ = .166, $\varphi$ = .03		f(1833.4) = .75, p = .454, d = .04	t(1265.7) = 1.50, p = .134, d = .08
Parental Dementia										
No (0)	66.16 (1.99)	65.35 (1024)	61.15 (1371)	62.87 (2206)	63.00 (189)	62.74 (2012)	62.88 (293)	66.18 (90)	2.81 (1.89)	12.99 (2.85)
Yes (1)	65.97 (1.67)	34.65 (543)	38.85 (871)	37.13 (1303)	37.00 (111)	37.26 (1195)	37.12 (173)	33.82 (46)	2.86 (1.84)	13.05 (2.86)
Significance Test	t(3374.5) = 3.12,	$\chi^{2}(1)$ p = .008	$\chi^2$ (1) = 6.96, $p = .008, \ p = .04$	$\chi^2$ (1) = .002, $p$ = .964, $\phi$ < .001	= .002, φ < .001	-	$\chi^2$ (2) = .66, p = .719, p = .01		(3549.8) = .25, p = .805, d = .01	t(3806) = .58, p = .564, d = .02

Aging Ment Health. Author manuscript; available in PMC 2023 May 01.

Bell et al.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 2

Author
Ś
lanus
cript

Author Manuscript

Age	Sex Difference	erences	Ethnicity l	thnicity Differences	ſ	<b>Race Differences</b>		Income (\$)	Education (Years)
[( <i>US</i> ) <i>W</i> ]	[ <i>M</i> ( <i>SD</i> )] Male [%( <i>n</i> )]	Female [% ( <i>n</i> )]	Hispanic [% $(n)$ ]	Non-Hispanic [%(n)]	White [% $(n)$ ]	Black [% ( <i>n</i> )]	Other [% $(n)$ ]	[( <i>U</i> ( <i>SD</i> )]	[( <i>US</i> ) <i>W</i> ]
p = .002, d = .11									

Bell et al.

Inter-Correlations Among Key Study Variables at Baseline (First Wave)

1. Age (years)						
2. Education (years)	03	·				
3. Income (\$) –.C	05 **	.32 **				
4. Depressive Symptoms .0	.004	16**	17 **	ı		
5. Current memory problems						
(1–5, higher is more problems) –	01	21 ***	15 **	.20**		
6. Perceived Memory Decline						
(0 = No, 1 = Yes)	01	.01	02	.16**	.30**	
7. Parental Dementia						
(0 = No, 1 = Yes) -	03	.02	.01	.03	.03	.03

#### Table 4

Multilevel Models Predicting Reports of Subjective Cognitive Impairment

		mory Problems <sup>a</sup> lodel 1		lemory Decline <sup>b</sup> lodel 2
	OR	95%CI	OR	95%CI
Time	1.16**	1.14 - 1.18	1.11**	1.08 - 1.14
Main Effects				
Parental dementia <sub>ti</sub>	1.02	0.86 - 1.21	1.21*	1.03 - 1.42
Depressive symptoms <sub>ti (wp)</sub>	1.15**	1.11 – 1.20	1.30**	1.25 – 1.35
$Black_i$ (ref = White)	2.26**	1.74 – 2.94	0.75	0.57 - 0.97
Other <sub>i</sub> (ref = White)	1.24	0.77 - 2.00		
Hispanic <sub>i</sub> (ref = Non-Hispanic)	1.66 **	1.18 - 2.32	0.59*	0.42 - 0.83
Interactions <sup>C</sup>				
Depressive symptoms <sub>ti (wp)</sub> x	1.10*	1.03 - 1.17	-	-
Parental dementia <sub>ti</sub>				
Covariates				
Age <sub>i</sub> (years)	1.00	0.95 – 1.06	0.99	0.94 - 1.05
Depressive symptoms $_{i \ (bp)}$	1.44 **	1.37 – 1.51	1.52**	1.44 - 1.60
$Female_i$ (ref = male)	0.82	0.69 - 0.97	1.22	1.02 - 1.45
Education <sub>i</sub> (years)	0.73**	0.69 - 0.76	1.07*	1.02 - 1.13
Income <sub>ti</sub> (\$1000)	0.99	0.99 – 1.00	0.99	0.99 – 1.00
Cohort 1 <sub>i</sub> (ref =Cohort 3)	0.64	0.31 - 1.30	0.66	0.33 – 1.32
Cohort 2 <sub>i</sub> (ref =Cohort 3)	0.42**	0.26 - 0.69	0.77	0.48 - 1.26
Cohort 4 <sub>i</sub> (ref =Cohort 3)	0.94	0.77 - 1.15	0.83	0.67 - 1.02

*Note.* Models 1 and 2 were separate mixed models; Model 1 was estimated with a multinormal distribution with a cumulative logit link; Model 2 was estimated using a binary logistic distribution with a logit link; bp = between-person centered; i = observation for each person; t = observation for each wave; wp = within-person centered.

\* p .01.

<sup>a</sup>Esimating the probability that current memory problems is higher values from 1 to 5, indicating more problems.

 $b_{\text{Esimating the probability that perceived memory decline} = 1$ , code for yes that perceived memory decline is reported.

<sup>c</sup>Only significant interactions kept in the final models.