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Persistence of Pain and Cognitive Impairment in Older Adults

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Abstract

BACKGROUND: No studies have examined the longitudinal association between the persistence of pain and its relationship to cognitive problems in older adults. The objective of this study was to examine how the persistent of pain associates with cognitive performance, cognitive impairment, and subjective memory decline.

METHODS: Across 10 biennial waves, 8,515 adults ages 65 and over were assessed from the Health and Retirement Study ($M_{\text{age}} = 74.17$, $SD = 6.87$, 59.2% female). At each wave, individuals were asked to report on pain presence, and if present, rate its intensity and interference with daily activities such as housework or chores. Using running frequencies or averages, we calculated the persistence of pain using these three pain measures. Cognition was assessed using cognitive performance and different cognitive impairment cutoffs. Incident subjective memory decline was additionally measured as new self-reported memory change in the last 2 years. General estimating equations examined concurrent associations between persistence of pain and cognitive variables, adjusting for demographics, depressive symptoms, and medical comorbidities.

RESULTS: Persistence of pain presence was associated with an increased risk of cognitive impairment. Only persistence of pain interference, not pain intensity, was significantly associated with poorer cognitive performance or being classified as cognitively impaired. For every 2 years, persistence of pain interference was associated with 21% increased odds of cognitive impairment. Only one of three pain variables were related to incident subjective memory decline.

CONCLUSIONS: Persistence of pain is associated with poorer cognitive performance in community-dwelling older adults, especially when involving ongoing interference in chores and work. Facilitating pain management might be important for helping to maintain later-life cognition and reduce dementia risk.

Keywords

Pain; chronic pain; cognition; cognitive impairment; subjective memory

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Conflict of Interest Declaration: The authors declare the absence of known competing financial or personal relationships that could have influenced the work reported in this paper.

INTRODUCTION

Recent studies show that persistent pain involves atrophy in shared networks for pain and cognition indicated by lower gray matter volume.^{1, 2} Such neurodegeneration makes cognitive decline a concern in the one in five older adults experiencing persistent pain³ – especially as they already face inherent risks of cognitive disorders such as Alzheimer’s disease (AD) and related dementias increase with age.⁴

In mostly middle-aged clinic samples, studies show that people with pain have poorer cognitive function than controls.^{5–7} Pain in older adults in the general population may also affect cognition. Cross-sectional studies have had mixed results in older adults, even suggesting that poor cognition may be associated with less reporting of pain in the oldest-old.^{8–10} In 2 longitudinal studies, chronic pain assessed at baseline predicted memory decline and/or increased risk of dementia after 4 years¹¹ or 12 years.¹² However, in a third longitudinal study, pain was not predictive of 4-year memory decline except for a marginal association with severe pain.¹³ These studies were limited in their ability to examine the effect of persistent pain. Perhaps the strongest measure of persistent pain was the longitudinal analysis in the Health and Retirement Study (HRS) that required pain be reported in both of the first 2 waves, the pain measure predicted increased dementia risk and memory decline after 10 years.¹² Interestingly, a meta-analysis suggested that studies require more than 4.5 years to detect pain-related changes in cognition.¹⁴

Rather than asking about persistent pain at a single time point or limiting it to 2 occasions, in the present study we examined the persistence of pain with age over multiple waves and its association with cognitive function at each of those waves. Thus, rather than a longitudinal predictive study, we conducted a concurrent longitudinal analysis of persistent pain and cognition at multiple study waves. Such analysis will help us understand if people with persistent pain are more likely to show cognitive problems in real time as it accrues rather than many years later, extending this previous literature. Secondly, pain is multidimensional, but most studies have focused only on the persistence of *pain presence* (i.e., *whether or not someone is experiencing pain*).¹⁵ Here we also examined persistence of *pain intensity* (i.e., *the degree of pain perceived*)¹⁶ and *pain interference* (i.e., *the degree that pain disrupts daily work and activities*).¹⁵ We assessed cognitive performance measured both continuously and based on impairment cutoffs, and we also assessed the relationship between persistence of pain and incident subjective memory decline. Associations were evaluated in community-dwelling adults age 65 and older in the nationally-representative HRS.

METHODS

Participants

Participants were from HRS, a representative dataset for the United States older population that examines contextual factors affecting later life health. Researchers led this study from the Institute for Social Research at the University of Michigan, where they also obtained ethical approval in accordance with the Declaration of Helsinki.¹⁷ Recruitment of adults 50 and over began biennially in 1992 and involved stratified random sampling across the contiguous United States. Further information is described in publications¹⁸ and data are

accessible online (<https://hrs.isr.umich.edu/data-products>). We used data in which consistent questions on pain were first collected biennially, from 1998 to 2016, spanning 18 years over 10 biennial waves. Pain assessment was introduced at the 1998 wave, and 9,513 participants 65 years and older had self-reported pain data. We excluded participants with no cognitive data at baseline ($n = 4$), missing covariate data ($n = 50$), and with baseline memory disease or stroke ($n = 994$). Thus, our final sample consisted of 8,515 older adults ($M_{\text{age}} = 74.17$, $SD = 6.87$, 59.2% female). Detailed baseline descriptives of this sample are provided in Table 1. Sample size and descriptives are similar to those reported in previous work using HRS pain data.¹²

Measures

Persistence of pain—Three pain questions were included consistently in the main HRS battery starting in 1998. Participants were asked about *pain presence* as, “Are you often troubled with pain?” to which they responded Yes or No. For *pain intensity*, participants were asked, “How bad is the pain most of the time:” rated mild, moderate, or severe. If someone reported no pain presence, we labeled their pain intensity as none. Lastly, participants were asked about *pain interference* as, “Does the pain make it difficult for you to do your usual activities such as household chores or work?” rated as either Yes or No.

Persistence of pain presence and persistent pain interference were each operationalized as a running frequency (e.g., $\sum_{t=k}^{t=10} \text{pain presence, } t-1 + \text{pain presence, } t$, $t = \text{time point}$, $t-1 = \text{frequency at previously available time point}$, $k = \text{end time point for participant}$). These scores represent the total number of years that pain endured, ranging from 0 (no pain ever) to 10 (pain duration of across all possible waves).

Persistence of pain intensity was calculated as a running average ($(\sum_{t=k}^{t=10} \text{pain intensity, } t-1 + \text{pain intensity, } t)/k$). These scores represent the average severity of pain occurring at each time point and can range from 0 (no pain intensity ever) to 3 (duration of severe pain intensity). At times when participants did not provide data, running variables were left missing.

Cognitive Performance—Cognitive tests in HRS and their validation have been thoroughly discussed elsewhere.^{18, 19} Briefly, the cognitive tests included measures of episodic memory, attention/processing speed, and vocabulary measured at every wave. The memory test was adapted from the Consortium to Establish Research on Alzheimer’s Diagnosis and included an immediate free recall of 10 words, followed by a delayed free recall of those words. Scores indicate the number of words correctly recalled. HRS measured attention/processing speed through the backward counting tasks, including counting 10 numbers backward from 86 and five iterations of serial 7s from 100. The total score indicates the number of digits correctly stated. In the vocabulary tasks, participants defined five words from one of two sets. In HRS, a total cognitive performance score is derived from summing the values. Scores ranged from 0 to 35, where higher values indicate better performance.

In addition to using cognitive performance as an outcome, we also categorized people as showing cognitive impairment based on different thresholds. First, we standardized cognitive performance and residualized for the effects of age, sex, education, and baseline cognitive performance. We then applied standard deviation cutoff thresholds to define cognitive impairment. For binary analyses, we defined impairment as performing greater than or equal to 1.50 standard deviations below average performance, a common threshold.²⁰ We further classified cognitive impairment severity using a three-level ordinal variable (0 = no impairment, 1 = performing greater than or equal to 1.50 *SD* and less than 2.00 *SD* below average, and 2 = performing greater than or equal to 2.00 *SD* below average). This ordinal variable allowed us to determine if pain-cognition associations were being driven primarily by the most severely impaired individuals.

Incident Subjective Memory Decline—At each wave, participants were asked about subjective memory decline as “*Compared with (previous wave/two years ago), would you say your memory is better now, about the same, or worse than it was then?*” Due to low variability for “better,” we re-coded responses into a binary variable as 0, denoting better or same, and 1, indicating worse performance.²¹ Incident subjective memory decline was defined as subjective memory decline reported newly after baseline or not (newly reported = 1, not newly reported = 0).

Covariates

Demographics.: At baseline assessment, we used demographic information on age, lifetime educational attainment, and household income. Education was categorized as less than high school, high school, and greater than high school. Income was classified as <\$15,000, \$15,000 to 30,000, >\$30,000 to \$60,000, >\$60,000.

Medical Comorbidities.: For health conditions, participants were asked “*Has a doctor ever told you that you have...?*” about multiple conditions, including heart disease, diabetes, lung disease, arthritis, and cancer. We calculated a sum at each wave ranging from 0 (no conditions reported) to 5 (all conditions reported).

Current Smoking Status.: Participants were asked, “*Do you smoke cigarettes now?*” (Yes/No).

Current Alcohol Consumption.: Current alcohol consumption was assessed by asking if one consumed alcohol in the last three months (Yes/No).

Depressive Symptoms.: Depressive symptoms were captured using the eight-item geriatric version of the Center of Epidemiological Studies Depression scale (CES-D).²² On this test, individuals report yes/no if they experienced any of the 8 symptoms in the past week (score range = 0 to 8). Higher scores index more depressive symptoms.

Statistical Analysis

We used general estimating modeling procedures to assess associations between persistence of the three pain measures and cognition. This specifically involved calculating general

estimating equations (GEEs) in SAS, which provides results similar to regression approaches but is able to account for nesting of data within people (SAS Institute Inc., 2014). Gaussian, binomial, and multinomial distributions were applied to the GEEs based on the properties of the outcome variable (continuous, binary, and ordinal, respectively). Our models were nested within participant and inverse probability weighting accounted for age-related attrition. Our time variable was age at each assessment centered at the minimum age of 65. Covariates included time-invariant covariates of sex, race/ethnicity, baseline income, and education, as well as time-varying covariates of current smoking status, currently drinking status, and number of waves in the study. All data preparation and analysis code are publicly available (<https://github.com/trbellucsd/HRSPainandCognition>).

In our main models predicting cognition, we calculated models separately for persistence in each pain variable due to multicollinearity among pain measures (r s ranged from .76 to .92). We follow up on significant associations between pain variables and cognitive performance by looking at associations with specific measures of immediate memory, delayed memory, attention/processing speed, and vocabulary. For persistence of pain presence and pain interference, associations were interpreted as the change in the odds (*OR*) or units (*b*) of outcomes when persistent pain lasted an additional 2 years (1 wave). We examined the association with persistence of pain intensity for every additional one-unit increase in average pain intensity. Significance was determined with an α at .05 and a lack of the null value in the 95% confidence intervals (CIs).

RESULTS

Descriptives of at baseline and change over time.

Cognition over time—On average, cognitive function declined from 21.74 ($SD = 5.45$) at wave one to 15.55 ($SD = 4.67$) at the last wave. At baseline, 6.3% had cognitive impairment ($n = 540$). After adjustment for baseline cognitive impairment, new cases of cognitive impairment rose from 3.1% at the second wave after adjusting for baseline cognitive performance ($n = 215$) to 22.1% at the last wave ($n = 283$). Regarding subjective memory decline, 19.0% had subjective memory decline at baseline ($n = 1614$). Incident subjective memory decline rose from 10.0% at the second wave ($n = 699$) to 20.7% by the last wave ($n = 266$). Descriptives of all variables across time are shown in Supplemental Table S1.

Persistence of Pain Presence over Time—At baseline, 25.8% reported pain at baseline ($n = 2198$). Over time, 45.1% endured no pain presence ($n = 3,843$), 20.9% endured persistent pain present for at least two years ($n = 1,778$), 11.4% endured persistent pain present for four years ($n = 974$), and 22.6% endured persistent pain for six or more years ($n = 1,928$). In a full GEE model shown in Table 2, older age ($b = .06$, 95% CI: .06 to .07), more medical comorbidities ($b = .12$, 95% CI: .05 to .19), current smoking ($b = .27$, 95% CI: .16 to .39), and more follow-up waves ($b = .35$, .31 to .38, all $ps < .01$) were associated with greater persistence of pain presence. Black adults reported less endured persistent pain than White adults ($b = -.33$, 95% CI: $-.15$ to $-.13$) whereas Other race individuals endured more persistent pain presence than White adults ($b = .31$, 95% CI .08 to .43, all $ps < .01$).

Persistence of Pain Intensity over Time—At baseline, 6.9% reported mild pain intensity ($n = 590$), 14.0% reported moderate pain intensity ($n = 1191$), and 4.8% reported severe pain intensity ($n = 484$). Regarding persistent pain intensity, 71.7% of individuals endured no persistent pain intensity ($n = 6,110$), 17.8% endured persistent pain intensity in the mild range (1.00 to 1.99; $n = 1,521$), 8.9% endured persistent pain intensity in the moderate range (2.00 to 2.99; $n = 759$), and 1.6% endured persistent pain intensity in the severe range (3.00 and greater; $n = 133$). Shown in Table 2, more persistence of pain intensity was associated with more depressive symptoms ($b = .06$, 95% CI: .02 to .10) and more waves completed ($b = .25$, 95% CI: .21 to .29, all p s < .01).

Persistence of Pain Interference over Time—At baseline, 15.9% reported pain interference ($n = 1353$). With regard to persistence of pain interference, 61.0% endured no pain interference ($n = 5,200$), 17.3% endured persistent pain interference over two years ($n = 1,474$), 8.4% over four years ($n = 718$), and 13.3% over six years ($n = 1,131$). Shown in Table 2, duration of persistent pain interference was associated with older age ($b = .04$, 95% CI: .04 to .05), identifying as female ($b = .35$, 95% CI: .21 to .48), more comorbidities ($b = .06$, 95% CI: .01 to .11), smoking ($b = .18$, 95% CI: .08 to .28), and more waves completed ($b = .22$, 95% CI: .19 to .25, all p s < .05). Black older adults reported endured less persistent pain interference than Whites ($b = -.21$, 95% CI: $-.38$ to $-.04$, $p = .014$).

Main Models: Associations between Persistent Pain Duration and Cognition

Persistence of Pain Presence and Cognition—Persistence of pain presence was related to worse cognitive performance, whether it was based on continuous or categorical measures (see Supplemental Table S2). For every 2 years of duration, persistent pain presence was associated with a .48-point lower continuous cognitive score (95% CI: $-.65$ to $-.32$, $p < .001$), worsening with longer duration shown in Figure 1 Panel A. Looking at performance in specific measures (near the end of Supplemental Table S2), this primarily involved lower performance in immediate memory ($b = -.11$, 95% CI: $-.15$ to $-.07$) and delayed memory ($b = -.11$, 95% CI: $-.15$ to $-.06$, $p < .001$), as well as vocabulary ($b = -.07$, 95% CI: $-.14$ to $-.004$, $p = .038$).

Looking at categorical cognitive measures, for every additional 2 years of duration, persistent pain presence was associated with 31% increased odds of being cognitively impaired (95% CI: 1.18 to 1.45, $p < .001$). When looking at exact point estimates, odds of cognitive impairment increased linearly with more years with persistent pain. In Figure 2 Panel A, we show the linear increase in odds of cognitive impairment for 2, 4, and 6+ years of persistent pain. In addition, for every additional 2 years of duration, persistent pain presence was associated with 55% higher odds of more severe cognitive impairment (> 2 SDs below average) (95% CI: 1.30 to 1.85, $p < .001$). Persistence of pain presence was also associated with 13% increased odds of developing subjective memory decline (95% CI: 1.03 to 1.25, $p = .013$).

Persistence of Pain Intensity and Cognition—Persistence of pain intensity was not associated with cognitive performance, odds of being cognitively impaired, or subjective

memory decline. Despite similar odds ratios as those for persistence of pain presence, the confidence intervals included 1 ($p > .10$, Supplemental Table S2).

Persistence of Pain Interference and Cognition—Persistence of pain interference was related to cognitive performance, whether it was based on continuous or categorical measures (see Supplemental Table S2). For every additional 2 years of duration, persistent pain interference was associated with a .38-point lower cognitive performance (95% CI: $-.56$ to $-.23$, $p < .001$), worsening with longer duration shown in Figure 1 Panel B. Looking at performance in specific measures (near the end of Supplemental Table S2), this primarily involved lower performance in immediate memory ($b = -.09$, 95% CI: $-.13$ to $-.05$) and delayed memory ($b = -.09$, 95% CI: $-.13$ to $-.05$, $p < .001$).

For every additional 2 years of duration, persistent pain interference was associated with 21% increased odds of being cognitively impaired (95% CI: 1.07 to 1.37, $p = .003$). Odds of cognitive impairment increased linearly with longer persistence of pain interference shown in Figure 2 Panel B. In addition, for every additional 2 years of duration, there was 38% higher odds of more severe cognitive impairment severity (95% CI: 1.21 to 1.57, $p < .001$). Persistence of pain interference was not associated with subjective memory decline ($p = .164$). Full models showing demographic and health characteristics are provided in Supplemental Table S2.

DISCUSSION

Our analyses assessed how persistent pain relates to cognition as it endures over time. Discrepant results emerged when looking across persistent pain measures. Namely, persistence of pain interference but not pain intensity was associated with cognitive impairment. This finding is of particular interest as most studies have used pain intensity rather than pain interference to capture severity, possibly explaining previous non-significant results.²³ Still, it is also worth noting that in this community-dwelling sample, the association was small but meaningful. In particular, for every additional 2 years of duration, persistent pain interference resulted in 21% increased odds of being in the cognitively impaired range. The relationship remained significant when predicting across impairment thresholds, suggesting that results generalize across impairment severities.

These findings partially support the idea that pain limits cognition.²⁴ Yet they also call into question the idea that any pain severity is sufficient to exert this effect and that subjective reports capture this phenomenon.²⁵ First, regarding pain severity, it was participants with persistent interference – not with persistently high pain intensity – who were most likely to be cognitively impaired over time. This suggests that people reporting pain significant enough to disrupt to daily activities are also likely to be facing cognitive difficulties. As such, pain interference might help to identify individuals at more advanced stages of cognitive decline, more so than tracking pain intensity. Second, subjective memory decline was unrelated to either persistence of pain interference or intensity, suggesting that although linked cross-sectionally,^{26, 27} subjective memory decline may not capture longitudinal changes in cognitive function related to persistence of pain.

An association between persistence of pain and cognitive impairment may be related to atrophy found in overlapping neural networks, including the frontal cortex^{1, 28} and hippocampus.²⁹ Both regions are important for modulating pain and cognitive information, and thus, may underpin associations between persistent pain interference and cognitive impairment. Direct, indirect, and reverse paths between neurodegeneration and pain persistence are possible. First, as shown in animal studies, persistent pain may directly lead to brain atrophy as a result of neuroinflammation-driven synaptic loss and deposition of amyloid and tau proteins (for review see ³⁰). Second, persistent pain may be indirectly related to atrophy through comorbid conditions such as depression. Depression is present in 30%–60% of pain patients³¹ and is also associated with neuroinflammation,³² brain atrophy,³³ and higher risk of cognitive impairment.³⁴ Lastly, persistent pain and cognitive impairment could also be shared symptoms that are a result of neurodegeneration.³⁵ While etiology remains to be determined, atrophy in shared neural networks might explain links between persistent pain and higher risk of cognitive impairment.

Persistent reports of pain in older adults may help practitioners flag individuals at risk for deleterious neurocognitive changes. Most cognitive health assessments focus on vascular and metabolic health, but perhaps pain should be incorporated. Historical evaluations are especially critical in individuals presenting with cardiometabolic and inflammatory conditions as pain is highly comorbid. Clinical reviews of current and previous pain would be important as most older adults go unscreened; moreover, those identified with pain often do not receive adequate treatment.^{36, 37} Tracking persistent pain might also aid in explaining who with cognitive impairment experiences significant functional disruption,³⁸ and are at higher risk for more dementia.³⁹ It is promising that some studies show that neurological aberrations related to pain reverse when treating pain in older adults.^{28, 40} How this translates to cognitive function, though, remains to be seen.

Our study does have important limitations to note. All data were observational, and we cannot glean causal links from this study. There was no directionality tested in this study, although some experimental evidence shows that pain produces cognitive decline in older adults.⁴¹ Persistent pain and cognitive problems might also represent covarying symptoms of some unspecified pathology. In addition, more extensive assessments than those used in the HRS might also help to clarify the relationships of pain to cognition and subjective decline. Finally, we do not know whether participants were taking pain medications that might affect both the experience of pain and cognitive performance. Opioid use has been a particular concern and has shown mixed effects with cognition and dementia risk,^{42, 43} but many other medications can affect cognition as well. It is possible that the use of certain medications moderates or mediates observed associations. Nevertheless, strengths of our study include generalizable results between persistent pain and cognition in older adults through the use of a representative sample, weighting for attrition, and controlling for total time in the study.

CONCLUSION

Contrary to popular beliefs endorsed in the general population, pain is not a normal part of aging.⁴⁴ Instead, it may be a key vital sign to determine adverse changes in brain health and cognitive function. Our study showed that persistence of pain interference was associated

with increased risk of cognitive impairment in a large, nationally representative sample of community-dwelling older adults. Persistence of pain interference over an extended time might eventually produce a likelihood of cognitive impairment similar to heart disease and diabetes.^{45, 46} Thus, the role of persistent pain in cognitive function warrants further attention.

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 is a publicly available dataset accessed through 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.

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We certify that this work is novel as no previous study has examined the association between persistence of pain in a large representative United States sample.

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Key points:

- Persistent pain is common in community-dwelling older adults.
- Persistent pain interference but not pain intensity is associated with cognitive impairment.
- Cognitive impairment severity increases linearly with increased persistence of pain .

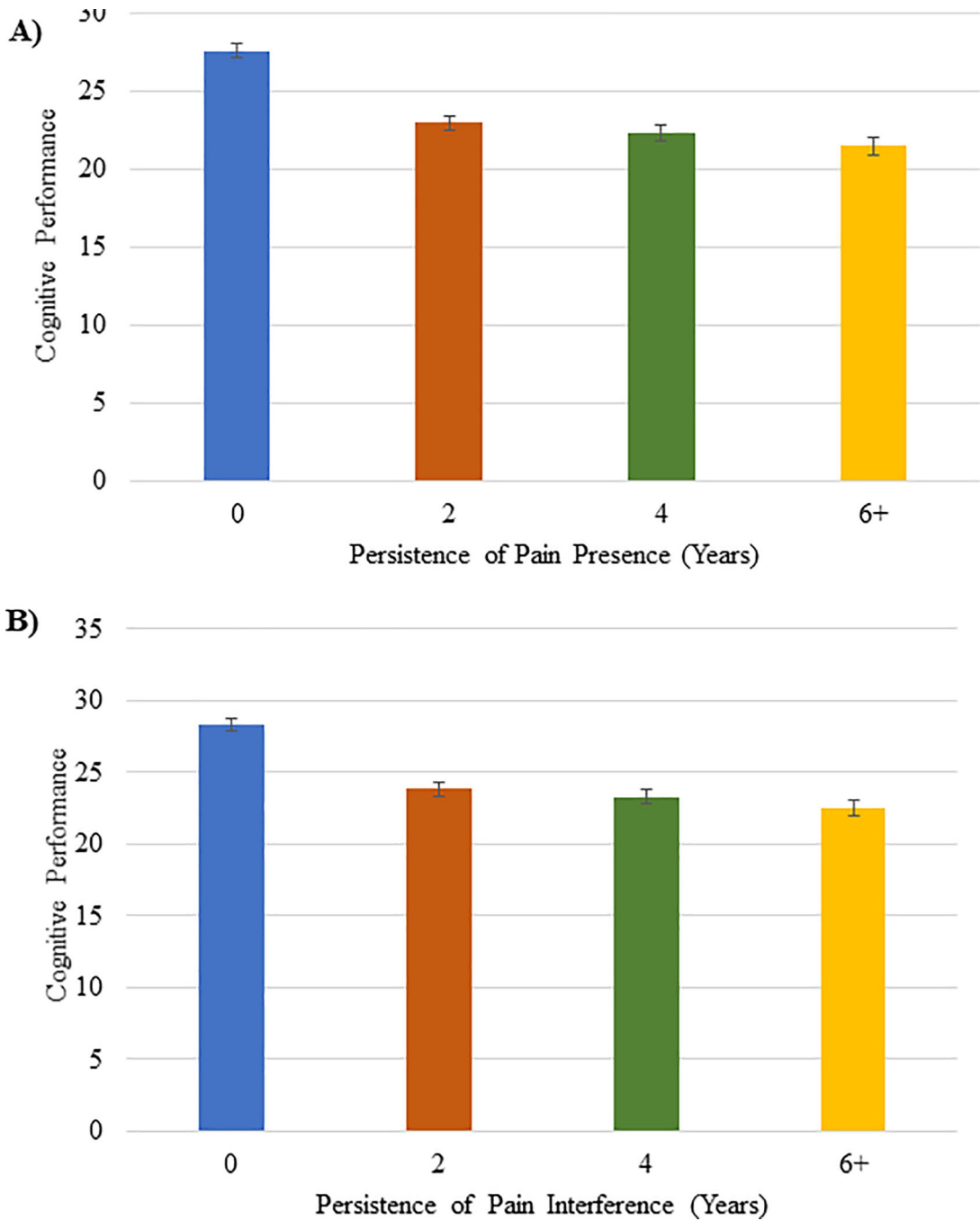


Figure 1. Mean objective cognitive performance by years of persistence of pain.

Note. Cognitive performance was calculated using a total accuracy on items assessing memory, attention/processing speed, and vocabulary – corrected for age, sex, education, and baseline cognitive function (after baseline). Persistence of pain was determined as a running frequency of the number of years reporting pain in the Health and Retirement Study. On the graph, 15 is the minimum on the Y axis as <5% of observations lie below this point. Error bars modeled by standard error.

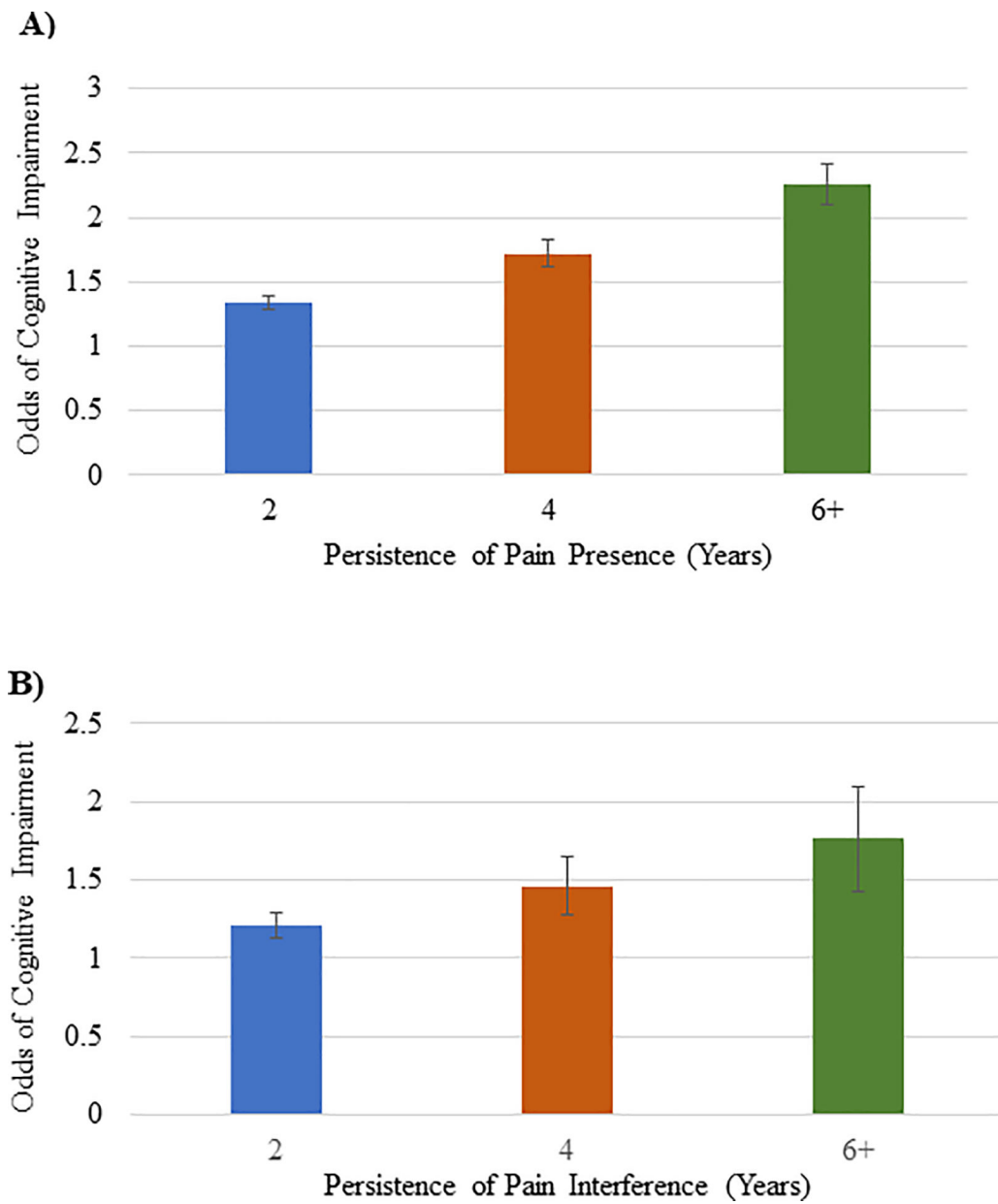


Figure 2. Odds of cognitive impairment by years of persistence of pain.

Note. Cognitive impairment was measured as cognitive performance 1.5 standard deviations below sample mean, after correcting for age, sex, education, and baseline cognitive function (after baseline).

Table 1.Baseline descriptives of the analytical sample ($n = 8,515$).

Variable	<i>n</i> (%)	<i>M</i> (<i>SD</i>)
Pain Presence		
Yes	2198 (25.8)	
No	6317 (74.2)	
Pain Intensity		
0	6233 (74.3)	
1	590 (6.9)	
2	1191 (14.0)	
3	423 (4.8)	
Pain Interference		
Yes	1353 (15.9)	
No	7162 (84.1)	
Cognitive Score ^a		21.74 (5.43) ^b
Cognitive Impairment ^a		
Yes (1.5 <i>SDs</i> below mean)	540 (6.3)	
Yes (1.5 to 2.0 <i>SDs</i> below mean)	297 (3.5)	
Yes (> 2.0 <i>SDs</i> below mean)	243 (2.9)	
No	4747 (92.3)	
Subjective Memory Decline		
Yes	1614 (19.0)	
No	6901 (81.1)	
Age		74.17 (6.87)
Sex (Female)	5040 (59.2)	
Education		
<HS	2814 (33.1)	
HS	2829 (33.2)	
>HS	2875 (33.8)	
Income		
0 to 15k	2396 (28.1)	
15k to 30k	2670 (31.4)	
30k to 60k	2203 (25.9)	
>60k	1246 (14.6)	
Race		
White	7278 (85.5)	
Black	1006 (11.8)	
Other	229 (2.7)	
Comorbidities		
0	1455 (17.1)	1.70 (1.22)
1	2593 (30.5)	

Variable	<i>n</i> (%)	<i>M</i> (<i>SD</i>)
2+	4467 (52.5)	
CESD		1.59 (1.85)
Currently Smoking (Yes)	883 (10.4)	
Currently Drinking Alcohol (Yes)	3867 (45.4)	

Note. CESD = Centers for Epidemiological Studies Depression scale, geriatric version.

^a Adjusted for cognitive performance at baseline in later waves.

^b We rescaled the residualized cognitive scores into the range of the original cognitive test in HRS for descriptives purposes.

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Table 2.Demographic and health predictors of persistent pain variables ($n = 8,515$).

	Persistence of Pain Presence <i>b</i>	Persistence of Pain Intensity <i>b</i>	Persistence of Pain Interference <i>b</i>
	(95% CI)	(95% CI)	(95% CI)
	Model 1	Model 2	Model 3
Age	.06*** (.06 to .07)	.01 (.04 to .01)	.04*** (.04 to .05)
Female Sex	.51*** (.34 to .67)	-.10 (-.22 to .03)	.35*** (.21 to .48)
Race (Reference = White)			
Other	.31** (.08 to .53)	-.07 (-.35 to .21)	.19 (-.02 to .40)
Black	-.33** (-.53 to .13)	.15 (-.03 to .33)	-.21* (-.38 to -.04)
Education (Reference = HS)			
<HS	-.18 (-.41 to .04)	.15 (-.03 to .33)	-.05 (-.22 to .12)
>HS	.08 (-.19 to .36)	.14 (-.23 to .50)	.14 (-.07 to .36)
Income (\$15k to \$30k)			
<15k	.14 (-.05 to .34)	-.12 (-.35 to .11)	.05 (-.11 to .21)
\$30k to \$60k	.17 (-.02 to .35)	.28 (-.03 to .59)	.16* (.02 to .30)
>\$60k	.13 (-.07 to .32)	-.03 (-.30 to .24)	-.001 (-.16 to .16)
Depressive Symptoms (CESD)	-.02 (-.05 to .01)	.06** (.02 to .10)	.004 (-.02 to .03)
Comorbidities	.12** (.05 to .19)	-.06 (-.15 to .02)	.06* (.01 to .11)
Smoking presence (Yes)	.27*** (.16 to .39)	.08 (-.11 to .27)	.18*** (.08 to .28)
Alcohol consumption (Yes)	-.02 (-.13 to .08)	.15 (-.08 to .38)	-.03 (-.10 to .05)
Total waves completed	.35*** (.31 to .38)	.25*** (.21 to .29)	.22*** (.19 to .25)

Notes. HS=completed High School; CESD = Centers for Epidemiological Studies Depression scale, geriatric version. Each column header represents an outcome predicted by the listed variables in the rows in separate general estimating equations.

**
 $p < .001$

**
 $p < .01$

*
 $p < .05$