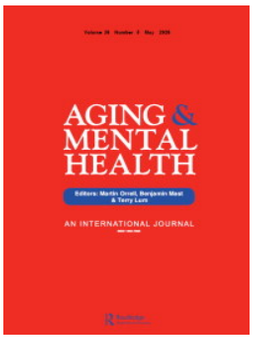

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Depression and cognition: is there a bidirectional relationship?

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ABSTRACT

Objectives: The association between depression and cognition is well-established, but there is uncertainty regarding the direction of causation. We evaluated the relationship between depression and poor cognition in both directions.

Method: We used population-based samples from the Health and Retirement Study (HRS, 2008–2020) and the Midlife in the United States (MIDUS, Waves 2 and 3) study. We used an incidence modeling approach, excluding individuals who exhibited the outcome of interest at the start of the survey interval to help mitigate potential reverse-causality. Adjustment for potential confounders included sociodemographic characteristics, alcohol consumption, and major health conditions.

Results: In the fully-adjusted model for HRS, depression exhibited a significant effect on subsequent cognitive impairment two years later (OR = 1.28, 95% CI 1.12–1.46), whereas the effect of cognitive impairment on subsequent depression was weaker and not significant (OR = 1.10, 0.98–1.23). The pattern of results was similar in MIDUS. Depression was significantly associated with subsequent poor cognition approximately nine years later (OR = 2.01, 1.30–3.12), but poor cognition had no significant effect on subsequent depression (OR = 0.75, 0.93–1.43).

Conclusion: Our results suggest that depression may have an inverse causal effect on cognition, but we found little evidence that good cognitive function had a protective causal effect on depression.

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Depression; cognition; cognitive impairment; bidirectionality; United States

Introduction

Depression is considered one of the leading risk factors for dementia, but the causal direction of the relationship between depression and cognition is unclear (Snyder, 2013). A vast literature demonstrates a cross-sectional, inverse association between depression and cognition (e.g. see meta-analyses by Dotson et al., 2020; Semkovska et al., 2019; Snyder, 2013). Although depression may affect cognition, many researchers acknowledge the possibility of reverse-causality (Brenowitz et al., 2021; John et al., 2019; Lindert et al., 2021) or a bidirectional relationship (Huang et al., 2022; Persin et al., 2025).

In support of the argument that depression has a causal effect on cognition, a recent meta-analysis reports that depression is significantly associated with subsequent cognitive decline (John et al., 2019). Many studies demonstrate that depression (or depressive symptoms) at baseline predicts subsequent cognitive decline or cognitive impairment (e.g. Brenowitz et al., 2021; Han et al., 2021). The evidence for reverse-causality appears to be weaker. Results from one meta-analysis show that better cognition is associated with lower levels of subsequent depression, but the association is no longer significant after controlling for depressive symptoms at baseline (Scult et al., 2017). The authors conclude that the association may be driven by the effect of subclinical depressive symptoms on cognition. However, the authors exclude studies where the mean age at the depression assessment was greater than 65. Therefore, this meta-analysis cannot determine whether cognitive decline predicts depression later in life. Among studies that include older adults, one study shows that individuals with mild cognitive impairment are at increased risk of subsequent depression (Mirza et al., 2017), and a couple of studies find that worse cognition predicts greater subsequent depressive symptoms (Bae, 2021; Lu & Ruan, 2023). Yet, several studies show no evidence of an inverse association between cognition and subsequent depression or depressive symptoms (Gale et al., 2012; van den Kommer et al., 2013; Yuan et al., 2023).

Some studies explicitly test for a bidirectional relationship between depressive symptoms and overall cognition. Two studies report reciprocal effects (Bae, 2021; Lu & Ruan, 2023); Others find that depression

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or depressive symptoms predict worse subsequent cognition or faster cognitive decline, but not the converse (Gale et al., 2012; van den Kommer et al., 2013). One study finds no significant effect in either direction (Yuan et al., 2023). These studies evaluate the effect of each variable on subsequent change in the other variable either explicitly—by modeling individual-level trajectories over time (Gale et al., 2012; van den Kommer et al., 2013)—or implicitly—by using a cross-lagged panel model with autoregressive effects that allow the variable at one wave to affect the same variable at the next wave (Bae, 2021; Lu & Ruan, 2023; Yuan et al., 2023).

A novelty of the current study is that we use an incidence modeling approach that is commonly used in epidemiology. Specifically, we focus on dichotomous measures of depression and poor cognition, excluding from analysis those who report the outcome of interest at the start of the survey interval. The advantage of this method is that it helps establish that the predictor precedes the outcome, reducing the probability that the independent variable was a consequence of earlier changes in the dependent variable. We model the relationship in both directions. Specifically, when testing whether depression predicts subsequent poor cognition, we exclude individuals with poor cognition at baseline to mitigate the possibility that depression is a result of a pre-existing cognitive deficit. In the opposite direction—when assessing whether poor cognition predicts subsequent depression—we exclude those who report depression at baseline to reduce the possibility that poor cognition is a result of concurrent depression. If we find a significant effect in one direction, but not in the opposite direction, it suggests the direction of causation may be one-way. If we instead find significant effects in both directions, we would conclude the relationship is bidirectional.

Our study also expands on the prior literature by using data from two United States population-based samples to cover a wider age range than most of the other studies reviewed here. One sample includes middle-aged Americans (some as young as 33), while the other targets older individuals (above age 50). Replication of the results across two independent datasets lends further credence to the findings. If the findings are similar—despite differences in methods and question wording—it provides reassurance that the findings are not merely the result of a peculiarity in a particular sample (Mroczek et al., 2022).

Materials and methods

Data

The data came from two surveys with overlapping age ranges: Waves 2008–2020 from HRS; Waves 2 (2004–05) and 3 (2013–14) from MIDUS. Table S1 summarizes sample designs, response rates, and restrictions on the analytic sample for each survey. Ethical approval of the protocol for HRS (#HUM00061128) was issued by the University of Michigan Health Sciences/Behavioral Sciences Institutional Review Board (IRB) at the University of Michigan. The protocols for the MIDUS main survey (#2016-1051) and cognitive sub-study (#2016-1051) were approved by the Education and Social/Behavioral Science IRB at the University of Wisconsin-Madison. Informed written consent was obtained from all study participants in both surveys. The current analysis did not require additional ethics approval because it comprised a retrospective study of de-identified data that was kept anonymized (Exemption 4). For HRS and the MIDUS main cohort, we used publicly available data with unrestricted re-use permitted *via* an open license. We applied to and obtained permission from the Inter-University Consortium for Political and Social Research (ICPSR) to use restricted data for the MIDUS Milwaukee sample.

HRS

HRS began in 1992 with follow-up waves every two years and refresher cohorts added every six years; see Text S1 of Supplementary Material for more details. Our analysis used data from Waves 2008–2020, when the Composite International Diagnostic Interview Short Form (CIDI-SF) scale for major depression was administered to all respondents. The institutionalized population was not included in the initial sampling frame, but HRS followed those who became institutionalized after study entry. We excluded institutionalized respondents from HRS to enhance comparability with MIDUS. Individuals interviewed by proxy—who were not administered cognitive tests or questions regarding depression—were also excluded from this analysis. We further limited our analysis to the age-eligible sample, who were aged 54 and older in 2008. Younger cohorts were added in 2010 and 2016 such that the sample represented individuals aged 50 and older in those years. By 2020, respondents were aged 54 and older. Finally, because our outcome was measured at the subsequent wave, we excluded observations in which the respondent did not participate in the survey two years later (e.g. a respondent was included for the 2008–2010 interval only if s/he completed interviews at both 2008 and 2010). Our analytic sample comprised 87,377 observations from 23,420 respondents across six intervals spanning from 2008–10 to 2018–20. The number of respondents per interval ranged from 12,804 respondents (2008–10) to 17,235 (2010–12), with an average of 2.8 intervals per person.

MIDUS

The MIDUS study began in 1995–96 (Wave 1); see Text S2 of Supplementary Material for more details. A new sample of African Americans in Milwaukee was added at Wave 2, approximately 9 years later. At each wave, the initial interview (by phone or computer-assisted personal interviewing, CAPI) was followed by a mail-in self-administered questionnaire (SAQ). Starting at Wave 2, a cognitive battery was administered in a separate phone interview. We restricted this analysis to the 2,940 respondents who participated in cognitive testing at both Waves 2 (aged 33–84 at cognitive testing) and 3 (aged 42–94). The median length of the interval between cognitive testing at Waves 2 and 3 was 9.25 years.

Measures

Depression

Depression was measured by the CIDI-SF scale for major depression (coded 0–7), where values of 4 or higher were classified as depressed (Kessler et al., 1999). The scale was coded following standard practice (see Text S3 for details). In MIDUS, the CIDI-SF scale for major depression was included in the initial phone/CAPI interview.

Cognition

For HRS, we used the Telephone Interview for Cognitive Status (TICS), which was administered to all respondents since 1995/96. The cognitive tests included immediate and delayed recall of ten nouns (both tests scored 0–10), serial 7s subtraction (scored 0–5), and backwards counting (scored 0–2). For self-respondents who completed the interview, HRS imputed missing data on the cognitive tests (see McCammon et al., 2023 for details). Scores were summed to derive a measure of overall cognition (range 0–27). Respondents who scored less than 12 were categorized as cognitively impaired based on the Langa-Weir classifications (Crimmins et al., 2011).

For MIDUS, the Brief Test of Adult Cognition by Telephone (BTACT) was administered separately from the initial phone/CAPI interview. Cognitive tests included immediate and delayed recall of 15 words based on the Rey Auditory Verbal Learning Test (Lezak, 1995; Rey, 1964), backward digit span (Wechsler, 1997), category verbal fluency (Tombaugh et al., 1999), Stop and Go Switch Task (Lachman & Tun, 2008), number series (Schaie, 1996), and the 30 Seconds and Counting Task, a measure of processing speed (Lachman & Tun, 2008). We standardized the scores for each cognitive task at both waves based on the distribution at Wave 2. The composite score was based on the average of the standardized scores from all 7 tasks (Cronbach's $\alpha = 0.77$ for Wave 2, 0.75 for Wave 3). The final scores at both waves were re-standardized based on the distribution at Wave 2. Convergent and discriminant validity have been demonstrated among a subsample of individuals who were administered both the BTACT and an in-person comprehensive cognitive battery (Lachman et al., 2014). Unlike HRS, the BTACT does not have a validated cutoff for cognitive impairment. Thus, we chose a cutoff for poor cognition (less than 1.25 SD below the mean at Wave 2) such that the proportion of MIDUS respondents aged 60–69 with poor cognition at Wave 2 (15%) equals the proportion of HRS respondents of the same age range (across 2008–2018) who were coded as cognitively impaired. Among the full sample of MIDUS respondents, 11% scored below that cutoff at Wave 2.

Control variables

We adjusted for potential confounders that were likely to affect both depression and cognition. Sociodemographic control variables included sex, age, race/ethnicity, education, and marital status. Age was measured at the time of the cognitive testing. Race and ethnicity were based on self-identification and coded into the following categories: non-Hispanic White, non-Hispanic Black, non-Hispanic other races, and Hispanic. Educational attainment was based on degree completion, recoded into five categories: less than high school degree or GED; high school degree or GED; some college or associate's degree; bachelor's degree; graduate degree.

Alcohol consumption was measured by the self-reported number of drinks per week. Based on the CDC (2024) definition of heavy drinking (i.e. 8 or more drinks per week for women; 15 or more drinks per week for men), we categorized alcohol consumption into four categories: none, 1–7 drinks per week, 8–14 drinks per week, and 15 or more drinks per week. We did not include other behavioral factors (e.g. physical inactivity, social connections, and social support) because they are likely to be endogenous (e.g. depression and cognition are likely to influence the respondent's willingness and ability to engage in physical activity).

We also adjusted for four major health conditions: heart trouble, stroke, diabetes, and cancer. HRS asked if a doctor ever told the respondent s/he had diabetes or high blood sugar, whereas MIDUS asked whether the respondent experienced diabetes or high blood sugar in the past 12 months. For the other three health conditions, both surveys asked whether the respondent had ever had the condition. In MIDUS, the question about diabetes was asked in the SAQ for the main cohort but in the CAPI for the Milwaukee sample. All other control variables for MIDUS came from the initial phone/CAPI interview.

Analytic strategy

All analyses were conducted in Stata 18.5. We used multiple imputation to handle missing data (Rubin, 1996; Schafer, 1999). See Text S4 for details.

We estimated two sets of models. First, we modeled whether depression predicted subsequent poor cognition (in MIDUS) or cognitive impairment (in HRS). Second, we modeled the relationship in the opposite direction: did poor cognition/impairment predict subsequent depression? In both cases, the key predictor and all covariates were measured at the beginning of the survey interval (i.e. Wave 2/2004–04, for MIDUS; Wave t for HRS, where $t = 2008$, 2010, … 2018), while the outcome was measured at the end of the survey interval (i.e. Wave 3/2014–15 for MIDUS; Wave $t+2$ for HRS). That is, the relationship between the predictor and the outcome is evaluated over an interval of approximately 2 years for HRS versus around 9 years for MIDUS. Like any incidence model, we excluded observations for survey intervals in which the respondent reported the outcome of interest at the start of the survey interval to mitigate the possibility that the key predictor was a result of a pre-existing occurrence of the outcome.

For each outcome, we fit two models. The first controlled only for the sociodemographic confounders. In preliminary models, we tested a quadratic specification for age to allow for a non-linear age pattern. Based on the Bayesian Information Criterion, the quadratic specification improved model fit when predicting cognition, but the linear specification for age yielded better model fit when predicting depression. In the second model, we further adjusted for alcohol consumption and major health conditions. Because we had up to 6 observations per respondent in HRS (i.e. for survey intervals 2008–10, 2010–12, … 2018–20), we included an individual-level random intercept to account for intra-individual correlation and used a robust variance estimator to correct the standard errors for clustering within primary sampling units. For MIDUS, we used a robust variance estimator to correct for family-level clustering.

We performed several sensitivity analyses. First, given differences in the age range covered by the two datasets, we refit the models on the subset of respondents within the age range in common (50–84). Second, we tested whether the effect of depression on subsequent cognitive decline varied by age because the literature suggests that depression is a risk factor for dementia, but in late life, some of the association is caused by preclinical dementia (Livingston et al., 2024). Therefore, we suspected the association might be stronger in midlife than in later life. Third, we evaluated the sensitivity of the results for MIDUS to alternative cutoffs (15th and 20th percentiles of the distribution at Wave 2) for defining poor cognition. Finally, we fit an auxiliary set of models that used continuous measures of depressive symptoms and cognition to evaluate the effect of depressive symptoms measured at baseline on subsequent change in cognition and, conversely, the effect of cognition measured at baseline on subsequent change in depressive symptoms (see Text S5 for more details regarding the modeling strategy).

Results

Table 1 presents descriptive statistics for HRS and MIDUS. The HRS sample was older (mean age = 66.8, range = 50–104) than the MIDUS sample (mean age = 55.0, range = 33–84). Thus, it is not surprising that the prevalence of major health conditions (e.g. heart trouble, stroke, diabetes, cancer) was higher in HRS than MIDUS.

Modeling subsequent cognitive impairment/poor cognition

At the start of the survey interval, 19% of the HRS sample were cognitively impaired and 11% of the MIDUS sample exhibited poor cognition (Table 1). These observations were excluded from the logit models predicting subsequent cognitive impairment (HRS) and poor cognition (MIDUS).

Among those in HRS who were not cognitively impaired at the start of the survey interval, 12% were cognitively impaired two years later. Among MIDUS respondents who did not exhibit poor cognition at Wave 2, 14% had poor cognition by Wave 3 (~9 years later). Adjusted for sociodemographic confounders (Table 2, Model 1), depression was associated with subsequent cognitive impairment in HRS (OR = 1.36, 95% CI 1.19–1.54) and poor cognition in MIDUS (OR = 2.02, 1.31–3.11). When we further adjusted for alcohol consumption and selected health conditions in Model 2, the ORs for depression were weakened only slightly (HRS: OR = 1.28, 1.12–1.46; MIDUS: OR = 2.01, 1.30–3.12).

Modeling subsequent depression

During the 12-month period prior to the start of the survey interval, 8% of the HRS sample and 10% of the MIDUS sample were depressed (Table 1). These observations were excluded from the logit models predicting subsequent depression.

Among those in HRS who were not depressed at the start of the survey interval, 4% were depressed two years later. Among MIDUS respondents who were not depressed at Wave 2, 6% were depressed by Wave 3 (~9 years later). In the model that was adjusted for sociodemographic confounders (Table 3,

Table 1. Descriptive statistics, HRS 2008–2018 and MIDUS wave 2 (2004–05).

	HRS 2008–2018	MIDUS Wave 2
Female, %	58.8	56.8
Age, mean (SD)[a]	66.8 (9.9)	55.0 (11.2)
Race/ethnicity		
Non-Hispanic White, %	63.7	85.3
Non-Hispanic Black, %	19.2	9.9
Non-Hispanic Other race, %	3.5	2.0
Hispanic, %	13.7	2.8
Education		
Less than high school graduate or GED, %	17.1	4.8
High school graduate or GED, %	52.6	24.8
Some college or associate's degree, %	6.0	29.3
Bachelor's degree, %	14.3	24.1
Graduate degree, %	10.0	17.0
Marital status		
Married, %	58.1	70.2
Partnered, %	5.0	4.3
Widowed, %	16.3	5.4
Divorced/separated, %	15.1	12.2
Never married, %	5.5	7.8
Alcohol consumption		
None, %	61.4	39.1
1–7 drinks per week, %	27.9	47.2
8–14 drinks per week, %	7.0	9.7
15 or more drinks per week, %	3.7	4.0
Health		
Ever had heart trouble, %	22.1	15.1
Ever had a stroke, %	7.2	2.1
Diabetes, %[b]	23.7	8.6
Ever had cancer, %	13.8	12.4
Cognitive impairment, %	18.7	–
Poor cognition, %	–	10.8
Depression, %	7.6	9.6
No. of observations[c]	87,377	2,940

Note. SD = Standard deviation.
[a]The age range at cognitive testing is 50–104 for HRS and 33–84 for MIDUS.
[b]Indicates whether the respondent ever reported diabetes or high blood sugar for HRS, whereas MIDUS refers to diabetes or high blood sugar in the past 12 months.
[c]The analytic sample for HRS represents 87,377 observations for 23,420 respondents observed for up to 6 survey waves (2008, 2010, …2018). The analytic sample for MIDUS represents 2,940 respondents. The denominator for the percentages shown in this table is the number of observations (which, in the case of MIDUS, is the same as the number of respondents).

Table 2. Odds ratios (and 95% CIs) for depression from logistic regression models predicting subsequent cognitive impairment in HRS and poor cognition in MIDUS.

	HRS			MIDUS		
	(1)	(2)	(3)	(1)	(2)	(3)
Depression	1.36*** (1.19–1.54)	1.28*** (1.12–1.46)	1.26*** (1.11–1.44)	2.02** (1.31–3.11)	2.01** (1.30–3.12)	1.99* (1.18–3.35)
Observations	71,020	71,020	68,549[a]	2,623	2,623	1,638[a]

Note. The models for HRS excluded 16,357 observations for which the respondent was coded as cognitively impaired at the beginning of the survey interval. The models for MIDUS excluded 317 respondents who exhibited poor cognition at Wave 2. All models adjusted for sociodemographic characteristics. Models 2 and 3 also adjusted for alcohol consumption and major health conditions (see Table S2 for the full results).
[a]For Model 3, the analysis samples were restricted to the age range in common (50–84).
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 3. Odds ratios (and 95% CIs) for cognitive impairment (in HRS) and poor cognition (in MIDUS) from logistic regression models predicting subsequent depression.

	HRS			MIDUS		
	(1)	(2)	(3)	(1)	(2)	(3)
Cognitive impairment	1.15* (1.03–1.29)	1.10 (0.98–1.23)	1.11 (0.99–1.25)			
Poor cognition				0.78 (0.40–1.49)	0.75 (0.39–1.43)	0.88 (0.42–1.84)
Observations	80,753	80,753	76,728[a]	2,656	2,656	1,751[a]

Note. These models excluded observations for respondents who were depressed during the 12-month period prior to the start of the survey interval ($N = 6{,}624$ observations for HRS; $N = 284$ respondents for MIDUS). All models adjusted for sociodemographic characteristics. Models 2 and 3 also adjusted for alcohol consumption and major health conditions (see Table S3 for the full results).
[a]For Model 3, the analysis samples were restricted to the age range in common (50–84).
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Model 1), cognitive impairment was significantly associated with subsequent depression in HRS (OR = 1.15, 1.03–1.29), but poor cognition was not significantly associated with subsequent depression in MIDUS (OR = 0.78, 0.40–1.49). After further adjustment for alcohol consumption and selected health

conditions in Model 2, the OR for cognitive impairment was no longer significant even in HRS.

Sensitivity analyses

When the samples were restricted to the age range in common (50–84), the pattern of the results remained similar. In the fully-adjusted models predicting cognitive impairment (HRS) and subsequent poor cognition (MIDUS), the ORs for depression remained similar (HRS: OR = 1.26, 1.11–1.44; MIDUS: OR = 1.99, 1.18–3.35; Table 2, Model 3). For the fully-adjusted models predicting subsequent depression (Table 3, Model 3), the ORs cognitive impairment in HRS (OR = 1.11, 0.99–1.25) and for poor cognition in MIDUS (OR = 0.88, 0.42–1.84) were still not significant.

We found no evidence that the effect of depression on subsequent cognitive decline varied significantly by age. However, the direction of the coefficients was consistent with a weaker effect at older ages.

With regard to the sensitivity of the MIDUS results to alternative cutoffs for poor cognition, the results were similar to those presented in Tables 2 and 3. When we defined poor cognition as below the 15th percentile of cognition at Wave 2 (less than 1.05 SD below the mean), the effect of depression on subsequent poor cognition was nearly identical (OR = 2.05, 95% CI 1.35–3.10, $p < 0.01$) and the effect of poor cognition on subsequent depression remained not significant (OR = 0.74, 95% CI 0.41–1.32 p~0.31). When we defined poor cognition as below the 20th percentile of cognition at Wave 2 (less than 0.86 SD below the mean), the effect of depression on subsequent poor cognition was slightly weaker (OR = 1.77, 95% CI 1.18–2.67, $p < 0.01$) and the effect of poor cognition on subsequent depression remained not significant (OR = 0.84, 95% CI 0.50–1.40, p~0.50).

Finally, results using the alternative modeling strategy were partially consistent with the results from the incidence models (see Text S5 for details). There were two notable differences. First, in MIDUS, depressive symptoms at Wave 2 had no significant effect on the subsequent change in cognition, whereas the effect was significant in the incidence model presented in Table 2. Second, in both datasets, better cognition at baseline was associated with *bigger* subsequent increases in depressive symptoms, whereas the effects were not significant in the incidence models presented in Table 3. However, we note that better cognition was cross-sectionally associated with fewer depressive symptoms at baseline, and although the disparity in depressive symptoms across cognitive levels converged as they aged, we found no significant evidence that those who started with better cognition ended up with more depressive symptoms than those with worse cognition.

Discussion

Although there is the possibility of a bidirectional relationship between depression and cognition, we found no evidence of that. Our results suggest that the effect of depression on subsequent cognition is stronger than the converse. In HRS, depression exhibited a significant effect on subsequent cognitive impairment, whereas the effect of cognitive impairment on subsequent depression was weaker and not significant in the fully-adjusted model. There was a similar pattern in MIDUS: depression was significantly associated with subsequent poor cognition, but poor cognition had no significant effect on subsequent depression.

Comparisons with prior research

Prior studies have used continuous measures of depressive symptoms and cognitive function to evaluate bidirectionality. Regarding the question of whether depressive symptoms affects subsequent cognitive decline, like most prior studies (Bae, 2021; Gale et al., 2012; Lu & Ruan, 2023; van den Kommer et al., 2013), our results for HRS indicated that depressive symptoms were associated with faster cognitive decline. In another prior study (Yuan et al., 2023), the effect was in the same direction, but only marginally significant. However, in MIDUS, we found no significant effect of depressive symptoms on subsequent cognitive decline, although the effect was significant in our incidence model. The discrepant findings for MIDUS when using continuous measures may be because we had only two waves of measurement with a long (9–10 years) interval between them (e.g. highly depressed individuals may have recovered, died, or been lost-to-follow-up before they could be re-interviewed 9–10 years later). Most of the prior studies had 3–5 survey waves with 2–4 year intervals. Our analyses of HRS are based on 7 waves fielded every 2 years.

In contrast, regarding the question of whether cognition affects subsequent changes in depressive symptoms in late-life, our results (based on continuous measures) were inconsistent with the prior studies, none of which found cognition associated with *bigger* increases in depressive symptoms. Most of the prior studies found cognition had no significant effect on subsequent changes in depressive symptoms (Gale et al., 2012; van den Kommer et al., 2013; Yuan et al., 2023), which is consistent with our results from the incidence models.

Two other studies reported that better cognition was associated with *smaller* increases in depressive symptoms (Bae, 2021; Lu & Ruan, 2023), but those studies used a cross-lagged panel model, which is likely to yield biased estimates of the causal effect of cognition on subsequent changes in depressive symptoms. As demonstrated by Glymour et al. (2005), adjusting for baseline measures of the outcome (i.e. controlling for lagged values of the dependent variable) is likely to bias the results, particularly when there is measurement error in the repeated values of the dependent variable or when changes in that variable began prior to baseline. In this case, controlling for lagged values of depressive symptoms may inflate the association between cognition and subsequent changes in depressive symptoms, especially if depressive symptoms began earlier in life—before the study began—or measurement of depressive symptoms is unstable or unreliable. In more recent work, both Tennant et al. (2022) and Glymour (2022) agree that controlling for baseline levels yields misleading estimates when the lagged dependent variable is a potential mediator.

The studies that used the more appropriate method—growth curve modeling—were based on data from England (Gale et al., 2012) and the Netherlands (van den Kommer et al., 2013) with much smaller samples than HRS. Also, van den Kommer et al. (2013) controlled for social participation, which is likely to be endogenous (i.e. depressive symptoms and poor cognition may inhibit social participation) and thus, may have obscured the total effect of depressive symptoms. We do not have a theoretical justification to explain our finding from the sensitivity analysis—better cognition predicted bigger increases in depressive symptoms during late-life. Nonetheless, despite convergence, we found no evidence that those who started with better cognition ended up with more depressive symptoms as they aged than those who started with worse cognition.

Our approach and findings

One strength of our analysis is the longitudinal, prospective design based on population-based samples covering a wide age range starting in midlife. Although we cannot prove that depression has a causal effect on cognitive decline, the exclusion of individuals exhibiting poor cognition at baseline helps mitigate potential reverse-causality.

Our most robust finding was that depression appears to predict subsequent cognitive impairment. We found the same pattern in both datasets; the pattern was similar in HRS when we used continuous measures of depressive symptoms and cognition, and prior studies have shown the same pattern. It was only for MIDUS when fitting models based on continuous measures that the effect was not significant.

In contrast, we found weak and inconsistent evidence that cognition predicts subsequent depression in late-life. There was no significant effect in the incidence models for either dataset, and prior studies using continuous measures and an appropriate modeling strategy also found no significant effect. Although we did find a significant effect in both datasets when we used continuous measures, the effect was in the *opposite* direction from expectations. In conclusion, we find little evidence to suggest that better cognition protects against late-life depression.

Potential mechanisms

In terms of biological mechanisms, Butters et al. (2008) proposed that depression contributes to cognitive decline through two primary pathways: 1) increased production of glucocorticoids (hypercortisolemia), which causes hippocampal loss; and 2) increased risk of vascular disease. In turn, hippocampal atrophy and vascular disease adversely affect brain/cognitive reserve, leading to earlier or more frequent cognitive impairment (Butters et al., 2008). Other potential biological mechanisms include inflammation, a decrease in nerve growth factors (e.g. brain-derived neurotrophic factor, BDNF), and increased deposition of amyloid-β plaques, which may be related to glucocorticoid levels or neurotransmitter (e.g. norepinephrine, serotonin) imbalances (Dafsari & Jessen, 2020). The effect of depression on health behavior may further amplify the biological mechanisms. For example, physical inactivity, smoking, and other substance use may increase the risk of vascular disease.

Limitations

The inconsistencies between the results for the two datasets could be a result of the lack of comparability between MIDUS versus HRS in study design and measures. First, unlike HRS, we do not have a validated cutoff to identify cognitive impairment (or dementia) for MIDUS. Second, MIDUS has less statistical power than HRS because of a smaller sample and less frequent survey waves. Consequently, the significance level tends to be lower in MIDUS even when the effect size is larger. Third, the HRS sample is more ethnically diverse and may better represent minorities than MIDUS.

Another key limitation of this study is that individuals with dementia are likely to be institutionalized and would be excluded from our analysis. Even if

they were not institutionalized, those suffering from dementia may have been unable to participate in the survey. Only 3% of the HRS analysis sample would be categorized as demented based on the Langa-Weir classification (Crimmins et al., 2011). Thus, our analysis can capture the early stages of cognitive decline, but it is unlikely to provide information about the relationship between depression and dementia.

Attrition owing to loss-to-follow-up and mortality could also bias our results. If individuals who experienced depression or cognitive impairment were less likely to participate in the survey or were more likely to die before they could be re-interviewed, it may have attenuated the estimated effects in one or both directions. Although we adjusted for various confounders, it is possible that depression and cognitive decline are symptoms of the same underlying omitted variables. For example, genetic factors may contribute to both depression and cognition (Hagenaars et al., 2016). In terms of brain function, cognition is generally associated with the prefrontal cortex and hippocampus, whereas emotion is more closely linked with the amygdala and anterior cingulate cortex; however, cognitive and emotional processes interact and influence each other through inter-connected neural networks (Okon-Singer et al., 2015; Pessoa, 2018). Thus, these overlapping circuits could contribute to both depression and cognitive decline.

Implications

If depression has a causal effect on cognition, then interventions that ameliorate depression may slow aging-related cognitive decline. Some experts suggest that treatment with antidepressants may promote neurogenesis (i.e. generation of new neurons) in the hippocampus, inhibit amyloid production, and reduce inflammation, all of which may delay cognitive impairment (Dafsari & Jessen, 2020). Yet, evidence from randomized trials regarding the cognitive benefits of antidepressant medication is mixed. Some studies report no post-treatment improvement in cognition among depressed individuals (Culang et al., 2009; Nebes et al., 2003), even after remission of depression (Shilyansky et al., 2016). A recent meta-analysis of 8 studies among adults aged 50 or older with late-life depression reported improvement in memory and learning, but not other cognitive domains (Ainsworth et al., 2024). Other research indicates that cognitive deficits often persist even after remission of depression (Koenig et al., 2015; Semkovska et al., 2019).

Even if the relationship between depression and cognition is not causal (e.g. both depression and cognitive impairment are a consequence of a common set of causes), physical activity may improve both mood and cognition. Dotson et al. (2021) recommend exercise as an ideal treatment for late-life depression accompanied by cognitive impairment because it affects neurobiological mechanisms linked with mood and cognitive function. For example, aerobic exercise may improve both cognitive and affective symptoms of depression by increasing BDNF production (Guerrera et al., 2020). Two meta-analyses found no evidence that exercise improved cognition among depressed individuals (Brondino et al., 2017; Sun et al., 2018), but a more recent meta-analysis (Ren et al., 2023) suggests that exercise may have a beneficial effect on global executive function and working memory among people with depression.

Another alternative therapy that has been used to treat depressive symptoms is eye movement desensitization and reprocessing (EMDR) (Carletto et al., 2021; Seok & Kim, 2024). It may be particularly effective for treatment-resistant depression (Minelli et al., 2019). Although it is unclear whether EMDR could help prevent or ameliorate aging-related cognitive decline, recent experimental work suggests that EMDR may help improve cognition among older adults with post-traumatic stress disorder (Gielkens et al., 2024).

Conclusion

In two US datasets covering an age range beginning in midlife, we found evidence supporting the notion that depression may have an inverse causal effect on cognition, but there was little evidence that cognitive function has a protective causal effect on depression. Our findings suggest that the consequences of depression for cognition in later life may be evident long before the manifestation of dementia.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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Data availability statement

The data that support the findings in this study are openly available from HRS at https://hrs.isr.umich.edu/data-products/ and from MIDUS at https://midus.colectica.org/ and https://www.icpsr.umich.edu/web/ICPSR/series/203.

For HRS, we used the 2020 Cross-Wave Tracker File (Final, Version 1, January 2024), the RAND HRS Longitudinal File 2020 (version 2, May 2024), the RAND HRS Fat files for 2008–2020, and the Langa-Weir Classification of Cognition Function 1995–2020 (version 2, May 2023).

For MIDUS, we used the data from MIDUS1 (Core) for Project 1 (main survey, version 19, https://doi.org/10.3886/ICPSR02760.v19); MIDUS 2 (Core) for Project 1 (main survey, version 8, https://doi.org/10.3886/ICPSR04652.v8), Project 3 (Cognitive, version 7, https://doi.org/10.3886/ICPSR25281.v7), and the Milwaukee survey (MKE1, version 7, https://doi.org/10.3886/ICPSR22840.v7); from MIDUS 3 (Core) for Project 1 (main survey, version 7, https://doi.org/10.3886/ICPSR36346.v7), Project 3 (Cognitive, version 3, https://doi.org/10.3886/ICPSR37095.v3), and the Milwaukee survey (MKE2, version 2, https://doi.org/10.3886/ICPSR37120.v2); and the Mortality file for the Core Sample (1/23/2024 version, https://midus-study.github.io/public-documentation/Mortality/Core/MIDUS_Core_MortalityCauseData_N2459_20240123.sav; not yet published at ICSPR).

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