Psychiatric history and later-life cognitive change: effect modification by sex, race, and ethnicity

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ABSTRACT

Objective: We explored associations between psychiatric history and cognitive functioning, and differences by sex and race/ethnicity (SRE) in 20,155 Health and Retirement Study (1995–2014) participants aged 65 or older.

Methods: Multi-level growth curve models examined cognition scores and their trajectories over time by SRE.

Results: A history of psychiatric, emotional, or nervous problems was significantly related to cognition scores and rates of decline. Hispanic and Black participants had significantly lower cognition scores at age 75 and steeper rates of decline than White females, and Black race and the Hispanic race/ethnicity-sex interaction erased the protective effects of being female.

Conclusions: Future research should include specific psychiatric diagnoses. Population level findings as reported here, along with aggregate findings from similar studies, can inform interventions and policies regarding support for populations that are vulnerable to mental illness and to subsequent cognitive decline.

Introduction

As individuals age, they often experience declines in cognition, which occur non-monotonically and may be impacted by various demographic, lifestyle, socioeconomic and health factors (Brown & Mutambudzi, 2022). Women outperform men in several components of cognitive function and therefore on composite cognition scores, at baseline and over time (Ahrenfeldt et al., 2019; McCrarry et al., 2016). Current literature shows evidence of differences in cognition declines over time by race/ethnicity, with minorities faring worse than their white counterparts (Díaz-Venegas et al., 2016; Glymour & Manly, 2008). These differences may be partially attributed to varied structural and intermediary determinants of health across the life course, starting in early life and through adulthood (Glymour & Manly, 2008; Zhang et al., 2016). Building on previous research identifying the influence of psychiatric history on cognitive outcomes in later life (Brown, 2010; Brown & Mutambudzi, 2022; Brown & Wolf, 2018), the current study explores the association between psychiatric history and cognitive outcomes and the differences in these outcomes among men and women of different racial and ethnic groups.

Research has shown that non-white groups are more vulnerable to early life disadvantage which extends through adulthood (Najdowski & Bernstein, 2018; Williams & Sternthal, 2010; Zhang et al., 2016) and are more likely to have poor mental health outcomes (Koivistoinen et al., 2018; Rosenfield, 2012). According to the World Health Organization (WHO) poor mental health is the most important contributor to the burden of disease and disability accounting for 12.3% of disability adjusted life years (DALYs) lost and 31% of all years lived with disability at all ages and in both sexes globally (World Health Organization, 2002). Sex and race/ethnicity (SRE) differences in mental health outcomes have been reported in the literature (Kiely et al., 2019). The literature suggests that across the life course women have higher rates of poor mental health outcomes, however this gender gap is reduced in later life (Eack & Newhill, 2012; Kiely et al., 2019; Miranda et al., 2008; Williams, 2018). This early and continuous disadvantage is likely to contribute to differential rates of cognitive decline in later life, with women faring worse. There is also evidence of disparities in the association between race and mental health (Williams, 2018). Black and Hispanic adults reportedly exhibit lower rates of mental health outcomes such as psychiatric disorders and major depression relative to Whites. They are, however, more likely to have severe or debilitating cases, with subsequent adverse health outcomes including cognitive impairment with lower probability of receiving quality treatment (Eack & Newhill, 2012; Miranda et al., 2008; Williams, 2018).

Specific mental illness diagnoses, as well as the broader definition of a history of psychiatric, emotional, or nervous problems, have been associated with greater risk of later-life cognitive decline or dementia (Brown, 2010; Brown & Mutambudzi, 2022; Brown & Wolf, 2018; Gildengers et al., 2004). Older adults with bipolar disorder, for example, have steeper rates of cognitive decline than non-bipolar older adults of similar ages and education levels (Gildengers et al., 2009; Montejo et al., 2022). Similarly, schizophrenia has been associated with cognitive decline in older adults (Liebers et al., 2016). Analysis of large, nationally representative panel data and corresponding Medicare beneficiary data found that the odds of receiving a dementia diagnosis were greater for older adults with bipolar disorder, schizophrenia, and major depressive disorder diagnoses (Brown & Wolf, 2018). Many studies evaluating these associations for specific diagnoses used smaller samples,
cross-sectional data, or did not explore these associations by race, ethnicity, or sex (Gildengers et al., 2004, 2009; Liebers et al., 2016). Panel studies using large, representative samples of older adults collect data on broader definitions of mental health or psychiatric history, rather than specific diagnoses (Brown, 2010). To our knowledge, there are currently no studies examining SRE differences in the association between psychiatric history and changes in cognitive functioning over time in later life. The current study analyses longitudinal panel data from a representative sample of older adults that allows for the disaggregation of the association between a broad definition of psychiatric, emotional, or nervous problems and cognitive function by sex and race/ethnicity. Assessing this association without accounting for the complex intersectionality of race/ethnicity and sex limits our understanding of the lifecourse associations between psychiatric history and cognitive health in older adults.

To obtain a comprehensive understanding of the association between psychiatric history and later-life cognitive outcomes, it is important to account for ascribed characteristics, childhood disadvantage, and various mid- to late-life characteristics that influence risk of adverse mental health and may also independently contribute to poor cognitive status in older adults. Further, the intersectionality of race/ethnicity and sex in these associations, which may have a complex relationship with baseline cognition and patterns of decline over time, should be further assessed given the life-long disparities that differentially contribute to incidence and severity of mental health in women and minorities. This is important when developing strategies for reducing lifetime risk, incidence, prevalence, and pace of cognitive decline as well as reducing racial disparities that may be attributed to early life and accumulating disadvantage throughout the life course.

The objectives of this study were therefore to explore SRE differentials in the relationships between psychiatric history and cognitive functioning in later life, while accounting for early life disadvantage and relevant covariates. We hypothesize that individuals with a longer-term history of psychiatric, emotional, or nervous problems will have lower cognitive scores at age 75 and steeper rates of cognitive decline as they age, net the effects of early childhood status and later life sociodemographic factors, health status and health behaviors. Furthermore, we hypothesize that the effect of this relationship will be greater for Hispanic and Black men and women than for White men and women, net the effects of early childhood status, later-life sociodemographic factors, health status, and health behaviors. These findings will enhance our understanding of some of the disparate experiences throughout the life course that contribute to differential cognitive functioning and/or severity of cognitive decline in later life, thereby providing knowledge that will allow for improved long-term care provision to future generations of older Americans.

Materials and methods

**Sampling strategy**

This study used ten waves (1995–2014) of Health and Retirement Study (HRS) and Asset and Health Dynamics among the Oldest Old (AHEAD) data. The 1995 AHEAD study was merged with the HRS in 1998 and participants were interviewed every two years thereafter. The HRS oversampled Hispanics and Blacks at the rate of 2:1 to improve representation. Additional detailed sampling procedures have previously been described and are available elsewhere (HRS Staff, 2008). Participants were included in the sample if they had completed at least one valid cognition interview. Participants were excluded if they identified as “other” race. Participants were excluded from a wave if they (1) were younger than 65 in that wave, (2) did not participate in the cognitive assessment, or (3) completed the interview by proxy. This resulted in a sample of 20,493 participants and a total of 91,779 observations. Early-life characteristics were unavailable for 813 observations, such that our final models analyzed 90,966 observations from 20,155 individuals: 6745 White men, 8996 White women, 1068 Black men, 1747 Black women, 654 Hispanic men, and 945 Hispanic women.

**Data collection**

Data for the current study were collected from core interview data in 1995, 1998, 2000, 2002, 2004, 2006, 2008, 2010, 2012 and 2014. Cognition scores (dependent variable) were derived from the modified Telephone Interview for Cognitive Status (TICS-m). The focal independent variables for psychiatric history were constructed at each time point, while race/ethnicity and sex were included in the models as effect modifiers. Baseline controls included childhood disadvantage index (CDI) scores, child health, and educational attainment. Time-varying controls were collected at each wave and included later-life SES, health and health behaviors, and attrition.

**Measurement**

**Dependent variable: cognition scores**

To measure cognitive status participants aged 65 and older were administered a modified version of the TICS-m instrument. The TICS-m was modified to measure six tasks—immediate and delayed recall, serial 7’s subtraction, counting backwards, object naming, orientation to date, month, year, and day of the week, and naming the current President and Vice President—with a maximum score of 35 points evaluating memory and executive function and weighting fluid cognitive measures more heavily than in the original TICS (Freedman et al., 2001). Higher scores imply better cognitive functioning. Herzog and Wallace (1997) suggested a cut-off score of 8 to indicate severe cognitive impairment. As previously established by Herzog and Wallace (1997), self-interview participants (not requiring a proxy to complete the interview) who are missing on an individual item or group of items in the cognition test, are very likely to have refused because they are cognitively unable to do so. Sloan and Wang (2005) determined that imputing these missed items as zeroes does not undermine the accuracy of results in the HRS TICS-m, and these imputations restored many “missing” participants to the analysis, resulting in a more accurate representation of mean cognition scores and their relationship to other factors in the model (Sloan & Wang, 2005). Attrition analysis, stratified by the year a respondent entered the study and conducted for study entrants in waves 1995 through 2012, showed that participants who left the study in the following wave had lower cognition scores than participants who did not leave the study.

**Focal independent variable: psychiatric history**

The HRS inquires into psychiatric, emotional, or nervous problems, rather than specific diagnoses or categories of mental disorders. Although this measure captures a history of psychiatric, emotional, or nervous problems, we are calling it
psychiatric history. Psychiatric history data were identified by two questions: “Have you ever had or has a doctor ever told you that you have any emotional, nervous, or psychiatric problems?” and “Do you now get psychiatric or psychological treatment for these problems?” Participants scoring as “don’t know” or “refused” (from 1 to 31, depending on the wave) were assigned the modal value of zero. These questions were used to create three variables: longer-term history (yes to “ever had” but may or may not currently be getting treatment), current treatment (yes to “ever had problems” and also reported currently getting treatment for those problems), and incident cases (yes to “ever had problems” after having said no in at least one previous wave). The three psychiatric history variables in this analysis were coded as time-varying covariates to control for participants who had experienced recent onset of psychiatric problems. This coding attempted to separate incident cases from those with a past history of psychiatric problems and to account for psychiatric problems that may be prodromal to the onset of dementia. All three variables are included as time varying, in an attempt to control for cases that are prodromal to dementia onset (Wetherell et al., 1999).

Effect modifiers
The effect modifiers in this analysis are sex and race/ethnicity (SRE) and are baseline nonvarying variables. Participants identifying as a race or ethnicity other than Hispanic, White, or Black are excluded because there are too few cases to ensure sufficient statistical power for analysis (Moody-Ayers et al., 2005). Race/ethnicity are coded into three categories and combined with sex to create SRE variables: White males and females (reference), Black males and females, and Hispanic males and females.

Early-life characteristics
The childhood disadvantage index (CDI) is a scaled index derived from four childhood measures collected for the full sample in 1998 and for new participants in later waves and included as baseline values. These variables—maternal and paternal education (less than or greater than 8 years—more education indicated better status), family SES (higher values indicated better status), and father’s usual occupation (more prestigious occupations indicated better status)—were each converted to dichotomous variables indicating no disadvantage (0) or disadvantage (1). A participant’s disadvantage score could range from 0 to 4 and varied based on the number of items for which they provided data. Because not all participants provided data for all four measures, the disadvantage scores were standardized using a scaled index. The index was scaled to range between 0 (no disadvantage) and 1 (most disadvantaged), indexed on the number of measures (between one and four) to which participants responded (Brown, 2010). Sensitivity analyses indicate that participants missing observations on all four variables in the index were more disadvantaged than participants who provided data for one or more of the variables, and they were assigned a value of 1, to indicate the highest level of disadvantage (Brown, 2010). While there is some evidence of association between childhood health and childhood disadvantage (Brown, 2010), childhood health has also been identified as a life course factor influencing health in adulthood (Ferraro & Shippeee, 2009; Haas & Rohlfison, 2010); therefore, child health (fair/poor = 1) is included as a control variable. Tests confirmed no interaction between CDI and psychiatric history.

Later-life characteristics
Other independent variables linked to differences in cognitive outcomes by race and sex were included as controls in the fully specified model (Glymour & Manly, 2008). These include a baseline measure of respondent education (less than high school, more than high school, reference category: high school or equivalent). Time-varying measures include marital status (widowed, divorced/separated, never married, reference category: married or partnered), household income (continuous), self-rated health (higher numbers represent better health), and dichotomous indicators of vision and hearing impairment, the presence of chronic health conditions, and whether the respondent was currently smoking or ever drinking alcohol.

Analytic strategy
This study used multi-level growth curve models (GCM) to compare baseline cognition scores and analyze trajectories of cognitive change for different groups as they age. GCMs model change in the dependent variable, identifying within-person and between-person variability or cumulative inequality using covariates and controls (Ferraro & Shippee, 2009; Hox et al., 2002; Singer & Willett, 2009). GCMs are designed specifically for analyzing trajectories in repeated measure longitudinal or panel data (Bollen et al., 2004; Kelley-Moore & Ferraro, 2004; McDonough & Berglund, 2003).

Time was measured using chronological age, centered on the grand mean of the sample (Alley et al., 2007; Slivinski & Mogile, 2012), which means that the linear age effect identified in these analyses is the age effect at centered age (75). Centering age on the sample’s grand mean allows the intercept to represent a respondent of average age at the baseline to determine changes in cognitive function based on the difference in age between the individual and the group and to accommodate the inclusion of different cohorts at different time points in the study and the assumption of within-person and between-person age effects for different cohorts. This slope may vary at different ages, and this variation is visible in the plotted trajectories in Figure 1.

Bivariate associations of cognition, psychiatric history, and CDI by SRE were evaluated first, followed by a discussion of the growth curve modeling results. Cognition scores were calculated for each group over time using results of the fully specified GCM, inclusive of all control variables listed in Table 1, and used to plot prediction lines or cognition trajectories. These trajectories illustrate the combined effects of SRE and psychiatric history on cognitive function and decline as participants aged through the study. Effect modification was assessed by adding SRE and psychiatric history interaction terms to the fully specified models; however, these interactions were nonsignificant and therefore we did not do any further stratiﬁcation. All analyses were performed using the Statistical Analysis System (SAS) statistical software package, version 9.4. Due to the nature of this secondary data analysis, the Syracuse University Institutional Review Board waived the need for approval.

Results
Bivariate comparisons of cognition scores, psychiatric history, and childhood disadvantage by SRE (Table 1) revealed significant differences in mean cognition scores, psychiatric history, and childhood disadvantage across all six SRE groups. The
Figure 1. (a) Predicted Cognition Scores by Age and Sex, non-Hispanic Blacks and non-Hispanic Whites, 1995–2014 HRS/AHD. (b) Predicted Cognition Scores by Age and Sex, Hispanics and non-Hispanic Whites, 1995–2014 HRS/AHD.

Table 1. Sample characteristics by sex and race/ethnicity, 1995–2014 HRS/AHD.

| Variable                          | Full sample | White females | White males | Black females | Black males | Hispanic females | Hispanic males | Sig
|-----------------------------------|-------------|---------------|-------------|---------------|-------------|-----------------|----------------|------
| Age                               | 75.03       | 75.58         | 75.01       | 74.35         | 73.78       | 73.97           | 73.45          | *** |
|                                   | (7.13)      | (7.31)        | (6.90)      | (7.31)        | (6.59)      | (6.92)          | (6.37)         |      |
| Cognition Scores (mean)           | 21.08       | 22.08         | 21.60       | 17.96         | 17.44       | 17.99           | 18.66          | *** |
|                                   | (5.58)      | (5.37)        | (4.97)      | (6.17)        | (5.66)      | (5.71)          | (5.33)         |      |
| Psychiatric history (%)           |             |               |             |               |             |                 |                |      |
| Longer Term History               | 14.46       | 17.62         | 9.58        | 14.68         | 9.08        | 25.23           | 10.72          | *** |
| Current Treatment                 | 2.43        | 2.71          | 1.81        | 2.12          | 2.24        | 4.94            | 2.34           |      |
| Incident Case                     | 2.76        | 3.20          | 2.11        | 2.71          | 1.96        | 4.07            | 2.64           |      |
| Childhood Disadvantage Index (mean) | 0.37       | 0.33          | 0.32        | 0.52          | 0.56        | 0.63            | 0.64           | **  |
|                                   | (0.33)      | (0.32)        | (0.31)      | (0.35)        | (0.35)      | (0.34)          | (0.33)         |      |
| Childhood Disadvantage Index (%)  | 31.38       | 34.83         | 35.55       | 19.82         | 16.61       | 13.46           | 11.48          | *** |
|                                   | 11.75       | 7.82          | 7.79        | 22.81         | 22.70       | 32.17           | 33.49          | *** |
| Subjects                          | 20,155      | 8,996         | 6,745       | 1,747         | 1,068       | 945             | 654            |      |
| Percent of Sample                 | 100.00      | 46.52         | 33.13       | 8.28          | 4.62        | 4.53            | 2.93           |      |

Note: Values of each characteristic for White females are compared to the rest of the sample, and the remaining sex and race/ethnicity groups are compared to White women.

*p<.05; **p<.001; ***p<.0001.
mean cognition score for the sample was 21.08 (range 0–35), well above the cut-off score of 8 indicating severe impairment. White females (reference group: 22.08, \( p < .0001 \)) had significantly higher cognition scores than the rest of the sample. Compared to White females, White males (21.60, \( p < .0001 \)), Black females (17.96, \( p < .0001 \)), Black males (17.44, \( p < .05 \)), Hispanic females (17.99, \( p < .0001 \)), and Hispanic males (18.66, \( p < .0001 \)) had significantly lower cognition scores. White females (17.62%) were significantly less likely than Hispanic females (25.23%, \( p < .0001 \)) to report a history of psychiatric problems, but significantly more likely than White males (9.58%, \( p < .0001 \)), Black males (9.08%, \( p < .0001 \)), Black females (14.68%, \( p < .0001 \)) and Hispanic males (10.72%, \( p < .0001 \)). Within each racial/ethnic group, larger proportions of females reported a history of psychiatric problems relative to males—17.62% of White females compared to 9.58% of White males, 14.68% of Black females compared to 9.08% of Black males, and 25.23% of Hispanic females compared to 10.72% of Hispanic males.

Multi-level growth curve models (Table 2) showed that a self-reported history of psychiatric, emotional, or nervous problems was associated with cognition scores at age 75 (\( \beta = -0.53, SE = 0.06, p < .0001 \)) and as participants aged (\( \beta = -0.02, SE = 0.01, p < .0001 \)). Participants who reported currently being treated for psychiatric, emotional, or nervous problems also had lower cognition scores (\( \beta = -0.37, SE = 0.09, p < .0001 \)); however, incident (newly identified) cases of psychiatric, emotional, or nervous problems had higher cognition scores (\( \beta = 0.23, SE = 0.08, p < .001 \)). Finally, participants reporting more childhood disadvantage (higher CDI) had significantly lower cognition scores (\( \beta = -0.57, SE = 0.09, p < .0001 \)) at age 75. Analysis also showed substantively larger differences in cognition function based on SRE, independent of psychiatric history, when controlling for childhood disadvantage and health, adult socioeconomic status, later-life health and health behaviors, and attrition. For Whites only, being female was protective of cognitive function scores compared with males (\( \beta = -0.84, SE = 0.06, p < .0001 \)). Hispanic participants had lower cognitive scores compared to White females at age 75 (\( \beta = -2.34, SE = 0.13, p < .0001 \)). The race/ethnicity-sex interaction effect for Hispanic males (\( \beta = 0.85, SE = 0.20, p < .0001 \)) cancelled out the negative effect of being male (\( \beta = -0.84, SE = 0.06, p < .0001 \)), resulting in cognition trajectories for males that were almost identical to those of Hispanic females. The race/ethnicity-sex interaction effect for Black males was nonsignificant (\( \beta = 0.29, SE = 0.16, p = 0.07 \)); however, the effect of race was larger for Blacks than for Hispanics (\( -3.25, vs. -2.34, p < .0001 \)), and added to the negative effect of being male (\( \beta = -0.84, SE = 0.06, p < .0001 \)) for Black participants.

When plotting prediction lines based on these multi-level growth curve models, we found substantively and statistically significant differences in cognition scores between White females with no history of psychiatric problems and Black females (4.15 points lower) and males (4.70 points lower) with a history of psychiatric problems at age 75 (Figure 1). The cognition scores of any participants with a history of psychiatric problems declined more steeply (\( \beta = -0.84, SE = 0.01, p < .01 \)) with each additional year, resulting in greater differences in cognition scores at age 85 between White females with no history of psychiatric problems and Black females (4.36) and males (4.91) with this history.

We also found statistically significant differences, albeit smaller, in cognition scores between White females and Hispanic females (3.24 points lower) and males (3.23 points lower) with a history of psychiatric problems at age 75 (not displayed). Participants with a history of psychiatric problems declined more sharply with each additional year, resulting in greater differences in cognition scores at age 85 between White females with no history of psychiatric problems and Hispanic females (3.45) and males (3.44) with this history. These associations persist with the addition of controls for later-life SES, health status, health behaviors, depressive symptoms, and attrition, loss to follow-up, and death.

**Discussion**

To the best of our knowledge, this is the first study to examine the association between psychiatric history and cognitive decline trajectories while accounting for the intersectionality of race and sex. Overall, we found that participants with a history of psychiatric, emotional, or nervous problems had lower cognition scores at baseline and steeper declines as they aged. We further observed differences in outcomes at the intersection of race/ethnicity and sex, with minorities faring worse. Similar to previous studies we found racial disparities in cognitive decline, with the greatest disparities among Black older adults and those who have a history of psychiatric problems (Barnes et al., 2011; Diaz-Venegas et al., 2016). Our findings further contribute to current literature by demonstrating that gender is protective of cognitive function for White women only.

Our findings indicated that participants with chronic health conditions such as diabetes and stroke have lower cognitive functioning. Psychiatric history has also been associated with these and other chronic health outcomes which may lead to variations in pace of cognitive decline with potential SRE

### Table 2. Multi-level growth curve model of cognitive function, 1995–2014 HRS/AHD.

| Fixed effects                                      | Estimate | SE    | \( p \)-value | Sig. |
|---------------------------------------------------|----------|-------|---------------|-----|
| **For Initial Level (at age 75)**                 |          |       |               |     |
| Intercept                                         | 22.3011  | 0.12  | <.0001        | *** |
| White males (reference group: White females)      |          |       |               |     |
| Black females                                     | -3.25    | 0.10  | <.0001        | *** |
| Black males                                       | 0.29     | 0.16  | <.0001        | *** |
| Hispanic females                                  | -2.34    | 0.13  | 0.0657        | *** |
| Hispanic males                                    | 0.85     | 0.20  | <.0001        | *** |
| Childhood Disadvantage Index (CDI)                | -0.57    | 0.09  | <.0001        | *** |
| Poor childhood health                            | -0.33    | 0.11  | 0.0038        | **  |
| Missing on childhood                              | -1.30    | 0.09  | <.0001        | *** |
| Longer-term psychiatric history                   | -0.53    | 0.06  | <.0001        | *** |
| Current psychiatric treatment                     | -0.37    | 0.09  | <.0001        | *** |
| Incident psychiatric problems                     | 0.23     | 0.08  | 0.0031        | **  |
| Rate of change                                    | -0.26    | 0.00  | <.0001        | *** |
| Age\(^a\)                                         | -0.01    | 0.00  | <.0001        | *** |
| Psychiatric history                               | -0.02    | 0.01  | 0.0024        | **  |
| Random effects                                    |          |       |               |     |
| Between-Person Variation                          | 10.04    | 0.16  | <.0001        | *** |
| Intercept/Slope Covariance                        | 0.22     | 0.01  | <.0001        | *** |
| Linear Change                                     | 0.04     | 0.00  | <.0001        | *** |
| Autoregression                                    | 0.10     | 0.01  | <.0001        | *** |
| Within-Person Variation                           | 9.65     | 0.07  | <.0001        | *** |
| Participants                                      | 20 155   |       |               |     |
| Observations Used                                 | 90 966   |       |               |     |

Includes controls for later-life socioeconomic status (household income and educational attainment), marital status, health status (subjective health, hypertension, diabetes, heart disease, stroke, vision and hearing impairment), health behaviors (currently smokes, ever drinks alcohol), depressive symptoms, attribution, death, and loss to follow-up.

\(^{a}p<.05; {^{**}p<.001; {^{***}p<.0001.\)
differences (Levine et al., 2021; Marden et al., 2017). For example, comorbid chronic conditions which are known risk factors for cognitive decline disproportionately affect minority women, with greater severity and challenges in accessing quality care (Centers for Disease Control and Prevention & National Association of Chronic Disease Directors, 2020; Chinn et al., 2021). Our findings alluded to poorer cognitive functioning for participants who reported poor childhood health, greater childhood disadvantage and less education, while higher education (i.e. more than high school) was protective of low cognitive functioning scores. These factors reflect some of the social structures and processes that affect the distribution of additional risk factors and apportion disadvantage to Black communities starting in early life through exposures to negative psychosocial, emotional, and physical childhood experiences, which accumulate over time and further impact adult SES and physical and mental health, factors associated with cognitive health.

Greater attention to how historical, economic, political, and cultural social structures and processes influence health-improving or harming factors across the life course is needed to fully comprehend the observed disparities in cognitive health. Social stressors that minorities are uniquely exposed to, access to resources, and discrimination are important factors needing further exploration to more comprehensively understand disparities in cognitive health outcomes. This will require a greater emphasis on a life-course approach that seeks to understand the ways in which resources and adversity accumulate over time to affect adult health. It will also require greater attention to stressors uniquely experienced by minorities.

Our finding of higher cognitive scores in those with new reports of psychiatric history is counterintuitive. We cannot rule out that this finding may have been impacted by reporting bias and respondents understanding of the question, as our HRS measurement for psychiatric history was based on self-report, non-specific (i.e. broad and all-inclusive), and has not been validated against a clinical diagnosis (Holden et al., 2015). Further, previous research has alluded to heterogeneity in cognitive decline among those with clinically diagnosed psychiatric disorders (e.g. bipolar disorder; Millett & Burdick, 2021). Our measure unfortunately does not distinguish between different types of disorders, and it is likely that we may have observed findings that align with the literature, had we used clinical data and assessed each psychiatric disorder separately (Brown & Wolf, 2018; Gildengers et al., 2009; Montego et al., 2022). Future research should validate this measure against clinical diagnosis and further examine incident cases of psychiatric diagnosis and cognition by specific disorders.

**Limitations**

These findings should be viewed in light of several limitations. First, there are possible confounding effects in the TICS-m instrument, including assumptions made about non-response on individual items, although this effect was considered primarily in relationship to data collection in later waves, and the possibility of differing reasons for non-response between waves. Previous studies of the AHEAD cognition data have ruled out the possibility of mode effects or bias being introduced by using telephone interviews with older populations (Herzog & Wallace, 1997). Second, self-reports of psychiatric history may be vulnerable to underreporting due to social stigma, or to overreporting due to the broad range of conditions covered by the question, resulting in either under- or over-estimation of the strength of association between the variables of interest. Third, cognitive functioning can be affected by different psychiatric disorders. The prevalence rates for severe and persistent psychiatric disorders like schizophrenia (1.11%), bipolar disorder (2.64%), major depressive disorder (4.19%), are lower than what we found in our sample for the broader category of “psychiatric, emotional, or nervous problems” that is report on in the HRS (Brown & Wolf, 2018). Specific disorder data are not available in the HRS. We were therefore unable to examine the impact of different psychiatric disorders on cognitive trajectories over time. Finally, there is potential cultural bias in instruments measuring cognition across racial and ethnic groups. The TICS-m instrument has been previously assessed for cultural bias and it has been determined that any existing bias does not eliminate racial disparity in cognition scores (Jones, 2003). Despite these limitations, our findings were robust and benefited from the use of a large representative cohort with 20 years of follow-up data.

**Conclusions**

Our findings indicate that members of minority groups with a history of psychiatric problems evidence lower cognitive function in later life, and as a result, have a greater need for community-based long-term care than their peers without this history. Future research should include longitudinal analyses of different components of cognitive function, specific psychiatric diagnoses, and life history data that capture socioeconomic and psychosocial experiences throughout the life course. The findings of these supplemental studies along with the current evidence may provide insights to reduce disparities in both conditions and inform clinical care, and public health interventions.

**Acknowledgements**

We appreciate the instrumental support of the Falk College of Sport and Human Dynamics and the Aging Studies Institute at Syracuse University.

**Disclosure statement**

The authors report there are no competing interests to disclose.

**Funding**

The author(s) reported there is no funding associated with the work featured in this article.

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