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Research Article

Cumulative Stress Exposure and Cognitive Function Among Older Adults: The Moderating Role of a Healthy Lifestyle

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Abstract

Objectives: Although chronic stress is a risk factor for poor age-related cognitive health, there is limited research that has examined how cumulative stress across the lifespan affects cognitive aging. There may also be resilience factors that minimize the effects of cumulative stress on cognitive health. Engaging in a healthy lifestyle is protective against cognitive decline and may therefore interact with cumulative stress to buffer the stress–cognition relationship. The objective of the current study was to examine the moderating role of a healthy lifestyle, comprised of physical activity, social engagement, and sleep quality, in the relationship between cumulative stress exposure (CSE) and baseline and change in cognitive performance (global cognition, episodic memory, executive function) over 9 years among 1,297 older adults in the Midlife in the United States cohort (M_{age} = 69.0 ± 6.4, 57.8% female).

Methods: CSE and healthy lifestyle behaviors were indexed using self-reported questionnaires at baseline, and cognitive function was assessed using a battery of standardized neuropsychological tests at baseline and follow-up.

Results: Controlling for age, sex, education, race, marital status, employment status, hypertension, diabetes, and depression, higher CSE was associated with poorer baseline performance and slower decline over time in global cognition and executive function, but not episodic memory. A healthy lifestyle did not significantly moderate the relationship between cumulative stress and cognitive function. Exploratory analyses showed a significant cumulative stress–cognition relationship among females only.

Discussion: This study lends support for a lifespan model of cognitive aging and suggests that the cognitive health consequences of stress extend beyond immediate timescales.

Keywords: Brain health, Cognitive aging, Life course, Lifestyle, Stress

With an aging population on the rise, understanding the factors that facilitate healthy cognitive aging and reduce the risk of dementia is an urgent public health priority. In the absence of an effective disease-modifying treatment for dementia, research has largely shifted toward risk factor management, with the intention of delaying the onset of cognitive deterioration (Rockwood et al., 2020). A substantial body of work suggests that cognitive aging trajectories are largely explained by the interaction between modifiable risk and protective factors over the life course (Livingston et al., 2020). Psychological stress is one such modifiable risk factor for age-related cognitive decline that warrants attention due to its high prevalence rates and potential for intervention (Franks et al., 2022).

Decades of prior research have shown that high levels of chronic stress exert detrimental effects on brain health (Lupien et al., 2009), especially among older adults who are particularly vulnerable to the effects of stress on cognition (Sapolsky, 1999). Most studies to date have assessed perceptions of stress within the previous month, with mixed findings reported (e.g., Solder et al., 2021; Turner et al., 2017). This restricted time frame neglects stressful experiences accrued throughout the lifespan, which fails to capture the chronic and cumulative nature of stress that is central to its effects on health (Shields & Slavich, 2017). Understanding how psychological stress affects cognitive aging may be better conceptualized using a life course perspective. Indeed, multiple stressors accumulate and cluster over the lifespan such that, as the severity and duration of stressors increase, there is cumulative wear and tear on the brain and body (Ben-Shlomo & Kuh, 2002), leading to negative health consequences, including an increased risk for age-related cognitive decline and impairments (D'Amico et al., 2020a). Moreover, both acute and chronic stress exposures across multiple life domains should be assessed when measuring cumulative stress across the lifespan, as no single stressor can capture the full impact of stress on health (Pearlin et al., 2005; Wheaton, 1994). Taking a life course approach is especially important when examining the antecedents of cognitive health as factors that enhance

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or diminish cognitive resilience accumulate over different periods across the lifespan (Livingston et al., 2020). Mixed findings have been reported, however, on the association between cumulative life stress and cognitive function, suggesting that there may be individual difference factors that influence the relationship between stress and cognition. For example, Peavy et al. (2009) found that a greater number of stressful life events were associated with greater age-related cognitive decline, while Senft Miller et al. (2021) did not find a relationship between cumulative stress exposure (CSE) and cognitive function.

Not all individuals are equally affected by the same exposures to stress (Lupien et al., 2018), implying that there may be protective factors rendering some individuals better able to withstand the degenerative effects of stress on the brain. Additionally, the effects of stress on cognitive function may be malleable, evidenced by the brain's ability to adapt to environmental changes (McEwen & Morrison, 2013). It is therefore important to examine factors that may interact with stress to minimize its negative impact on cognition. Engaging in a healthy lifestyle may be a protective mechanism through which the negative effects of stress on cognitive aging can be attenuated.

A large and growing body of literature provides considerable evidence that healthy lifestyle behaviors, such as physical activity, social engagement, and getting good quality sleep, are modifiable factors that reduce the risk of age-related cognitive decline (Livingston et al., 2020). Considering the cumulative and combined effect of multiple healthy lifestyle behaviors on cognitive health, as opposed to individual behaviors in isolation, may be more practical as healthy lifestyle parameters often cluster and act synergistically to benefit cognitive function (Rabel et al., 2019). Engaging in healthy lifestyle behaviors confers cognitive benefits, in part, by enhancing cognitive reserve, or the ability to remain cognitively intact despite the presence of risk factors (Stern et al., 2019). Further, physical activity, social engagement, and good sleep hygiene are coping behaviors that have all been shown to reduce feelings of stress (Churchill et al., 2022). A healthy lifestyle may be a plausible explanation for the individual differences seen in the stress–cognition relationship, such that engaging in a healthy lifestyle may buffer the association between CSE and cognitive function among older adults (Lupien et al., 2018). To date, however, no studies have directly evaluated the moderating role of a healthy lifestyle in this relationship.

The current study examined the moderating effect of a healthy lifestyle on the association between cumulative stress over the lifespan and cognitive functioning in later adulthood. Specifically, this study examined whether a healthy lifestyle indicator (HLI) score, including physical activity, social engagement, and sleep quality, moderates the association between greater CSE and cognitive function in domains of episodic memory, executive functioning, and global cognition, at baseline and over 9 years among older adults. It was hypothesized that a higher HLI score would buffer the negative association between CSE and cognition at baseline and over time. Sex-stratified models were also conducted to explore whether the aforementioned associations differ by sex. Additional models were conducted to explore the moderating effect of individual components of the HLI score.

Method

Participants

Data for this study were drawn from the second and third waves of the National Survey of Midlife in the United States (MIDUS), a longitudinal cohort study designed to investigate the biopsychosocial factors associated with physical, mental, and cognitive health in middle-aged and older adulthood (Radler & Ryff, 2010). For the current study, MIDUSII was considered the baseline. From the 5,555 individuals who participated in MIDUSII, 1,089 were removed for not having completed the questionnaires pertaining to stress exposures across the lifespan and lifestyle behaviors. A total of 3,045 individuals were then excluded from analyses for meeting the following self-reported a priori exclusion criteria at MIDUSII: age less than 60, diagnosis of a neurological disorder, Parkinson's disease, a history of stroke, a history of a serious head injury, and/or having previously undergone chemotherapy or radiation treatment. Participants were then removed if they were missing information on age, sex, and/or educational attainment (n = 2). Participants were also excluded from the final analytical sample if they did not participate in the MIDUSII Cognitive Project or were missing all baseline cognitive assessments (n = 113). This resulted in a final analytical sample of 1,297. Of these participants, 806 completed the follow-up cognitive assessment at MIDUSIII (mean follow-up time: 9.2 ± 0.5 years). Those who were lost to follow-up were more likely to be older, male, have less than at least some postsecondary education, have higher levels of baseline CSE, have lower baseline healthy lifestyle adherence, and have poorer baseline global cognition, episodic memory, and executive function performance (see Supplementary Table 1). Supplementary Figure 1 provides a flowchart of the study sample.

MIDUS data collection was reviewed and approved by the Education and Social/Behavioural Sciences and the Health Sciences Institutional Review Boards at the University of Wisconsin–Madison. Ethics approval for secondary data analysis was approved by Toronto Metropolitan University's Research Ethics Board (REB 2021–385).

Measures

Sociodemographic and health-related information

The following self-reported variables were collected at baseline: age; sex (male or female); highest level of education attained; race self-identified as Black/African American, White, or other; marital status (married or not married); employment status (currently working or not currently working); diagnosis of diabetes, hypertension, and depression (yes/no); perceived socioeconomic status indexed using the MacArthur Scale of Subjective Social Status (Adler et al., 2000); and informal caregiving status (yes/no). Participants were also asked if they currently smoke cigarettes regularly (yes/no), their frequency of alcohol intake within the previous month ranging from 0 (*never*) to 5 (*everyday*), and their self-reported memory abilities compared to others of their same age on a scale ranging from 1 (*poor*) to 5 (*excellent*).

Cumulative stress exposure

Following previous MIDUS research (Chen et al., 2022), CSE was indexed using the following 10 stress domains assessed via self-reported questionnaires at MIDUSII: childhood stress, adulthood stress, financial stress, relationship stress,

work–family conflict, neighborhood stress, work-related psychological stress, work-related physical stress, perceived inequality, and perceived discrimination. Higher scores on each domain subscore indicate higher levels of stress, except for work-related physical stress and work–family conflict, where lower scores are indicative of greater stress. Accordingly, subscores for work-related physical stress and work–family conflict were multiplied by −1 so that higher scores on all stress domains indicate higher levels of stress. For the domains that were not applicable to a given participant (e.g., occupational stress for individuals who were unemployed, marital stress for single individuals, and child-related measures for participants without children), the lowest possible value on the item was assigned. The total CSE score was calculated by standardizing each of the 10 stress domain subscores into a *Z*-score and summing the *Z*-scores, with higher total scores representing greater CSE across the lifespan. See Supplementary Material for additional details on the measure items and scoring algorithm used to create the CSE composite score.

HLI score

Physical activity, social engagement, and sleep quality were assessed via self-reported questionnaires at MIDUSII. Total scores for both physical activity and social engagement were calculated using methods derived from previous MIDUS studies (Cotter & Lachman, 2010; Tun et al., 2013). Total sleep quality was assessed using the Jenkins Sleep Questionnaire (Jenkins et al., 1988). Further details on each questionnaire and individual lifestyle behavior scoring method are included in the Supplementary Material. To create the moderating variable, each of the three lifestyle behavior scores was converted into a *Z*-score and summed to create a total HLI score, with higher scores representing greater adherence to a healthy lifestyle.

Cognitive function

Cognitive function was assessed at MIDUSII and MIDUSIII using the Brief Test of Adult Cognition by Telephone (BTACT; Tun & Lachman, 2006), a battery of neurocognitive tasks designed to measure seven areas of cognitive functioning that are sensitive to aging. These tasks included the Rey Auditory–Verbal Learning Test to assess immediate and delayed episodic memory; and the backward digit span (working memory span), the category fluency test (verbal fluency), the number series completion task (inductive reasoning), the backwards counting task (speed of processing), and the Stop and Go Switch Task (attention switching) to assess executive function. For a detailed description of the test battery administration, see Tun and Lachman (2006). The BTACT has demonstrated good convergent validity compared to the in-person version among participants in the MIDUS sample (Lachman et al., 2014). The Supplementary Material includes scoring details for global cognition, episodic memory, and executive function composite scores at baseline and follow-up.

Statistical Analyses

All analyses were performed using R (R Core Team, 2023). A nominal Type I error rate of $\alpha = 0.05$ was used as a threshold for statistical significance in all analyses (i.e., $p < .05$). Missing responses for CSE (0.1%–15.0% missing) and healthy lifestyle behavior (0.5%–15.5% missing) items were imputed using maximum likelihood multiple imputations with five imputations and 10 iterations using the "mice" package (van Buuren et al., 2021). All analyses were conducted using the pooled imputed data set.

A total of three primary linear mixed-effects models were conducted using the "lme4" package in R (Bates et al., 2022) to examine the moderating effect of the HLI score on the relationship between CSE and cognitive function over time. The robust Kenward–Roger estimation (Kenward & Roger, 1997) was used for more precise standard errors. Time was coded linearly as 0 = baseline and 1 = follow-up. Each model was analyzed with CSE as the independent variable, the HLI as the moderating variable, and either global cognition, episodic memory, or executive function composite score as the dependent variable. Cumulative stress, the HLI, and all covariates were entered as fixed-effect factors and participant-specific intercepts were specified as the random effect. All models were adjusted a priori for age, sex, and educational attainment. Fully adjusted models included additional covariates of race (White or not White), marital status, employment status, depression, hypertension, and diabetes due to their known influence on psychological stress, lifestyle behaviors, and cognitive function (Jin et al., 2023; Moheet et al., 2016). All variables were standardized to *Z*-scores before being added into the models. R^2 model fit statistics for mixed models (Rights & Sterba, 2019) were derived separately for each imputed data set and then averaged across all imputed data sets for a total pooled R^2 statistic for each model.

Data were disaggregated by sex to explore whether the moderating role of an HLI in the relationship between CSE and cognitive change differs between males and females. To explore whether the moderating role of a healthy lifestyle may be driven by specific lifestyle behaviors, each individual moderating lifestyle behavior (i.e., physical activity, social engagement, and sleep quality) was explored independently.

Results

Participant Characteristics

A full summary of participant sociodemographic and health-related characteristics, including descriptive information about CSE, healthy lifestyle behaviors, and standardized baseline cognitive performance scores are shown in Table 1. Briefly, participants were, on average, 69 ± 6.4 years of age and 57.8% of the sample was female. The majority of the sample self-identified as White (89.1%), 57.5% had at least some postsecondary education, and perceived socioeconomic position was moderate with a mean score of 4.1 ± 3.1 out of a possible score of 10. Fifteen percent of the sample reported a diagnosis of diabetes, 45.3% reported a diagnosis of hypertension, and 13.5% reported a diagnosis of depression. See Supplementary Table 2 for the bivariate correlations between the sociodemographic and health-related variables, CSE, HLI score and its components, and cognitive function composite scores at baseline.

Moderation Models

Global cognition

In the partially adjusted model controlling for age, sex, and education, greater CSE was associated with poorer baseline global cognition ($B = -0.20$, 95% confidence interval [CI; −0.25, −0.14]), and lower decline over time ($B = 0.08$, 95% CI [0.02, 0.14]). The HLI score was not significantly associated with baseline cognition ($B = 0.02$, 95% CI [−0.10,

Table 1. Participant Sociodemographic and Health-Related Characteristics (n = 1,297)

	Mean ± *SD* or % (*n*)
Age in years at baseline	69.0 ± 6.4
Sex (% female)	57.8 (750)
Race (%)	
Black and/or African American	7.6 (98)
White	89.1 (1,151)
Other	3.3 (43)
Educational attainment (%)	
Less than high school	9.7 (125)
High school (or equivalent)	32.9 (427)
Some college	20.8 (269)
College diploma or associate degree	6.3 (82)
Bachelor's degree	13.9 (180)
Some graduate school	3.4 (44)
Master's degree	9.1 (118)
Doctoral or professional degree	4.0 (52)
Marital status (% married)	65.6 (851)
Employment status (% employed)	33.6 (432)
Diabetes (% yes)	15.2 (197)
Hypertension (% yes)	45.3 (587)
Depression (% yes)	13.5 (175)
Perceived socioeconomic position	4.1 ± 3.1
Self-reported memory abilities (%)	
Poor	0.8 (10)
Fair	9.2 (118)
Average	32.8 (420)
Good	45.2 (580)
Excellent	12.0 (154)
Current smoking (% yes)	13.6 (134)
Alcohol intake	
Everyday	8.0 (94)
5–6 days/week	4.7 (55)
3–4 days/week	7.0 (83)
1–2 days/week	12.3 (145)
<1 day/week	23.2 (274)
Never	44.8 (528)
Caregiver status (% yes)	12.7 (164)

	Z-score range
CSE	−9.32 to 24.13
Childhood stress	−0.73 to 11.74
Adulthood stress	−1.28 to 9.89
Financial stress	−1.24 to 2.48
Work-related psychological stress	−2.78 to 3.22
Work-related physical stress	−2.86 to 1.08
Neighborhood stress	−0.95 to 6.43
Work–family conflict	−3.44 to 1.74
Relationship stress	−0.91 to 7.91
Perceived inequality	−1.41 to 5.54
Perceived discrimination	−0.66 to 6.58
HLI	−6.50 to 4.71
Physical activity	−1.71 to 1.06
Social engagement	−3.35 to 3.68
Sleep quality	−2.89 to 1.65
MIDUSII global cognition composite	−16.57 to 14.30
MIDUSII episodic memory composite	−3.58 to 7.40
MIDUSII executive function composite	−15.12 to 10.16

Notes: CSE = cumulative stress exposure; HLI = healthy lifestyle indicator; MIDUS = Midlife in the United States; *SD* = standard deviation.

0.14]) or change over time (B = 0.03, 95% CI [−0.09, 0.14]). Moderation analyses were not statistically significant for baseline performance (B = −0.01, 95% CI [−0.04, 0.02]) or global cognition over time (B = −0.006, 95% CI [−0.04, 0.03]). Results were similar in the fully adjusted model. See Table 2 for all model estimates.

Episodic memory

In the partially adjusted model, greater CSE was associated with poorer baseline episodic memory (B = −0.05, 95% CI [−0.08, −0.03]), but not with change in episodic memory (B = 0.006, 95% CI [−0.03, 0.04]). The HLI score was not significantly associated with baseline episodic memory (B = 0.02, 95% CI [−0.10, 0.14]) or change in episodic memory (B = 0.04, 95% CI [−0.04, 0.11]). No statistical evidence was found in the CSE × HLI interaction for episodic memory at baseline (B = 0.001, 95% CI [−0.01, 0.01]) or change in episodic memory (B = −0.003, 95% CI [−0.02, 0.02]). Results were similar in the fully adjusted model. See Table 2 for all model estimates.

Executive function

In the partially adjusted model, greater CSE was associated with poorer baseline executive function (B = −0.14, 95% CI [−0.19, −0.10]) and less decline over time (B = 0.08, 95% CI [0.04, 0.12]). HLI was not significantly associated with baseline (B = 0.001, 95% CI [−0.09, 0.09]) or change in executive function (B = −0.006, 95% CI [−0.09, 0.08]). No statistically significant CSE × HLI interaction was found for baseline executive function (B = −0.01, 95% CI [−0.03, 0.009]) or change in executive function over time (B = 0.001, 95% CI [−0.02, 0.03]). Results were similar in the fully adjusted model. See Table 2 for all model estimates.

Exploratory Analyses

When stratifying the fully adjusted primary models by sex, CSE was associated with baseline global cognition (B = −0.16, 95% CI [−0.23, −0.09]), episodic memory (B = −0.05, 95% CI [−0.08, −0.01]), and executive function (B = −0.11, 95% CI [−0.17, −0.06]) among females but not males. Among females only, lower levels of baseline CSE were associated with faster rates of decline in global cognition decline (B = 0.10, 95% CI [0.03, 0.18]) and executive function (B = 0.09, 95% CI [0.04, 0.14]). No statistically significant associations were found between the HLI score and baseline cognition or cognitive change among males or females in any cognitive domain. Similarly, no significant CSE × HLI interaction was found on baseline cognition or change in cognition among males or females. See Supplementary Table 3 for all sex-stratified

Table 2. Linear Mixed-Effect Model Estimates Predicting Baseline and 9-Year Change in Cognition

	Partially adjusted models[a]				Fully adjusted models[b]			
	B	*SE*	*p* Value	95% CI	*B*	*SE*	*p* Value	95% CI
Global cognition	R^2 = 0.26				R^2 = 0.30			
CSE	−0.20	0.03	**<.001**	**−0.25, −0.14**	−0.14	0.03	**<.001**	**−0.20, −0.0**
HLI	0.02	0.06	.73	−0.10, 0.14	0.02	0.06	.76	−0.10, 0.13
Time	−1.47	0.11	**<.001**	**−1.68, −1.27**	−1.50	0.11	**<.001**	**−1.70, −1.29**
CSE × HLI	−0.01	0.01	.43	−0.04, 0.02	−0.006	0.01	.64	−0.03, 0.02
CSE × Time	0.08	0.03	**.008**	**0.02, 0.14**	0.08	0.03	**.009**	**0.02, 0.14**
HLI × Time	0.03	0.06	.66	−0.09, 0.14	0.03	0.06	.61	−0.09, 0.15
CSE × HLI × Time	−0.006	0.02	.74	−0.04, 0.03	−0.008	0.02	.65	−0.04, 0.03
Episodic memory	R^2 = 0.18				R^2 = 0.18			
CSE	−0.05	0.01	**<.001**	**−0.08, −0.03**	−0.04	0.01	**<.001**	**−0.07, −0.02**
HLI	0.02	0.03	.49	−0.04, 0.07	0.02	0.03	.47	−0.03, 0.08
Time	−0.44	0.06	**<.001**	**−0.56, −0.31**	−0.44	0.06	**<.001**	**−0.56, −0.31**
CSE × HLI	0.001	0.007	.93	−0.01, 0.01	0.001	0.007	.93	−0.01, 0.01
CSE × Time	0.006	0.03	.74	−0.03, 0.04	0.008	0.03	.67	−0.03, 0.04
HLI × Time	0.04	0.04	.32	−0.04, 0.11	0.04	0.04	.31	−0.03, 0.11
CSE × HLI × Time	−0.003	0.01	.76	−0.02, 0.02	−0.003	0.01	.75	−0.02, 0.02
Executive function	R^2 = 0.25				R^2 = 0.30			
CSE	−0.14	0.02	**<.001**	**−0.19, −0.10**	−0.10	0.02	**<.001**	**−0.14, −0.06**
HLI	0.001	0.05	.98	−0.09, 0.09	−0.002	0.04	.96	−0.09, 0.08
Time	−1.02	0.07	**<.001**	**−1.16, −0.87**	−1.04	0.07	**<.001**	**−1.19, −0.90**
CSE × HLI	−0.01	0.01	.27	−0.03, 0.009	−0.007	0.01	.50	−0.03, 0.01
CSE × Time	0.08	0.02	**<.001**	**0.04, 0.12**	0.08	0.02	**<.001**	**0.03, 0.12**
HLI × Time	−0.006	0.04	.88	−0.09, 0.08	−0.003	0.04	.94	−0.09, 0.08
CSE × HLI × Time	0.001	0.01	.93	−0.02, 0.03	−0.001	0.01	.95	−0.03, 0.02

Notes: Bolded values indicate statistical significance at $p < 0.05$. *B* = unstandardized regression coefficient; CI = confidence interval; CSE = cumulative stress exposure; HLI = healthy lifestyle indicator; *SE* = standard error.
[a]Adjusted for age, sex, and educational attainment.
[b]Adjusted for age, sex, educational attainment, race, marital status, employment status, depression, hypertension, and diabetes.

model estimates. Figure 1A–C displays change in cognition (A, global cognition; B, episodic memory; C, executive function) at low, moderate, and high levels of CSE among males and females.

In exploring the moderating effect of individual lifestyle behaviors in the relationship between CSE and cognitive function, higher levels of social engagement were associated with higher baseline global cognition (*B* = 0.34, 95% CI [0.14, 0.54]), episodic memory (*B* = 0.13, 95% CI [0.04, 0.23]), and executive function (*B* = 0.21, 95% CI [0.06, 0.35]). Better sleep quality was associated with lower baseline executive function scores (*B* = −0.19, 95% CI [−0.34, −0.04]). Physical activity, social engagement, and sleep quality were not significantly associated with cognitive change over time, nor moderated the association between CSE and baseline cognition or change in cognition over time. See Supplementary Tables 4–6 for the model estimates with physical activity, social engagement, and sleep quality as the moderator, respectively.

Discussion

The purpose of the current study was to examine if engagement in multiple healthy lifestyle behaviors (physical activity, social engagement, and sleep quality) moderated the relationship between stress exposure accumulated over the life course and cognitive function among older adults in the MIDUS cohort. As expected, greater CSE was associated with poorer cognitive performance at baseline. However, greater CSE was associated with less decline in global cognition and executive function over time. Finally, the results did not support the hypothesized moderating effect of an HLI score on the association between CSE and cognition. Exploratory analyses provided some support for the importance of disaggregating data by sex and stimulate discussion for the use of an HLI score.

The association found between higher levels of CSE and poorer cognitive performance is consistent with prior research showing a relationship between psychological stress and cognitive function among older adults (Peavy et al., 2009), as well as theoretical conjectures pertaining to the effects of accumulation of risk over time on health outcomes (Ben-Shlomo & Kuh, 2002). This study adds to the extant body of work by highlighting the need to consider a constellation of stressful exposures across the life course when examining the stress–cognition relationship. There are a number of mechanisms that may explain the association between higher levels of stress across the lifespan and poorer cognitive performance in older adulthood. In particular, chronically elevated glucocorticoids (i.e., cortisol) released by the hypothalamic–pituitary–adrenal (HPA) axis in response to perceived threats may exert neurotoxic effects on the brain regions most sensitive to age-related changes, including the hippocampus and prefrontal cortex, which are crucial structures that regulate learning

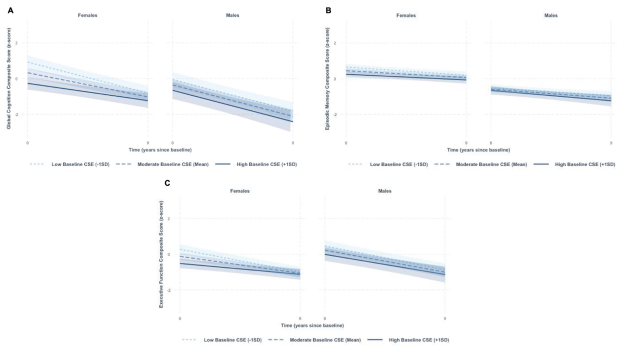

Figure 1. (A–C) Change in global cognition over time at low, moderate, and high levels of CSE among males and females for (A) global cognition, (B) episodic memory, and (C) executive function. CSE = cumulative stress exposure; *SD* = standard deviation. The plots were derived from the model estimates for one of the imputed data sets, which was virtually identical to the pooled results.

and memory and executive functioning (Lupien et al., 2009). Dysregulation of the HPA axis stemming from repeated exposure to chronic stressors over time may further disrupt the functioning of other physiological systems including the cardiometabolic and immune systems, leading to allostatic overload (McEwen, 1998), and eventually adverse health outcomes, including poor cognitive functioning (D'Amico et al., 2020a). Future research is needed to delineate the biopsychosocial mechanisms through which cumulative stress across the lifespan leads to poor cognitive health outcomes in later life.

While the association between cumulative life stress exposure and cognition functioning was statistically significant, the effect sizes were relatively small. Although it is plausible that CSE may account for a small proportion of the variance in age-related cognitive performance, the exposure-based framing of the items that contributed to the calculated composite score may provide an alternative explanation. More specifically, items of the composite score reflected stressor exposure (i.e., whether a discrete event occurred) without considering the degree to which exposure to the event was perceived as stressful. According to the Transactional Model of Stress (Lazarus & Folkman,1984), the degree to which one experiences distress is determined by the extent to which the stressor is evaluated as exceeding one's ability to cope. Measurements of stress that account for subjective appraisals of stress may be stronger predictors of cognitive health outcomes compared to count- or exposure-based measures as they are more sensitive to individual differences in stress appraisals (Hayman et al., 2014). Further research is needed to explore the relative associations between appraisal- versus exposure-based measures of stress and cognitive health.

Although episodic memory declined across the 9-year study period, no association was found with CSE. This is surprising given that episodic memory relies heavily on the functioning of the hippocampus, a glucocorticoid-dense structure that is particularly vulnerable to the neurotoxic effects of chronically high levels of circulating stress hormones (Sapolsky, 1999). However, change in episodic memory over time was relatively small compared to the other cognitive outcomes, possibly leading to less of an ability to detect an effect of stress on episodic memory. Unexpectedly, greater cumulative stress was associated with slower declines in global cognition and executive function over the 9-year follow-up period. Although counterintuitive to what one might expect based on the literature and the study hypotheses, it may be surmised that participants reporting higher cumulative stress displayed a floor effect in cognitive change over time as their cognitive scores were lower at baseline. Moreover, recent findings from the MIDUS study found that higher CSE was not associated with cognitive decline among the entire MIDUS cohort aged 25+ (Chen et al., 2022), suggesting that the nature of the cumulative stress–cognition relationship may be age-dependent. It is also possible that individuals experiencing a greater number of stressful events throughout the lifespan have accrued adaptive coping mechanisms that enhance their resilience to the effects of stress. Indeed, previous research has shown that moderate levels of cumulative lifetime adversity are associated with more favorable health outcomes by building effective coping resources to manage stress (Seery et al., 2010). As these interpretations are simple conjectures, future research is needed to understand the role of stress across the lifespan as a protective mechanism for age-related cognitive decline.

A healthy lifestyle composite comprised of physical activity, social engagement, and sleep quality was not associated with baseline or change in cognition after controlling for potential confounders. This is contrasted with a number of studies showing that higher engagement in a combination of multiple healthy lifestyle behaviors is associated with better cognitive performance (Anastasiou et al., 2018; Mamalaki et al., 2021) and less cognitive decline (Weng et al., 2018) among older adults. One possible explanation for these null findings is that previous studies typically include dietary pattern intake and nutrition as components of an overall healthy lifestyle, while the current study did not. Adherence to a healthy dietary pattern high in fruits, vegetables, lean meats, and healthy fats, and low in processed meats and refined sugars is a key lifestyle behavior that is associated with more favorable cognitive health outcomes (Loughrey et al., 2017) via health-promoting anti-inflammatory and antioxidant pathways (Féart et al., 2010) and reduction in chronic disease associated with cognitive impairment and neurodegeneration (e.g., diabetes, hypertension, and hypercholesterinaemia; Noce et al., 2021). Dietary intake also acts synergistically with other lifestyle behaviors, including physical activity and social engagement to confer cognitive benefits (Fiocco et al., 2012; Parrott et al., 2021). Further, a previous cross-sectional study reported that adherence to a Mediterranean diet moderates the association between perceived stress and cognition in older adults (D'Amico et al., 2020b).

The moderating role of a healthy lifestyle in the association between CSE and cognition was not supported in the current study. As noted above, the exclusion of dietary intake from the composite lifestyle score may have minimized the sensitivity of the moderating variable. Although dietary intake was not available in the core MIDUS study, the healthy lifestyle composite score encompassed three important lifestyle behaviors. Exploratory analyses showed that greater social engagement was associated with better cognitive functioning at baseline, while better sleep score associated with poorer cognitive performance. This may suggest that the contradictive association between social engagement and sleep diluted the sensitivity of the HLI score. It may be possible that poorer sleep quality was associated with better cognitive performance as older

adults with better executive functioning are more cognizant of problemed sleep.

Exploratory analyses provided support for possible effect modification by sex in the cumulative stress–cognition relationship. Specifically, the current study found that greater CSE was associated with baseline cognition and cognitive trajectory only in females. This sex-specific association may have diluted effect estimates stemming from the full-sample analytical models and is contrary to a recent study showing a relationship between higher levels of perceived stress and poorer cognitive function among males, but not females (Paolillo et al., 2022). The study, however, did not measure CSE, but indexed perceptions of stress within the previous month. It may be postulated that, although males may be more sensitive to stress experienced within proximal time frames, females may be more sensitive to the longer-term effects of stress with aging (Wolfova et al., 2021). This sex difference may be due to more stressful experiences across the lifespan reported among females in the current study.

Previous research also suggests that females may be more vulnerable to the biological embedding and proliferation of stress in early life and its impact on age-related cognitive health outcomes (D'Amico et al., 2022). It is important to note that greater levels of CSE were associated with less cognitive decline among females, which, as previously mentioned, may be due to floor effects in cognitive decline or an accrual of coping mechanisms over time that enhance resilience to stress. Although the current sex-specific analyses were exploratory, requiring future hypothesis-driven investigation, this study supports the need for sex- and gender-based analyses in cognitive aging research to better inform individualized recommendations for cultivating healthy brain aging.

Although this study is novel and leverages the data-rich MIDUS study, several limitations must be addressed. First, CSE was measured using retrospective self-reported questionnaires, which likely entailed recall bias, especially when reflecting on experiences in early life. Similarly, lifestyle behaviors were self-reported, which may have resulted in a biased estimate of engagement in healthy behaviors. Furthermore, it is possible that adherence to a healthy lifestyle may change over the lifespan, and that these lifestyle changes may differentially associate with cognitive functioning in later life (Livingston et al., 2020; Middleton et al., 2010). Measures in the current study were limited to engagement in lifestyle factors over the past week or month, failing to capture life course changes in lifestyle behaviors. The study sample was also relatively healthy, racially homogenous, and cognitively high functioning, which limits generalizability of the findings to more racially diverse groups with a broader range of functional abilities. This adds to the growing need for population-based cohort studies to prioritize participant recruitment among marginalized groups who are not typically included in research studies. Furthermore, there was significant attrition bias over the 9-year study period. Specifically, those who were lost to follow-up were more likely to be older, male, have lower educational attainment, have greater CSE, have a lower HLI, and have poorer baseline cognitive function. As such, results should be interpreted with caution as the cognitive trajectories reported in the current study may reflect those who are healthier at baseline. Finally, although the 9-year follow-up time is a strength of the study, having only two time points precludes the detection of possible nonlinear changes in cognition over time, such that the pattern of cognitive decline may differ as a function of baseline CSE and/or healthy lifestyle behaviors.

Despite these limitations, the current results contribute to the existing body of work highlighting the need to consider stressful exposures across the lifespan as important risk factors for age-related cognitive decline. Indeed, the current study lends support for a lifespan model of cognitive aging and suggests that the cognitive health consequences of stress extend beyond immediate timescales. Although no modulating effects of a healthy lifestyle were found in the current study, future research is needed to understand whether lifestyle behaviors or other resilience factors may offset the insidious effects of stress on cognitive health in order to cultivate and promote a healthy aging population.

Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* online.

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Conflict of Interest

None.

Data Availability

The MIDUS study data are available online at the Inter-university Consortium for Political and Social Research (https://www.icpsr.umich.edu/web/ICPSR/series/203). This study was not preregistered.

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