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Psychological Stress across the Lifespan and Cognitive Function among Older Adults: The Moderating Role of a Healthy Lifestyle

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Article

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Résumé

Cette étude visait à (a) analyser les liens entre les indices de gravité du stress tout au long de la vie (jeune âge, âge moyen, âge mûr) et la fonction cognitive chez les personnes âgées vivant dans la communauté et (b) examiner dans quelle mesure un score composite de mode de vie sain, comprenant l'activité physique, l'adhésion à un régime alimentaire sain, la participation sociale, la qualité du sommeil et la relaxation en pleine conscience modère les liens entre la gravité du stress tout au long de la vie et la fonction cognitive. Les participants (n = 226, âge moyen = 68,2 ± 6,5 ans, 68,1 % de femmes) ont rempli des questionnaires d'évaluation du stress et du mode de vie, et ont effectué trois tâches neurocognitives en ligne. Aucun lien direct entre la gravité du stress et la cognition n'a été décelé. Le score composite de mode de vie sain a modéré les liens entre la gravité du stress à un jeune âge, à un âge moyen et à un âge mûr et le contrôle inhibiteur. Des analyses exploratoires suggèrent que cet effet modérateur pourrait dépendre du sexe de la personne. Malgré les limites de l'étude et la nécessité de mener d'autres recherches, les résultats apportent un appui préliminaire à l'hypothèse du rôle du mode de vie dans l'amélioration de la résilience des personnes âgées face aux effets du stress sur la santé cognitive, ce rôle variant selon le sexe.

Abstract

This study aimed to (a) investigate the associations between indices of stress severity across the lifespan (early, middle, late life) and cognitive function among community-dwelling older adults, and (b) examine whether a healthy lifestyle composite score comprised of physical activity, healthy diet adherence, social engagement, sleep quality, and mindful relaxation moderates the associations between lifespan stress severity and cognitive function. Participants (n = 226, M_{age} = 68.2 ± 6.5, 68.1% female) completed questionnaires to measure stress and lifestyle behaviours, and three online neurocognitive tasks. No direct associations between stress severity and cognition were found. The healthy lifestyle composite score moderated the associations between early, midlife, and late-life stress severity and inhibitory control. Exploratory analyses suggest that this moderating effect may be sex-dependent. Despite study limitations and the need for additional research, findings provide preliminary support for the role of lifestyle behaviours in enhancing older adults' resilience to the effects of stress on cognitive health in a sex-specific manner.

Introduction

Understanding the interaction between modifiable risk and protective factors for age-related cognitive decline is an urgent public health priority from a dementia prevention perspective. Chronic stress is a well-known risk factor for poor physical and mental health outcomes in later life, including poor cognitive function and an increased risk of developing dementia in older adulthood (Franks et al., 2021, 2022; McManus et al., 2022). Recently, research on brain health and dementia prevention has emphasized the importance of taking a lifespan approach to understanding the factors that contribute to age-related cognitive decline (Livingston et al., 2020). Specifically, factors that enhance or diminish cognitive resilience accumulate over different periods across the lifespan. This is especially relevant in the context of stress as a risk factor, given that stress may accumulate over time to impact health (Kuh et al., 2003), including cognitive function (McManus et al., 2022).

Among the studies that have examined the association between psychological stress and cognitive function among older adults, most studies have assessed perceptions of stress within the previous month using instrumentation such as the Perceived Stress Scale (e.g., Korten et al., 2017).

This restricted timeframe neglects the potential cumulative nature of stress that is central to its effects on health. The Accumulation of Risk Model suggests that 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), which leads to negative health consequences, including poor cognitive function and an increased risk of cognitive impairments (D'Amico et al., 2020b). Of the studies that have taken a life course approach to understanding the effects of stress on cognitive aging, mixed findings have been reported (Chen et al., 2022; Ouanes et al., 2017). These studies have largely taken an exposure-based approach to assessing cumulative stress, with items inquiring about whether a discrete event occurred. Aligned with the Transactional Model of Stress and Coping (Lazarus & Folkman, 1984), the body's stress response system is activated when an event is appraised as both threatening and exceeding resources to cope. In other words, it is one's subjective perceptions of stress and not the event itself that leads to adverse health consequences. Therefore, 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. Limited research, however, has evaluated whether cumulative perceived stress severity across the lifespan is associated with cognitive function among older adults.

In addition to investigating the total accumulation of stress over the life course, it is also important to consider stress severity at different life stages as the timing of a stressor determines how much impact it will have on health outcomes. Indeed, previous research has shown that early life is a critical period whereby adverse experiences can have detrimental effects on cognitive health in later adulthood (D'Amico et al., 2022). Although stress experienced in early life (relative to midlife and later life) is thought to induce the greatest impact on cognitive health in later life from a theoretical perspective (Frodl & O'Keane, 2013), this conjecture has been scarcely explored empirically. Moreover, a small number of studies have directly examined stress severity in midlife as a predictor of cognitive function among older adults, with most studies to date focusing primarily on occupational stress (e.g., Sindi et al., 2017). Although occupational elements are particularly relevant in midlife, other stressor domains are also important to consider, such as interpersonal and discrimination-related stress. Further, examining the impact of midlife stress on cognitive aging is imperative as midlife is becoming increasingly recognized as a critical period in adult development whereby subtle cognitive changes may be particularly amenable to risk reduction strategies (Lachman et al., 2015). Understanding at which point across the lifespan stress may exert the greatest impact on age-related cognitive health is important in order to identify critical periods that warrant intervention and prevention efforts for optimal brain health.

Another theme in the stress and health literature is the notion that not all individuals are equally impacted by the same exposures to stress (Koolhaas et al., 2011). This suggests that there may be protective factors rendering some individuals better able to withstand the degenerative effects of stress on the brain. As such, understanding factors that may buffer the effects of stress across the lifespan on cognitive function is an important endeavour. Engaging in healthy lifestyle behaviours (e.g., physical activity, adhering to a nutrient-rich Mediterranean diet, social engagement, getting good quality sleep, and managing stress through relaxation techniques) is the leading recommendation for maintaining cognitive health with aging and reducing dementia risk (Rockwood et al., 2020). Considering the cumulative and combined effect of multiple healthy lifestyle behaviours on cognitive health, as opposed to individual behaviours in isolation, may be more practical as healthy lifestyle parameters often cluster and act synergistically to benefit cognitive function (Rabel et al., 2019). Moreover, engaging in healthy lifestyle behaviours can be used as coping mechanisms to manage stress (Ng & Jeffery, 2003). A healthy lifestyle can also enhance cognitive reserve, or the ability to remain cognitively intact despite age-related brain changes (Stern et al., 2019). A healthy lifestyle may account for individual differences in the stress-cognition relationship by buffering the effects of stress on cognitive function. However, limited studies have investigated whether engagement in a healthy lifestyle among older adults can modulate the effects of stress on cognition from a life course perspective.

In a community-dwelling sample of cognitively intact older adults, the objectives of the current study were threefold: to examine (a) the association between cumulative stress severity and cognitive function; (b) the association between stress severity at early, midlife, and late life and cognitive function; and (c) the moderating role of a healthy lifestyle composite score, including physical activity, Mediterranean diet adherence, sleep quality, social engagement, and mindful relaxation, in the relationship between (a) cumulative stress severity and cognitive function, and (b) stress severity at early, midlife, and late life and cognitive function. It was hypothesized that higher levels of cumulative stress severity would be associated with poorer cognitive function, and higher levels of stress severity across all three life stages would be associated with poorer cognitive function, with stress in early life having the largest effect. It was also hypothesized that a higher healthy lifestyle composite score would buffer all aforementioned associations. Given the known sex differences in the experience of stress (Bale & Epperson, 2015) and age-related cognitive function (LaPlume et al., 2022), sex differences were explored by stratifying the models by sex. Additional models were also conducted to explore the moderating effect of individual components of the healthy lifestyle composite score.

Methods

Participants

A total of 226 participants were included in the current online study. This sample size is based on a power calculation using the 'pwr' package in R for a multiple linear regression model with 6 predictor variables and a statistical power of $\beta = 0.08$ at significance level of $\alpha = 0.05$ to detect a small to medium effect size of $f^2 = 0.07$.

Participants were recruited through community organizations, social media advertisements, Prolific, and the Toronto Metropolitan University Senior Participants Pool. To be eligible to participate in the study, participants must have been at least 60 years of age, fluent in English to understand the study instructions, and have access to a computer or laptop with a reliable Internet connection. Participants recruited from the community ($n = 122$) were excluded if they self-reported the following criteria that are known to negatively impact cognitive functioning: (a) a neurological condition (i.e., dementia, Parkinson's disease, Huntington's disease, mild cognitive impairment, multiple sclerosis, epilepsy), (b) schizophrenia or bipolar disorder I or II, (c) having ever had a stroke, including a transient ischemic attack, (d) a concussion or serious head injury within the previous year, (e) having undergone general anesthetic within the previous year, (f) uncorrected vision problems that impact their ability to see (i.e., macular degeneration, blindness, partial blindness, glaucoma, blurred vision), or (g) colour-blindness.

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Participants recruited through Prolific (n = 104) were unable to be excluded from the study for a diagnosis of epilepsy or bipolar type I or II, having ever had a stroke including a transient ischemic attack, having a concussion or serious head injury within the previous year, or having undergone general anesthetic within the previous year. A total of 14 participants reported the presence of these criteria (epilepsy: n = 1; bipolar disorder: n = 1; stroke: n = 2; head injury: n = 2; anesthetic: n = 11). Compared to participants who met the inclusion criteria, these individuals did not differ on any key study variables and the models did not change when removing them from analyses; thus, they were retained in all analyses.

This study was approved by Toronto Metropolitan University's Research Ethics Board (REB 2021-105). Data were collected between September 2021 and April 2023. All participants provided informed consent.

Measures

Sociodemographic and health-related characteristics

Participants completed an online sociodemographic and health questionnaire that collected information on age, sex (male, female, other – please specify), gender (man, woman, non-binary, other – please specify), years of education, ethnicity, perceived socioeconomic status (self-reported as low, medium, or high), employment status, multilingualism, and health-related characteristics including the Jessen criteria for subjective cognitive decline (Jessen et al., 2014), smoking status, and a diagnosis of diabetes, hypertension, and depression. Females were also asked whether they currently or have ever received estrogen treatment, and their age of menopause.

Cumulative and life stage stress

Cumulative stress and stress at early, midlife, and late life were indexed using the Stress and Adversity Inventory (STRAIN; Slavich & Shields, 2018), an automated online assessment system that measures lifetime exposure to 55 acute and chronic stressors (e.g., death of relatives, negative health events, financial difficulties, interpersonal problems, childhood maltreatment). For each stressor that was endorsed, participants were asked follow-up questions pertaining to stressor severity (i.e., 'at its worst, how stressful or threatening was this for you?' from 1 (*very slightly or not at all*) to 5 (*extremely*), frequency (i.e., 'how many times has this happened to you?' from 1 to 5 times), timing (i.e., 'when did this happen?' from 0–3 months ago to 5+ years ago), duration (i.e., 'when did this end?' from 0–3 months ago to 5+ years ago), and age of event(s). For the current study, total stressor severity (i.e., the sum of severity ratings for each stressor endorsed) was used as a measure of cumulative stress. Stressor severity before age 18, between ages 18 to 59, and age 60 and older were used as measures of early, midlife, and late-life stress, respectively. The STRAIN has demonstrated good test-retest reliability (0.90 to 0.92) in a sample including cognitively intact older adults (Slavich & Shields, 2018).

Healthy lifestyle indicator

The Physical Activity Scale for the Elderly (PASE; Washburn et al., 1999), EPIC-Norfolk Food Frequency Questionnaire (FFQ; Bingham et al., 1997), Social Engagement and Activities Questionnaire (SEAQ; Marti & Choi, 2020), Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), and the mindful relaxation subscale of the Mindful Self-Care Scale (MSCS; Cook-Cottone & Guyker, 2018) were self-administered online using Qualtrics to assess physical activity, dietary intake, social engagement, sleep quality, and mindful relaxation practices, respectively. Total scores for Mediterranean diet adherence were calculated using the scoring method by Sofi et al. (2014). Further details on each questionnaire and individual lifestyle behaviour scoring method are included in the Supplementary Material. Each of the five lifestyle behaviour scores was first converted into a Z-score to facilitate comparisons across measures. PSQI scores were multiplied by −1 so that higher scores on each individual scale reflect greater lifestyle behaviour engagement. All Z-scores were then summed to create a total healthy lifestyle indicator score, with higher scores representing greater adherence to a healthy lifestyle.

Cognitive function

The word paired-associates task, Colour-Word Stroop Test (Stroop, 1935), and the 2-back task (Kirchner, 1958) were built using PsychoPy and administered online through Pavlovia to assess associative episodic memory, inhibitory control, and working memory, respectively. These tasks were chosen as they tap into cognitive domains that are sensitive to normal age-related changes and can be administered online asynchronously (Cyr et al., 2021).

The word paired-associates task presented participants with 32 unrelated, neutral, and concrete word pairs displayed one at a time in the centre of the screen for 4000 ms. Immediately following the encoding phase and after a 25-minute delay, participants completed a recall phase where they were presented with 16 intact, 16 recombined, and 16 new word pairs for 7000 ms each, and instructed to indicate whether the pair was intact, recombined, or new. An immediate and delayed associate memory sensitivity index was calculated by subtracting the false alarm rate (i.e., incorrectly responding 'intact' to recombined pairs) from the hit rate (i.e., correctly responding 'intact' to intact pairs), with higher scores reflecting better immediate and delayed associative episodic memory performance.

The Colour-Word Stroop Test required participants to indicate the font colour of 96 colour words displayed one at a time in the centre of the screen for 4500 ms, of which 48 were congruent (e.g., 'red' presented in red font) and 48 were incongruent (e.g., 'red' presented in blue font). The Stroop inhibition score was calculated by subtracting the reaction time on incongruent trials from the reaction time on congruent trials, with higher scores indicating poorer inhibitory control.

The 2-back task presented participants with a series of 40 individual letters displayed one at a time in the centre of the screen for 1000 ms. Participants indicated if each letter presented was identical to the one presented two letters prior. Recall accuracy (i.e., proportion of correct responses) was calculated, with higher scores reflecting better working memory performance.

Each of the four cognitive tasks scores was first converted into a Z-score to facilitate comparisons across measures, with Stroop inhibition scores multiplied by −1 so that higher scores indicate better performance. All Z-scores were then summed to create a global cognition composite score, with higher scores representing better global cognitive performance.

Statistical analyses

All analyses were performed using R. Bivariate correlations were conducted to determine the associations between the primary variables of interest (i.e., cumulative and life-stage stress severity, healthy lifestyle index, and cognitive function) and the following sociodemographic and health-related variables: age, sex (0 = female, 1 = male), years of education, perceived socioeconomic

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status (0 = low, 1 = medium, 2 = high), and daily energy intake (kcal). Gender, ethnicity, diabetes, hypertension, depression, current smoking status, subjective cognitive decline, employment status, multilingualism, age of menopause, and current estrogen use were treated as descriptive variables as they were not significantly associated with stress, lifestyle, or cognition.

Using the 'stats' package in R, a linear regression model, adjusting for age, sex, years of education, and perceived socioeconomic status, was conducted to determine the associations between life-stage stress severity (i.e., early, midlife, and late life) and cognitive function. A series of linear moderation models were conducted to determine the moderating role of a healthy lifestyle composite in the relationship between lifespan indices of stress severity and cognitive function. Moderation models were analysed with either cumulative stress severity, early life stress severity, midlife stress severity, or late-life stress severity as the independent variable; the healthy lifestyle indicator (HLI) as the moderating variable; and either the global cognition composite score, immediate associative memory score, delayed associative memory score, Stroop inhibition score, or 2-back accuracy score as the dependent variable. Statistically significant stress severity × HLI interactions were decomposed at 1 standard deviation below the mean HLI score (i.e., low healthy lifestyle adherence), the mean HLI score (i.e., moderate healthy lifestyle adherence), and 1 standard deviation above the mean HLI score (i.e., high healthy lifestyle adherence). All moderation models were adjusted for age, sex, years of education, daily energy intake (kcal), and perceived socioeconomic status.

To explore whether the moderating role of a healthy lifestyle in the relationship between indices of stress across the lifespan and cognitive function differs between males and females, all primary models were conducted disaggregated by sex. Moreover, to explore whether the moderating role of a healthy lifestyle may be driven by specific lifestyle behaviours, the aforementioned moderation models were conducted for each of the five lifestyle behaviours, independently (i.e., Mediterranean diet adherence, physical activity, social engagement, sleep quality, and mindful relaxation).

Results

Participant characteristics

Table 1 shows a full summary of participant sociodemographic and health-related characteristics, including descriptive information about cumulative and life-stage stress, healthy lifestyle behaviours, and cognitive function. On average, participants were 68.2 years of age (SD = 6.5, median = 67), 68.1% of the sample was female, the mean years of education was 16.0 (SD = 3.2), 72.1% reported a medium perceived socioeconomic position, and 87.9% identified as White/Caucasian. Participants reported, on average, 23.5 (SD = 14.5) acute and chronic stressors across the lifespan, with 3.2 (SD = 4.3) occurring during early life, 14.2 (SD = 9.0) occurring during midlife, and 4.9 (SD = 4.7) occurring during late life.

Table 2 provides the bivariate correlations between the sociodemographic and health-related covariates, lifespan indices of stress severity, HLI score and its components, and cognitive function scores. Older age was associated with less cumulative stress severity ($r = -0.23$, $p < 0.001$), less early life stress severity ($r = -0.16$, $p = 0.02$), and less midlife stress severity ($r = -0.45$, $p < 0.001$). Compared to females, males had lower immediate associative memory scores ($r = -0.17$, $p = 0.01$), and higher working memory scores ($r = 0.13$, $p = 0.04$). More years of

Table 1. Participant sociodemographic and health-related characteristics (n = 226)

	Mean ± SD (range) or % (n)
Age	68.2 ± 6.5 (60 – 92)
Sex (% female)	68.1 (154)
Gender (% women)	68.1 (154)
Years of education	16.0 ± 3.2 (7 – 30)
Ethnicity (% Caucasian)	87.9 (197)
Perceived SES (%)	
Low	15.5 (35)
Medium	72.1 (163)
High	12.4 (28)
Diabetes (% yes)	6.3 (14)
Hypertension (% yes)	27.7 (62)
Depression (% yes)	21.4 (48)
Subjective cognitive decline (% yes)	37.2 (84)
Current smoking status (% yes)	4.9 (11)
Employment status (%)	
Working	27.4 (62)
Retired	57.1 (129)
Unemployed	7.5 (17)
Other	8.0 (18)
Multilingual (% yes)	33.6 (76)
Age of menopause[a]	49.3 ± 5.4 (30 – 59)
Current or past estrogen use (% yes)[a]	27.3 (42)
Daily energy intake (kcal)	917 ± 415 (280 – 3,942)
STRAIN: total stressor severity score	58.9 ± 34.9 (4 – 178)
STRAIN: early life stress severity score	9.3 ± 10.7 (0 – 63)
STRAIN: midlife stress severity score	36.6 ± 20.7 (1 – 116)
STRAIN: late-life stress severity score	13.4 ± 10.0 (0 – 73)
STRAIN: total stressor count	23.5 ± 14.5 (1 – 105)
STRAIN: early life stressor count	3.2 ± 4.3 (0 – 33)
STRAIN: midlife stressor count	14.2 ± 9.0 (1 – 50)
STRAIN: late life stressor count	4.9 ± 4.7 (0 – 52)
SEAQ score	20.6 ± 6.3 (3 – 37)
PSQI score	7.1 ± 3.1 (0 – 16)
MSCS mindful relaxation score	3.07 ± 0.82 (1.00 – 4.83)
PASE score	136.8 ± 70.4 (8.7 – 413.4)
Mediterranean diet score	11.0 ± 2.3 (4 – 15)
HLI (Z-score range)	−7.39 – 6.91
Immediate associative memory score	0.310 ± 0.268 (−0.188 – 1.00)
Delayed associative memory score	0.220 ± 0.238 (−0.438 – 1.00)
Stroop inhibition score	−0.227 ± 0.161 (−1.168 – 0.801)
2-back accuracy score	0.789 ± 0.108 (0.375 – 1.00)
Global cognition composite score (Z-score range)	−8.51 – 5.60

Notes: HLI = healthy lifestyle index; MSCS = Mindful Self-Care Scale; PASE = Physical Activity Scale for the Elderly; PSQI = Pittsburgh Sleep Quality Index; SD = standard deviation; SEAQ = Social Engagement and Activities Questionnaire; SES = socioeconomic status; STRAIN = Stress and Adversity Inventory.
[a]Among females only.

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Table 2. Bivariate correlations between the study variables of interest

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Age (1)	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Sex (2)	**0.18**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Education (3)	**0.16**	**0.14**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Perceived SES (4)	**0.20**	**0.13**	**0.33**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
kcal (5)	0.03	**0.17**	−0.06	**−0.15**	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cumulative stress (6)	**−0.23**	**−0.22**	−0.07	**−0.27**	0.05	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Early life stress (7)	**−0.16**	0.02	−0.08	**−0.17**	0.06	**0.65**	–	–	–	–	–	–	–	–	–	–	–	–	–
Midlife stress (8)	**−0.45**	0.001	−0.02	**−0.16**	−0.04	**0.35**	**0.24**	–	–	–	–	–	–	–	–	–	–	–	–
Late-life stress (9)	−0.07	0.08	0.07	−0.08	0.04	**0.15**	0.12	0.11	–	–	–	–	–	–	–	–	–	–	–
Social engagement (10)	0.05	0.006	0.04	0.02	0.03	0.04	−0.07	0.03	0.12	–	–	–	–	–	–	–	–	–	–
Sleep (11)	−0.07	0.03	0.06	0.05	0.02	−0.05	−0.04	0.09	−0.04	0.06	–	–	–	–	–	–	–	–	–
Mindful relaxation (12)	0.01	−0.12	−0.01	0.04	0.11	0.02	−0.003	−0.11	**0.14**	0.15	0.03	–	–	–	–	–	–	–	–
Physical activity (13)	−0.08	0.005	−0.12	−0.03	0.08	−0.03	−0.04	0.04	−0.07	0.05	0.03	0.02	–	–	–	–	–	–	–
Mediterranean diet (14)	0.07	**0.15**	−0.05	0.03	**0.30**	−0.03	−0.06	−0.001	0.05	0.08	−0.13	0.03	**0.14**	–	–	–	–	–	–
HLI (15)	−0.005	0.03	−0.03	0.04	**0.22**	−0.02	−0.09	0.02	0.08	**0.55**	**0.41**	**0.50**	**0.51**	**0.46**	–	–	–	–	–
Immediate memory (16)	−0.07	**−0.17**	**0.17**	0.02	−0.10	0.08	−0.01	−0.01	−0.09	0.04	−0.003	−0.05	−0.008	−0.07	−0.04	–	–	–	–
Delayed memory (17)	−0.07	−0.12	**0.20**	0.09	−0.08	0.06	−0.02	0.02	−0.09	0.06	0.07	−0.08	0.002	−0.12	−0.03	**0.64**	–	–	–
Inhibitory control (18)	0.07	−0.02	−0.05	0.03	0.06	−0.006	−0.04	−0.04	0.03	−0.09	−0.11	0.06	−0.05	0.08	−0.04	**−0.15**	**−0.18**	–	–
Working memory (19)	−0.06	**0.13**	0.12	0.03	0.02	0.01	−0.07	0.07	−0.01	−0.03	−0.02	−0.12	0.07	0.05	−0.02	**0.25**	**0.18**	−0.05	–
Global cognition (20)	−0.06	−0.08	**0.21**	0.07	−0.03	0.07	−0.07	0.03	−0.05	0.001	−0.02	−0.05	−0.001	<0.001	−0.03	**0.75**	**0.72**	**0.27**	**0.59**

Notes: Bolded correlation statistics are statistically significant at $p < 0.05$.
HLI = healthy lifestyle index; SES = socioeconomic status.

Table 3. Associations between early, midlife, and late-life stress severity and cognitive function among the total sample and stratified by sex

	Immediate associative memory	Delayed associative memory	Inhibitory control	Working memory	Global cognition
Total sample (n = 226)	R^2 = 0.08	R^2 = 0.08	R^2 = 0.01	R^2 = 0.05	R^2 = 0.07
Early life stress severity	−0.006 [−0.13, 0.15]	−0.001 [−0.14, 0.14]	−0.03 [−0.17, 0.11]	−0.09 [−0.23, 0.06]	−0.07 [−0.21, 0.08]
Midlife stress severity	−0.04 [−0.19, 0.11]	−0.001 [−0.15, 0.15]	0.002 [−0.15, 0.15]	−0.05 [−0.10, 0.20]	0.01 [−0.14, 0.17]
Late-life stress severity	−0.10 [−0.23, 0.04]	−0.10 [−0.23, 0.04]	0.05 [−0.09, 0.18]	−0.04 [−0.17, 0.10]	−0.06 [−0.20, 0.08]
Females (n = 154)	R^2 = 0.08	R^2 = 0.11	R^2 = 0.05	R^2 = 0.04	R^2 = 0.12
Early life stress severity	−0.05 [−0.23, 0.13]	−0.12 [−0.31, 0.07]	0.03 [−0.10, 0.17]	−0.06 [−0.24, 0.11]	−0.09 [−0.27, 0.08]
Midlife stress severity	−0.03 [−0.21, 0.16]	0.07 [−0.12, 0.26]	−0.005 [−0.15, 0.14]	0.05 [−0.13, 0.23]	0.05 [−0.13, 0.22]
Late-life stress severity	−0.11 [−0.27, 0.06]	−0.08 [−0.25, 0.10]	0.03 [−0.10, 0.15]	−0.05 [−0.22, 0.11]	−0.09 [−0.25, 0.07]
Males (n = 72)	R^2 = 0.05	R^2 = 0.11	R^2 = 0.03	R^2 = 0.03	R^2 = 0.02
Early life stress severity	0.14 [−0.09, 0.38]	**0.20 [0.01, 0.39]**	−0.13 [−0.48, 0.21]	−0.15 [−0.42, 0.13]	0.02 [−0.29, 0.33]
Midlife stress severity	−0.08 [−0.35, −0.20]	−0.15 [−0.36, 0.07]	0.02 [−0.42, 0.38]	0.06 [−0.23, 0.34]	−0.07 [−0.39, 0.24]
Late-life stress severity	−0.05 [−0.30, 0.20]	−0.07[−0.28, 0.13]	0.10 [−0.27, 0.46]	0.02 [−0.24, 0.28]	0.05 [−0.26, 0.35]

Notes: Sex-stratified models controlled for age, years of education, and perceived socioeconomic status. Total model additionally controlled for sex. Data presented as β [95% confidence intervals].

Table 4. Associations between indices of stress across the lifespan, a healthy lifestyle index, and cognitive performance

	Immediate associative memory	Delayed associative memory	Inhibitory control	Working memory	Global cognition
Cumulative stress severity	R^2 = 0.08	R^2 = 0.08	R^2 = 0.03	R^2 = 0.04	R^2 = 0.07
Cumulative stress	0.06 [−0.09, 0.20]	0.05 [−0.10, 0.20]	0.005 [−0.14, 0.15]	0.03 [−0.12, 0.17]	0.05 [−0.10, 0.21]
HLI	−0.004 [−0.14, 0.13]	−0.007 [−0.15, 0.13]	−0.05 [−0.19, 0.09]	−0.01 [−0.15, 0.12]	−0.001 [−0.14, 0.14]
Cumulative stress × HLI	0.02 [−0.13, 0.18]	0.01 [−0.14, 0.16]	0.10 [−0.05, 0.26]	0.08 [−0.07, 0.23]	0.11 [−0.05, 0.27]
Early life stress severity	R^2 = 0.08	R^2 = 0.07	R^2 = 0.04	R^2 = 0.05	R^2 = 0.07
Early life stress severity	−0.01 [−0.15, 0.13]	−0.01 [−0.15, 0.13]	−0.004 [−0.14, 0.13]	−0.07 [−0.21, 0.07]	−0.06 [−0.21, 0.09]
HLI	−0.02 [−0.16, 0.12]	−0.01 [−0.15, 0.13]	−0.04 [−0.18, 0.10]	−0.02 [−0.15, 0.12]	−0.02 [−0.16, 0.13]
Early life stress severity × HLI	−0.06 [−0.23, 0.10]	−0.01 [−0.18, 0.16]	**0.20 [0.03, 0.37]**	0.09 [−0.08, 0.26]	0.10 [−0.08, 0.27]
Midlife stress severity	R^2 = 0.08	R^2 = 0.07	R^2 = 0.05	R^2 = 0.04	R^2 = 0.07
Midlife stress severity	−0.05 [−0.20, 0.10]	−0.01 [−0.16, 0.14]	0.02 [−0.13, 0.16]	0.04 [−0.11, 0.19]	0.002 [−0.15, 0.15]
HLI	−0.01 [−0.15, 0.12]	−0.007 [−0.14, 0.13]	−0.07 [−0.20, 0.07]	−0.03 [−0.16, 0.11]	−0.03 [−0.17, 0.11]
Midlife stress severity × HLI	0.03 [−0.10, 0.17]	−0.05 [−0.19, 0.08]	**0.19 [0.05, 0.32]**	0.04 [−0.09, 0.17]	0.09 [−0.05, 0.22]
Late-life stress severity	R^2 = 0.09	R^2 = 0.08	R^2 = 0.06	R^2 = 0.04	R^2 = 0.06
Late-life stress severity	−0.09 [−0.23, 0.04]	−0.10 [−0.23, 0.04]	0.06 [−0.07, 0.20]	−0.04 [−0.17, 0.10]	−0.07 [−0.21, 0.07]
HLI	−0.01 [−0.15, 0.12]	−0.006 [−0.15, 0.13]	−0.04 [−0.18, 0.09]	−0.02 [−0.16, 0.12]	−0.01 [−0.15, 0.13]
Late-life stress severity × HLI	−0.10 [−0.25, 0.05]	−0.04 [−0.19, 0.12]	**0.22 [0.07, 0.37]**	−0.01 [−0.16, 0.14]	0.04 [−0.12, 0.20]

Notes: All models controlled for age, sex, years of education, daily energy intake (kcal), and perceived socioeconomic status. Data presented as β [95% confidence intervals].
HLI = healthy lifestyle index.

education were associated with higher immediate (r = 0.17, p = 0.01) and delayed (r = 0.20, p = 0.004) associative memory scores, and higher global cognition composite scores (r = 0.21, p = 0.003). Lower perceived socioeconomic position was associated with higher cumulative stress severity scores (r = −0.27, p < 0.001) and higher early life stress severity scores (r = −0.17, p = 0.009).

Primary models

Adjusting for age, sex, years of education, and perceived socioeconomic status, stress severity at early, midlife, late life, and cumulative stress was not directly associated with cognitive outcomes (see Table 3). Significant interactions with HLI were observed for early stress, midlife stress, and late-life stress, but not cumulative stress.

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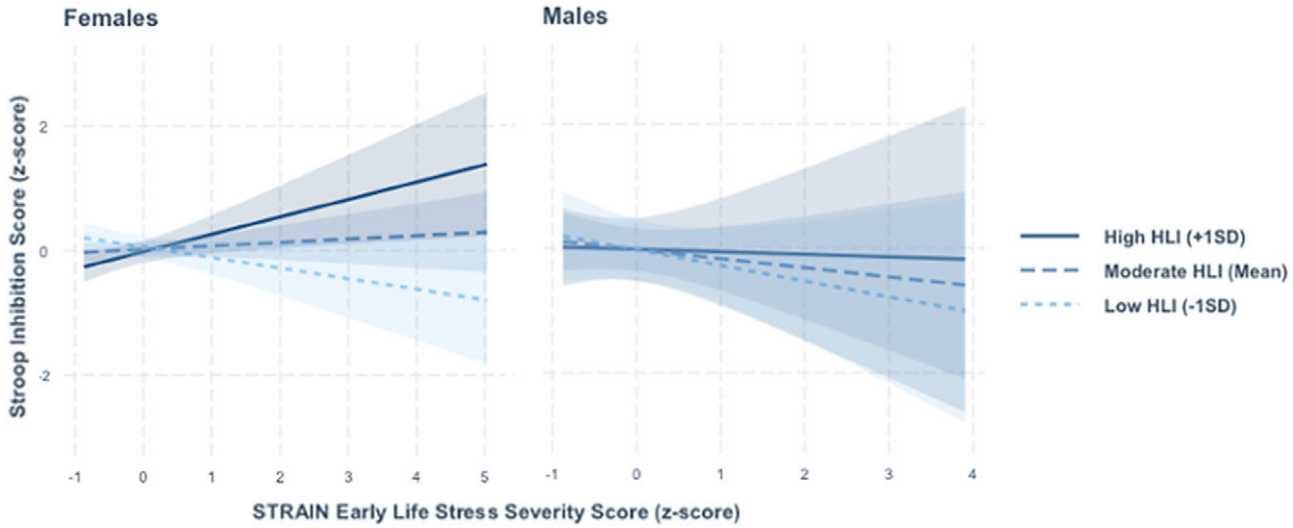

Figure 1. The association between early life stress severity and inhibitory control at a low, moderate, and high healthy lifestyle index among males and females. HLI = healthy lifestyle index; STRAIN = Stress and Adversity Inventory.

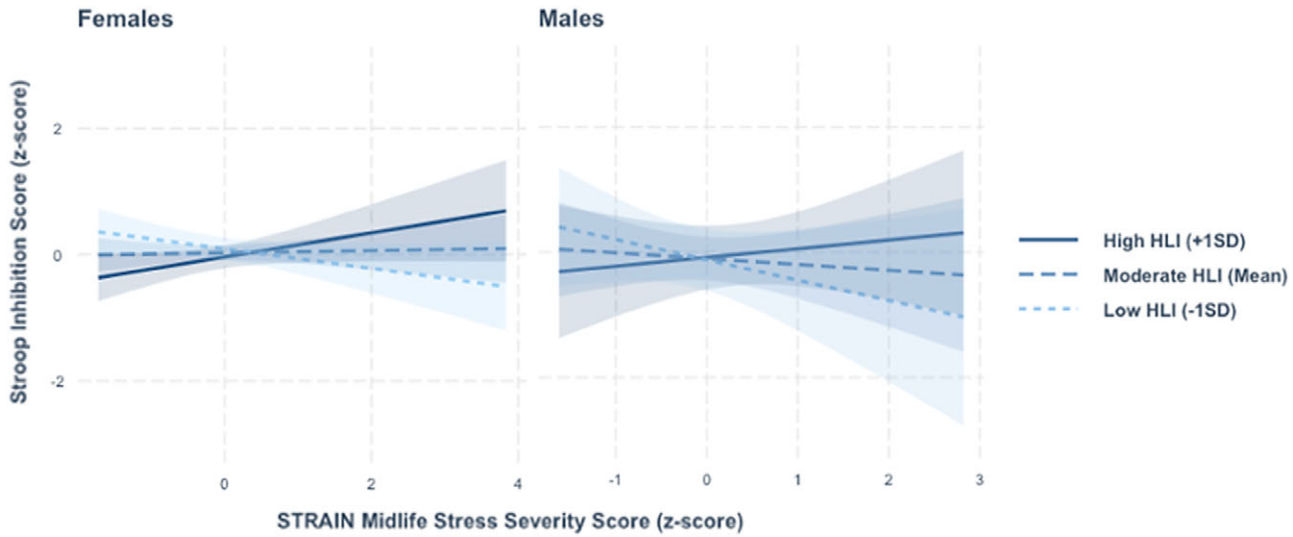

Figure 2. The association between midlife stress severity and inhibitory control at a low, moderate, and high healthy lifestyle index among males and females. HLI = healthy lifestyle index; STRAIN = Stress and Adversity Inventory.

Specifically, higher levels of early life stress was associated with poorer inhibitory control at a low HLI score (β = −0.20, 95% CI [−0.40, −0.001]), but not moderate (β = −0.01, 95% CI [−0.14, 0.13]) or high HLI score (β = 0.19, 95% CI [−0.04, 0.42]). Moreover, higher levels of midlife stress severity was associated with better inhibitory control at high HLI score ((β = 0.20, 95% CI [0.001, 0.40]), but not low (β = −0.17, 95% CI [−0.37, 0.02]) or moderate HLI score (β = 0.01, 95% CI [−0.13, 0.16]). Finally, higher late-life stress severity was associated with better inhibitory control at high HLI (β = 0.28, 95% CI [0.08, 0.48]), but not low (β = −0.16, 95% CI [−0.36, 0.04]) or moderate HLI (β = 0.06, 95% CI [−0.07, 0.19]). Table 4 shows all model estimates.

Exploratory models

Sex-stratified analyses

Analyses restricted to females failed to show a significant direct association between stress severity scores across the lifespan (early, midlife, late life, and cumulative) and cognitive outcomes. However, a significant early life stress × HLI interaction was found for inhibitory control (β = 0.21, 95% CI [0.06, 0.38]), such that greater early life stress severity was associated with better inhibitory control at a high HLI score (β = 0.28, 95% CI [0.06, 0.49]), but not low (β = −0.17, 95% CI [−0.37, 0.03]) or moderate HLI (β = 0.05, 95% CI [−0.07, 0.18]). Similarly, higher midlife stress severity was associated with better inhibitory control at high HLI (β = 0.19, 95% CI [0.01, 0.39]), but not low (β = −0.16, 95% CI [−0.34, 0.02]) or moderate HLI (β = 0.02, 95% CI [−0.12, 0.15]). No other interactions were statistically significant in females.

Analyses restricted to males showed that higher early life stress severity was associated with better delayed associative memory only (β = 0.20, 95% CI [0.01, 0.39]), and a higher HLI score was associated with poorer delayed associative memory only (β = −0.30, 95% CI [−0.55, −0.06]). Finally, a statistically significant late-life stress severity × HLI interaction was found for inhibitory control in males (β = 0.57, 95% CI [0.14, 0.99]), such

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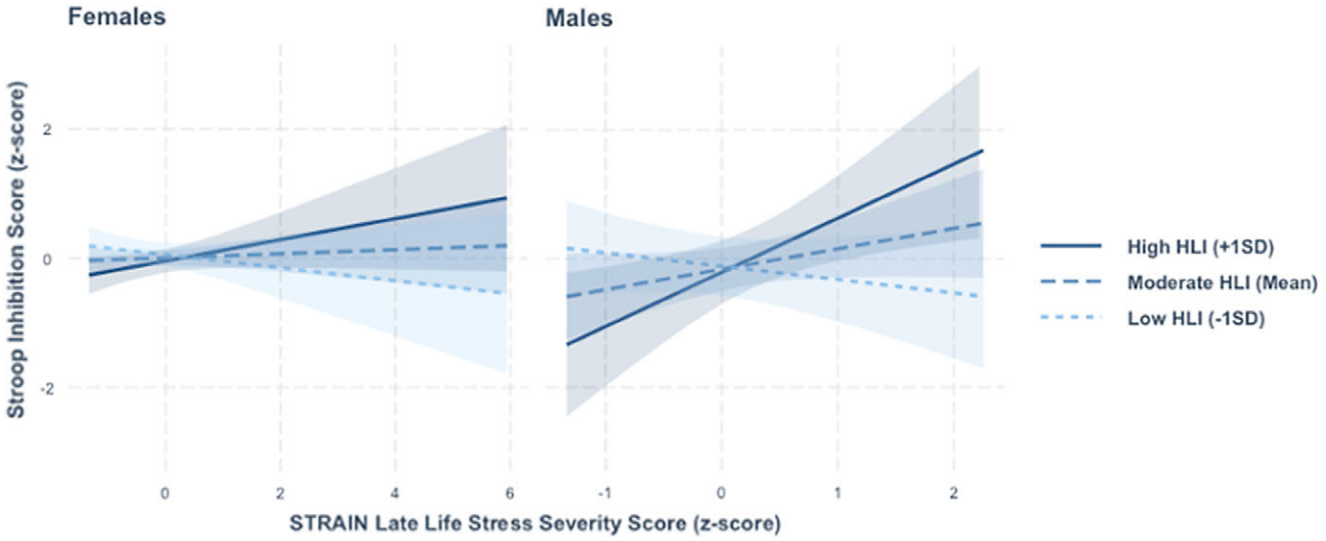

Figure 3. The association between late-life stress severity and inhibitory control at a low, moderate, and high healthy lifestyle index among males and females. HLI = healthy lifestyle index; STRAIN = Stress and Adversity Inventory.

that higher late-life stress severity was associated with better inhibitory control at high HLI ($\beta = 0.84$, 95% CI [0.24, 0.99]), but not low ($\beta = -0.21$, 95% CI [−0.66, 0.23]) or moderate HLI ($\beta = 0.31$, 95% CI [−0.07, 0.69]). No other interactions were statistically significant in males.

Supplementary Tables 1 and 2 provide all model estimates for females and males, respectively. Figures 1–3 show the simple slopes for the relationship between early, middle, and late-life adversity and inhibitory control at low, moderate, and high HLI between males and females.

Lifestyle-specific interactions

In exploring the moderating role of individual lifestyle behaviours, no direct association was found for any lifestyle behaviour and cognitive outcome. A statistically significant cumulative stress severity × Mediterranean diet interaction was found for global cognition only ($\beta = 0.16$, 95% CI [0.006, 0.31]), such that higher cumulative stress severity was associated with better global cognition composite scores at high Mediterranean diet adherence ($\beta = 0.25$, 95% CI [0.01, 0.49]), but not at moderate ($\beta = 0.09$, 95% CI [−0.06, 0.24]) or low ($\beta = -0.07$, 95% CI [−0.27, 0.12]) Mediterranean diet adherence. See Supplementary Tables 3–7 for all model estimates.

Discussion

Although stress across the lifespan has been found to be associated with poorer age-related cognitive health, significant heterogeneity in the stress-cognition relationship exists. This may be due to exposure-based assessment of stress and/or factors that modify the impact of stress on cognitive function. To address this, the current study sought to examine the association between lifespan indices of stress severity and cognitive function among older adults, as well as the moderating effect of a healthy lifestyle in the relationship between stress severity indices and cognitive function. In contrast to the study hypotheses, no direct associations were found between any of the lifespan stress severity scores and cognitive function. Although a healthy lifestyle did not moderate the association between cumulative stress and cognition, engaging in a healthy lifestyle did moderate the association between early, midlife, and late-life stress severity and inhibitory control. Exploratory analyses suggest that these associations slightly differ in males and females and provide additional insight into the role of individual lifestyle habits in the stress-cognition relationship.

The current study findings failed to support the Accumulation of Risk Model (Ben-Shlomo & Kuh, 2002), and do not suggest a differential risk effect on late-life cognitive performance depending on the timing of stress exposure over the life course. These findings are in contrast to previous research showing a relationship between stress across the lifespan and age-related cognitive function (Chen et al., 2021). However, previous research used a count-based approach to stress exposure over the lifespan, which does not include the perception of stress or the perceived severity of the threat. The null associations may be interpreted in at least three ways. First, a paucity of work has used the STRAIN as a tool to investigate the association between stress and cognitive aging. As such, it is possible that the STRAIN, a relatively new stress assessment instrument, is not sensitive to predicting cognitive performance in later adulthood. Furthermore, this is the first study to tease apart life course stress (i.e., early, middle life and later life) using the STRAIN. As such, further research is needed to elucidate the predictive validity of this tool in the context of life course stress and cognitive aging. Second, it is important to reflect on the study sample and variations in test scores. Specifically, study participants performed relatively well on the cognitive tasks, likely introducing a ceiling effect in detecting an association between stress and cognitive function. Moreover, participants reported, on average, relatively low severity ratings of stressful events across the lifespan compared to previous research (Slavich & Shields, 2018). Taken together, there may have been little statistical power to detect an association between stress and cognition due to the limited variation in stress severity scores and cognitive function. Third, the presence of an association between stress and cognition is dependent on lifestyle behaviours. In line with this third possible explanation, a moderation effect of a healthy lifestyle was found in the association between stress at different life stages and inhibitory control.

Partially supporting the study hypotheses, engaging in a healthy lifestyle was found to buffer the association between higher levels of

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early life stress severity and poorer inhibitory control. Namely, higher stress severity was associated with poorer inhibitory control among older adults with a low healthy lifestyle index score only, suggesting that engaging in at least a moderate healthy lifestyle may offset the negative impact of early life stress on cognitive inhibition in later life. Although limited to cognitive inhibition in the current sample, findings support the notion that engaging in healthy lifestyle behaviours may offset the neurotoxic effects of psychological stress experienced in early life on the brain, and, subsequently, cognitive functioning. Early life adversity can cause physiological dysregulation across immune and cardiometabolic systems (McEwen, 1998), which can lead to health consequences in later life, including poor cognitive function (D'Amico et al., 2020b, 2022). Research has also shown that a healthy lifestyle is associated with lower inflammatory markers and better cardiometabolic health among older adults (Sotos-Prieto et al., 2015). Accordingly, a healthy lifestyle may beneficially target the biological mechanisms through which early life stress impacts cognition.

Significant interactions on cognitive inhibition were also observed for midlife and late-life stress severity; however, these associations were not reflective of a stress-buffering hypothesis. Rather, greater midlife and late-life stress severity scores were associated with better inhibitory control when older adults also reported a high healthy lifestyle. This may suggest that health-promoting behaviours may confer a *resilience effect* to stress, such that stress at midlife and late life may support brain health if the individual is actively engaged in adaptive coping mechanisms, such as physical activity, healthy eating, socialization, and mindful relaxation. Indeed, although stressors are inherently challenging, stress can be adaptive depending on the context and the resources available for coping (Lazarus & Folkman, 1984). Research in adult mice shows that acute, mild, predicable, or controllable stress may facilitate adaptive neuronal and behavioural outcomes (Suri & Vaidya, 2015). Although a resilience effect was not anticipated, it is possible that the relatively low average STRAIN score of the current sample provided a basis for this effect to emerge. Furthermore, engaging in a healthy lifestyle may provide a supportive context through which moderate stress builds resilience and exerts a positive impact on cognitive health.

It is worth noting that statistically significant moderating effects were only observed for inhibition, and not for episodic memory, working memory, or the global cognitive composite score. Akin to the frontal lobe hypothesis of aging, tasks that assess executive functions, including inhibitory control, may be more sensitive to normal age-related changes compared to hippocampal-dependent tasks such as learning and memory (West, 2000), and thus more sensitive to the modulating effects of lifestyle behaviours on the relationship between stress and cognitive function. Indeed, a previous study found a buffering role of a healthy, Mediterranean diet in the relationship between perceived stress and executive function, but not episodic memory (D'Amico et al., 2020a). The frontal lobe hypothesis may be especially relevant in a high-functioning older adult sample. Future research that assesses multiple cognitive domains is needed to replicate the current study findings.

It must be noted that the healthy lifestyle index score did not directly associate with cognitive functioning. This was unexpected given the wealth of prior work showing that engaging in health-promoting lifestyle behaviours, such as physical activity, healthy dietary intake, social engagement, getting good quality sleep, and practicing mindful relaxation, have cognitive benefits for older adults (e.g., Mamalaki et al., 2021). In addition to the small variation in cognitive performance, participants also scored relatively high on the lifestyle behaviour assessments, demonstrating an overall active and engaged lifestyle among the study sample. Accordingly, the limited variation in scores reflecting a healthy, high-functioning sample may have reduced the ability to detect the expected association between lifestyle and cognitive performance. It is also possible that a composite healthy lifestyle score may dilute the benefits of previously reported effective lifestyle behaviours. However, effect sizes were greater in the models that included the healthy lifestyle composite score, compared to those with the individual behaviours, suggesting that multiple behaviours account for a larger proportion of variation in cognitive performance.

Exploration of individual behaviours as moderators revealed that individual lifestyle behaviours may also modulate the stress-cognition relationship. Indeed, higher cumulative stress severity was associated with better global cognition at a higher adherence to a Mediterranean dietary pattern, while no association between stress and global cognition was found at moderate and low Mediterranean diet intake. The Mediterranean diet is comprised of individual foods and nutrients with anti-inflammatory and antioxidant properties that are associated with better cognitive performance and a reduced risk of cognitive decline with aging (Féart et al., 2010). As such, it is possible that the health-promoting properties of the Mediterranean diet target the mechanisms through which cumulative stress impacts cognitive health and provide an adaptive context through which stress has positive impacts on cognitive function.

Contrary to the Stress Buffering Hypothesis (Cohen & Pressman, 2004), social engagement did not moderate the stress-cognition relationship. The same null effects were found for sleep quality. It is plausible, however, that poor sleep quality is both an antecedent and a symptom of ongoing cognitive problems in later life (Casagrande et al., 2022), which may explain why sleep does not buffer the effects of stress on cognition. Moreover, although midlife stress had a statistically significant interaction with both physical activity and mindful relaxation, simple slopes analyses did not show a differential effect at low, moderate, and high physical activity or mindful relaxation. This was unexpected, as both physical activity and mindful relaxation are reportedly associated with both enhanced cognitive function and reduced levels of stress (Casaletto et al., 2022; Lazar et al., 2000). It is possible that a larger variation in cognitive performance, stress severity, and lifestyle behaviour engagement may have resulted in significant conditional effects. Given that examining the moderating role of individual behaviours was exploratory, future research is needed to confirm these results.

Exploratory analyses revealed sex-specific associations in the relationship between stress and cognitive performance. While no direct associations between stress severity and cognitive performance were found in females, analyses in males showed that higher early life stress and a higher healthy lifestyle index score independently associated with better episodic memory. However, no moderating effects were found with respect to episodic memory. Rather, moderating effects of a healthy lifestyle in the stress-inhibition association were found in both females and males, with slight nuances. In females, a *resilience effect* was found in the association between early and midlife stress and inhibitory control, whereas this *resilience effect* was found in the association between late-life stress and inhibitory control in males. Given the exploratory nature of these findings, it is unclear which biopsychosocial mechanisms explain these sex-specific effects. These findings should also be interpreted with caution given the small sample size, especially among males, when disaggregating the data, which did not meet the

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target sample size provided by the power calculation. Nonetheless, the findings support the importance of taking a sex-based approach to brain health and aging research.

Although the current study contributes to the literature on stress, lifestyle, and cognitive aging, the study findings must be interpreted in light of several limitations. Firstly, the high-functioning and homogeneous nature of the sample characteristics cannot be extrapolated to the general population of older adults, limiting the generalizability of the current findings. Indeed, the sample was majority White, female, highly educated, of middle socioeconomic status, in good physical health, performed well on the cognitive tasks, reported relatively low levels of stress across the lifespan, and engaged in a healthy and active lifestyle. Although considerable efforts were made to recruit a diverse sample (e.g., leveraging existing partnerships and establishing new partnerships with local community organizations and support groups, targeting low-income neighbourhoods), the online setting of the study may have introduced several biases, as participants were required to own and know how to use a computer as well as have access to the Internet to complete the study tasks. This likely excluded individuals of lower socioeconomic status and older adults with low computer literacy. Including participants of varying socioeconomic backgrounds is important, especially in the context of stress, health, and aging. It is therefore important for academic researchers to prioritize building mutually beneficial relationships with the community when recruiting participants that reflect the demographic landscape of Canada's aging population. The findings from this study must be replicated in larger and more diverse samples to increase the statistical power.

Online and asynchronous data collection presents various other limitations including lack of quality control in ensuring that participants understood the instructions and were completing the study tasks without distraction. However, participants with implausible cognitive task data were removed from analyses to circumvent this issue. Furthermore, the self-reported and retrospective nature of the stress assessment could have entailed recall bias, especially when reflecting on experiences in early life. Similarly, lifestyle behaviours were self-reported, which may have resulted in a biased estimate of engagement. The method used to score the healthy lifestyle composite may also present with various limitation as it assumes equal weighting across all lifestyle behaviours. Future research may consider using data reduction techniques such as principal components analysis to address this limitation. Additional research may also include other lifestyle behaviours that are relevant in the context of stress and cognitive health, such as smoking and prescribed medication. Finally, the cross-sectional study design precludes any claims of causality between the study variables of interest.

Despite study limitations, this study adds to the body of work examining modifiable factors across the lifespan for cognitive health with aging and provides multiple avenues for future research in this area. Indeed, this work provides evidence suggesting that the association between stress at various life stages and cognitive function may be modified through engagement in health-promoting behaviours, and underscores the importance of incorporating a sex-based approach in research on lifespan models of stress, aging, and cognitive health.

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