Lifecourse Traumatic Events and Cognitive Aging in the Health and Retirement Study

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Introduction: Much of the heterogeneity in the rate of cognitive decline and the age of dementia onset remains unexplained, and there is compelling data supporting psychosocial stressors as important risk factors. However, the literature has yet to come to a consensus on whether there is a causal relationship and, if there is, its direction and strength. This study estimates the relationship between lifecourse traumatic events and cognitive trajectories and predicted dementia incidence.

Methods: Using data on 7,785 participants aged ≥65 years from the Health and Retirement Study, this study estimated the association between lifecourse experience of 10 traumatic events (e.g., losing a child) and trajectories of Telephone Interview for Cognitive Status from 2006 to 2016 using linear mixed-effects models and predicted incident dementia from 2006 to 2014 using cumulative incidence functions (data analysis was in 2020−2022). Inverse probability weights accounted for loss to follow-up and confounding by sex, education, race/ethnicity, and age.

Results: Experiencing 1 or more traumatic events over the lifecourse was associated with accelerated decline compared with experiencing no events (e.g., β= −0.05 [95% CI= −0.07, −0.02] Health and Retirement Study-Telephone Interview for Cognitive Status units/year; 1 vs 0 events). In contrast, experiencing traumatic events was associated with better cognitive function cross-sectionally. Furthermore, the impact of trauma on cognitive decline was of greater magnitude when it occurred after the age of 64 years. However, the magnitude and direction of association varied by the specific traumatic event. There were no associations with predicted incident dementia.

Conclusions: These results suggest that researchers and clinicians should not aggregate traumatic events for understanding the risk of accelerated cognitive decline.
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INTRODUCTION

Although there are several established demographic and clinical risk factors for Alzheimer Disease and Related Dementias, including chronological age, ApoE-e4 allele, sex, and education,[1−3] there is a growing body of research supporting a link between exposure to stressors and cognitive impairment or dementia risk. However, existing data on the impact of psychosocial stressors (e.g., traumatic events [TEs]) on Alzheimer's Disease and Related Dementias risk is missing crucial information. For example, there are little data on the importance of timing of exposures. This matters because studies have shown that traumas occurring specifically during childhood may influence later-

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life cognition.[4–7] Furthermore, the existing evidence of mixed results is primarily derived from cross-sectional studies in specialized populations (e.g., Holocaust survivors, former child laborers, or Aboriginal Australians), who have experienced above-average numbers of stressors in their lives.[5–8] The cross-sectional nature of these studies makes it impossible to rule out whether cognitive scores are associated with selection into the exposures. Moreover, it is unclear whether stressors impact cognitive decline over time. Finally, none of the existing studies have used population-based data, making it difficult to generalize findings.

The purpose of this study was to examine the impacts of experiencing TEs at different periods across the lifecourse on cognitive trajectories and dementia incidence in late life over 10 years of follow-up and to test whether the timing or total accumulation of trauma exposures impacted cognitive trajectories and incident dementia.

METHODS

Study Population

Data for this study came from the Health and Retirement Study (HRS), the largest ongoing nationally representative longitudinal survey of older adults in the U.S. HRS began in 1992 with >22,000 adults aged >50 years at baseline. Follow-up occurs every 2 years. HRS survey design and methods have been described previously.[9–11] This analysis used data from the 2006–2016 waves, with baseline psychosocial data from the 2006 and 2008 leave-behind Psychosocial and Lifestyle Questionnaires. Of the 9,950 participants who completed the baseline questionnaires, the cognitive trajectories analysis included 7,785 individuals who were aged ≥65 years at baseline (so the full, 35-point HRS-Telephone Interview for Cognitive Status [TICS] was assessed), had ≥2 cognitive data points, and had full covariate data. The predicted incident dementia analysis was further restricted to 4,780 individuals without predicted baseline dementia and dementia identification available from the Gianattasio algorithm.[12]

Measures

Participants were asked a 7-item question list developed for an ongoing study of health consequences of trauma in older adults from several sources[13,14]: (1) *Has a child of yours ever died?* (2) *Have you ever been in a major fire, flood, earthquake, or other natural disaster?* (3) *Have you ever fired a weapon in combat or been fired upon in combat?* (4) *Has your spouse, partner, or child ever been addicted to drugs or alcohol?* (5) *Were you the victim of a serious physical attack or assault in your life?* (6) *Did you ever have a life-threatening illness or accident?* And *(7) Did your spouse or a child of yours ever have a life-threatening illness?* Death of a spouse was added by classifying those whose marital status is widowed as an affirmative response to an eighth question. If participants indicated that they had experienced any of these events, they were subsequently asked in what year the event occurred.

Participants were also asked about experiences before age 18 years: (1) *did either of your parents drink or use drugs so often that it caused problems in the family?* and (2) *were you ever physically abused by either of your parents?* Appendix Figure 1 (available online) depicts the number of participants who experienced each event and the 50 most common combinations of events experienced.

Using participant responses, 2 measures were created: (1) an overall continuous TEs experienced across the life course and (2) a life period–specific dichotomous TEs experience. For overall, scores from both sets of questions were combined. For the life period–specific measure, variables for childhood (ages 0–17 years), early adulthood (ages 18–34 years), midlife (ages 35–64 years), and late life (ages ≥65 years) were created on the basis of the year participants reported the event to occur. If participants were missing data on the timing of their event, they were excluded from lifecourse period-specific analyses (sample is in Appendix Table 2, available online).

In HRS, global cognition is based on performance on an abbreviated version of the Telephone Interview for Cognitive Status (HRS-TICS). This continuous measure has a possible range of 0–35 and includes immediate and delayed recall, serial 7s, counting backward, object and person naming, and orientation to time. The psychometric properties of this HRS-TICS score have been assessed and determined to display satisfactory psychometric properties.[15]

HRS has no formal clinician-adjudicated diagnosis of dementia. Therefore, an approach previously described first by Hurd et al.[16] and subsequently modified by Gianattasio and colleagues,[12] which uses existing data collected from each wave of HRS to categorize participants' dementia status, was used. The Hurd paper took the biannually collected cognition data (HRS-TICS) and combined them with data on functional limitations, age, education, sex, and change in functional limitations and HRS-TICS in a model to predict dementia, training the algorithm in The Aging, Demographics, and Memory Study data set. This method also incorporated proxy data through the Informant Questionnaire on Cognitive Decline in the Elderly.[17,18] Gianattasio then defined race- and ethnicity-specific cutpoints, improving sensitivity and specificity within these groups. *Someone* was defined as having predicted incident dementia only if they remained below this threshold for the remainder of the follow-up.

Sociodemographic characteristics, including educational attainment, sex, age, HRS birth cohort, existing comorbidities (HRS comorbidities index: high blood pressure, diabetes, cancer, lung disease, heart disease, stroke, psychiatric problems, arthritis), and race/ethnicity, were collected in the 2006 or 2008 core interview, depending on the participant's respective baseline. Educational attainment was categorized as less than high school diploma, high-school diploma, some college, or a college diploma and higher. Race/ethnicity was categorized as non-Hispanic White, non-Hispanic Black, Hispanic, or other, and biological sex was self-reported as male or female.

Statistical Analysis

All statistical analyses were conducted in SAS 9.4 (SAS Institute, Inc., Cary, NC), and figures were created in R[19] using the ggplot2 package. Unweighted descriptive statistics were used to characterize the study population. To estimate the relationship between lifetime TEs and cognitive level and decline, linear mixed-effects models with a random slope and intercept were used. To assess the relationship between the exposures and predicted incident

dementia, stratified cumulative incidence functions were estimated using the Aalen–Johansen estimator, with death treated as a competing event. Inverse probability (IP) weights were used to account for informative censoring (on the basis of exposure, educational attainment, age, age,[2] baseline comorbidities, and change in cognition between 2 previous waves) and confounding (baseline age, age,[2] birth cohort, educational attainment, race/ethnicity, and biological sex). In addition, for lifecourse period-specific exposures, indicators (yes/no) of events in other lifecourse periods were included in confounding weights. Although events occurring after the exposure (e.g., midlife events occur after childhood events) are likely the mediators of the relationship, for this paper, we were particularly interested in the direct effects of events during each period, independent of subsequent events, so this mediating path was blocked. This minimally sufficient adjustment set was determined by directed acyclic graph analysis.[20] To further unravel the relationships and to check the built-in assumption in a summary score that each event matters the same amount to health, a sensitivity analysis was completed to assess the association between each individual TE and cognitive trajectories and predicted dementia.

RESULTS

The 7,785 adults who met the eligibility criteria for the cognitive trajectories analysis were 59% female and aged 74 years at baseline, on average. One quarter reported experiencing ≥3 TEs in their lifetime, whereas 22.4% reported never experiencing a TE. The mean baseline number of comorbidities was 2.2 (SD=1.4) and was higher with each consecutive TE category. The mean baseline HRS-TICS score was 22.1 (SD=4.6) and did not differ by number of events. The 4,741 with data on exposure timing were similar demographically to the full sample. The 4,780 adults who met the eligibility criteria for the predicted incident dementia analysis were older (mean age=77 years; range=67−99 years). Complete demographic and social characteristics of the populations for the cognitive decline analyses and predicted incident dementia analyses are shown in Table 1 and Appendix Tables 1 and 2 (available online), respectively.

Figure 1 and Appendix Table 4 (available online) depict the association between the total number of TEs experienced across the lifecourse (accumulation of risks model) and population mean cognitive level, before and after weighting. IP-weighted models showed that those experiencing any TEs (i.e., 1, 2, or ≥3) had higher mean HRS-TICS scores than those who experienced none. Those who experienced 2 events had HRS-TICS scores of 0.47 (95% CI=0.18, 0.75), and those with ≥3 events had scores of 0.43 (95% CI=0.14, 0.71) points higher than those who experienced no events. Figure 2 also shows the association between TEs experienced during a specific life period and cognitive function (Appendix Table 5, available online). Although there was no significant association between TEs experienced during midlife and HRS-TICS, there were significant associations for each other life period (sensitive periods model). Controlling for TEs experienced during other life periods to estimate the direct effects of each life period, those who experienced 1 or more TEs before age 19 years had a mean HRS-TICS score of 0.59 (95% CI=0.24, 0.94) points higher than those who experienced no TEs during early life, and those who experienced events during young adulthood had a mean HRS-TICS score of 0.49 (95% CI=0.11, 0.87) points higher. The association was largest among those who experienced TEs after the age of 65 years; these participants had a mean HRS-TICS score of 1.16 (95% CI=0.87, 1.46) points higher than those who experienced no events in later life.

Appendix Tables 2 and 3 (available online) and Figure 1 also show the association between the total number of TEs experienced across the lifecourse and cognitive decline over the course of follow-up. Experiencing any events over the entire lifecourse was statistically associated with the rate of change in HRS-TICS score in the study population. For each year of age, experiencing 1 or more TEs was associated with 0.04 (95% CI=0.01, 0.07) fewer HRS-TICS points, indicating an accelerated rate of cognitive decline compared with that among those who did not experience any TEs during their life. Figure 1 shows that this association is largely driven by those experiencing events later in life. For each year of age, experiencing one or more TEs after age 64 years was associated with 0.07 (95% CI=0.04, 0.10) fewer HRS-TICS points, indicating an accelerated rate of cognitive decline compared with that among those who did not experience any TEs later in life. However, experiencing TEs during other life periods was associated with a slightly slower decline or not associated with rate of decline.

Figure 2 depicts the IP-weighted cumulative predicted dementia incidence, stratified by experience of TEs. Panel a shows no meaningful difference in the risk of predicted dementia by the number of events experienced across the lifecourse. Panels b−e depict the cumulative incidence functions for each life period, showing no meaningful difference in the predicted dementia incidence by experiencing a TE during early life or midlife. Experiencing an event in young adulthood is associated with a slightly higher risk of predicted dementia from age 72 to 88 years, whereas experiencing a late-life event appears to initially be protective, with the association disappearing around age 80 years.

Analysis of each individual TE and its association with cognition and rate of decline indicates that type of event does matter. Those who experienced the loss of a child

Table 1. Baseline Sociodemographic and Cognitive Characteristics of Eligible Health and Retirement Study Participants, N=7,785

Variables	Overall, *n* (%)	0 traumatic events, *n* (%)	1 traumatic event, *n* (%)	2 traumatic events, *n* (%)	≥3 traumatic events, *n* (%)
Age, mean (range)	74 (65–104)	73 (65–94)	74 (65–99)	74 (65–96)	74 (65–104)
Sex, *n* (%), female	4,578 (58.8)	935 (53.7)	1,331 (59.3)	1,123 (60.8)	1,189 (60.9)
Education					
Less than high school	1,702 (21.9)	365 (21.0)	501 (22.3)	423 (22.9)	413 (21.1)
High-school diploma	2,998 (38.5)	706 (40.6)	887 (39.5)	689 (37.3)	716 (36.6)
Some college	1,584 (20.4)	320 (18.4)	403 (18.0)	385 (20.8)	476 (24.4)
College Diploma and higher	1,501 (19.3)	349 (20.1)	453 (20.2)	350 (19.0)	349 (17.9)
Race/ethnicity					
Non-Hispanic White	6,182 (79.4)	1,350 (77.6)	1,783 (79.5)	1,492 (80.8)	1,557 (79.7)
Non-Hispanic Black	948 (12.2)	208 (12.0)	262 (11.7)	233 (12.6)	245 (12.5)
Hispanic	535 (6.9)	155 (8.9)	156 (7.0)	102 (5.5)	122 (6.2)
Other	120 (1.5)	27 (1.6)	43 (1.9)	20 (1.1)	30 (1.5)
TICS Score, mean (SD)	22 (4.6)	22 (4.5)	22 (4.8)	22 (4.5)	22 (4.6)
Lifetime traumatic events					
0 events	1,740 (22.4)				
1 event	2,244 (28.8)				
2 events	1,847 (23.7)				
≥3 events	1,954 (25.1)				
Lifetime traumatic events (*n*, % 1 or more)					
Early life	708 (9.1)	0 (0.0)	197 (8.8)	219 (11.9)	292 (14.9)
Young adulthood	634 (8.1)	0 (0.0)	144 (6.4)	204 (11.0)	286 (14.6)
Mid life	1,484 (19.1)	0 (0.0)	506 (22.6)	447 (24.2)	531 (27.2)
Late life	1,534 (19.7)	0 (0.0)	593 (26.4)	468 (25.3)	473 (24.2)
Missing timing data	3,044 (39.1)	801	998	1,245	692
Comorbidities, mean (SD)	2.2 (1.35)	1.9 (1.25)	2.1 (1.28)	2.3 (1.32)	2.6 (1.46)

TICS, Telephone Interview for Cognitive Status.

or were the victim of a serious physical attack during their life had significantly lower HRS-TICS scores than those who did not experience these types of events (−0.95, 95% CI= −1.22, −0.68; −0.68, 95% CI= −1.15, −0.21 respectively). By contrast, those who had ever been in a major natural disaster or whose spouse, child, or other close relation had ever had a life-threatening illness or accident had higher HRS-TICS scores than those who had not experienced that (0.51, 95% CI=0.24, 0.79; 0.32, 95% CI=0.49, 0.97, respectively). The association of TEs with the rate of cognitive decline appears to be driven primarily by experiencing the death of a spouse, which for every 5 years of age was associated with 0.44 (95% CI= −56, −0.32) more HRS-TICS points of decline than among those who had not experienced the death of a spouse. These associations are depicted in Figure 3. This pattern was not borne out with predicted dementia incidence. However, there was an association only when comparing those who were physically abused by either parent during childhood (Appendix Figure 2, available online).

DISCUSSION

This study found that overall, lifetime experience of TEs was associated with faster cognitive decline in a U.S. population-based survey of adults aged >65 years. However, when investigating the life period–specific occurrence of these events, several notable trends were identified. The associations with both cognitive level and rate of decline appear to be largely driven by events experienced later in life. In sensitivity analyses of specific TEs, the death of a child and being the victim of a serious physical assault were associated with lower cognitive

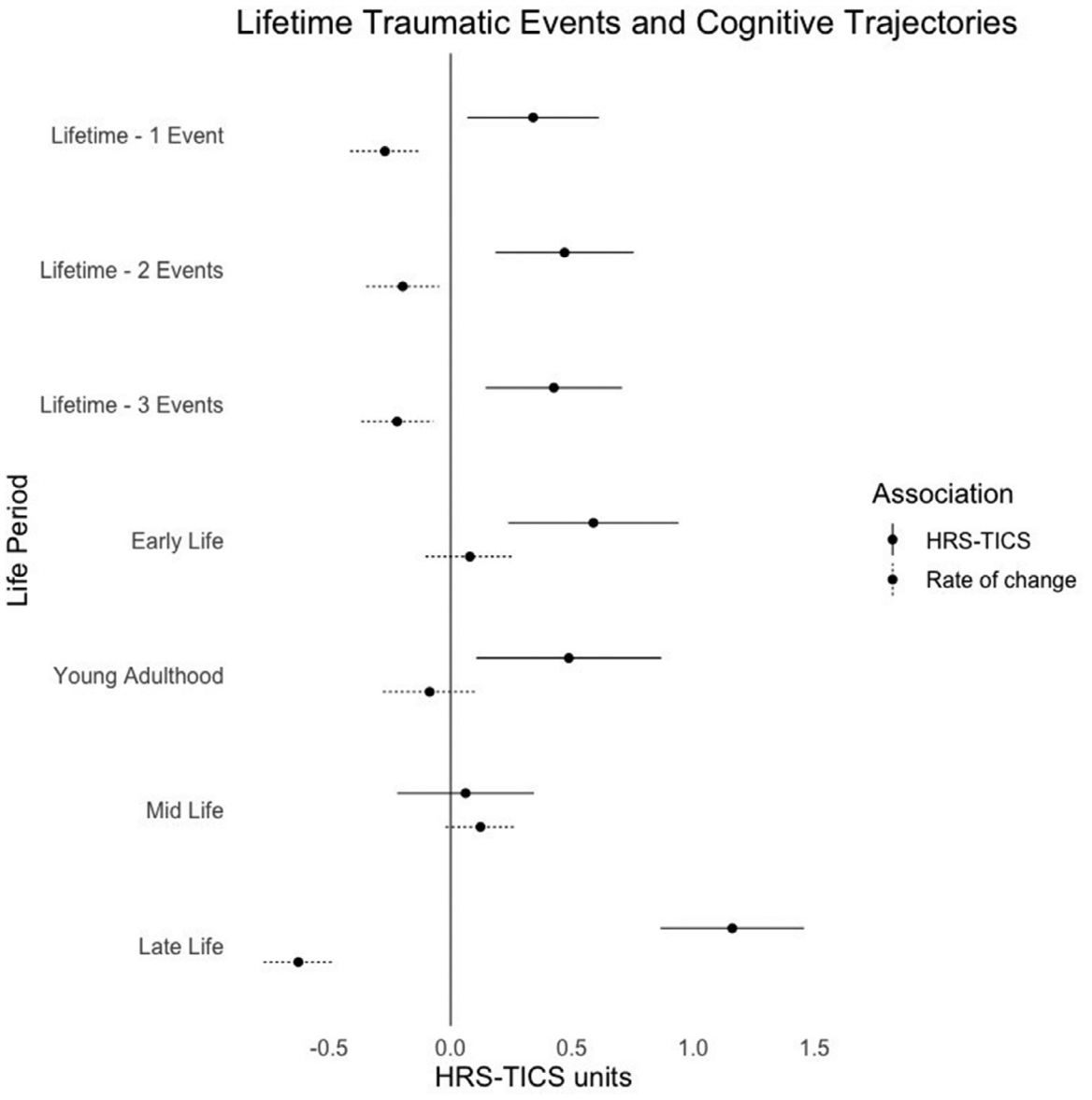

Figure 1. Fixed-effects associations between lifecourse experiences of traumatic events and mean population cognitive function and cognitive decline.

Note: This figure shows the fixed-effects beta estimates and 95% CIs for the exposure (main effect) and exposure X age (slope) terms from linear mixed-effects models assessing the association between lifecourse traumatic events (accumulation and sensitive period models, specified on the y-axis) and cognitive function (HRS-TICS, specified on the x-axis; a higher score is equivalent to higher cognitive function). For example, those who experienced 1 or more traumatic events before the age of 19 years had a mean HRS-TICS score of 0.59 (95% CI=0.24, 0.94) points higher than those who experienced no traumatic events during early life. For each year of age, experiencing 1 or more traumatic events after age 64 years (late life) was associated with 0.07 (95% CI=0.04, 0.10) fewer HRS-TICS points, indicating an accelerated rate of cognitive decline compared with that among those who did not experience any traumatic events later in life.

HRS-TICS, Health and Retirement Study Telephone Interview for Cognitive Status.

function. By contrast, being in a major natural disaster or having a close relative experience a life-threatening illness or accident were associated with better cognition. Moreover, lifetime TE exposure was associated with higher population mean estimates of cognitive function; however, the magnitudes of these associations were relatively small. A model regressing cognition on age estimates that cognition decreases by 0.32 HRS-TICS units/year; the present mean cognition estimates are roughly equivalent to 1−3 fewer years of cognitive aging, whereas the rate of change estimates are even smaller but may be clinically meaningful when accumulated over

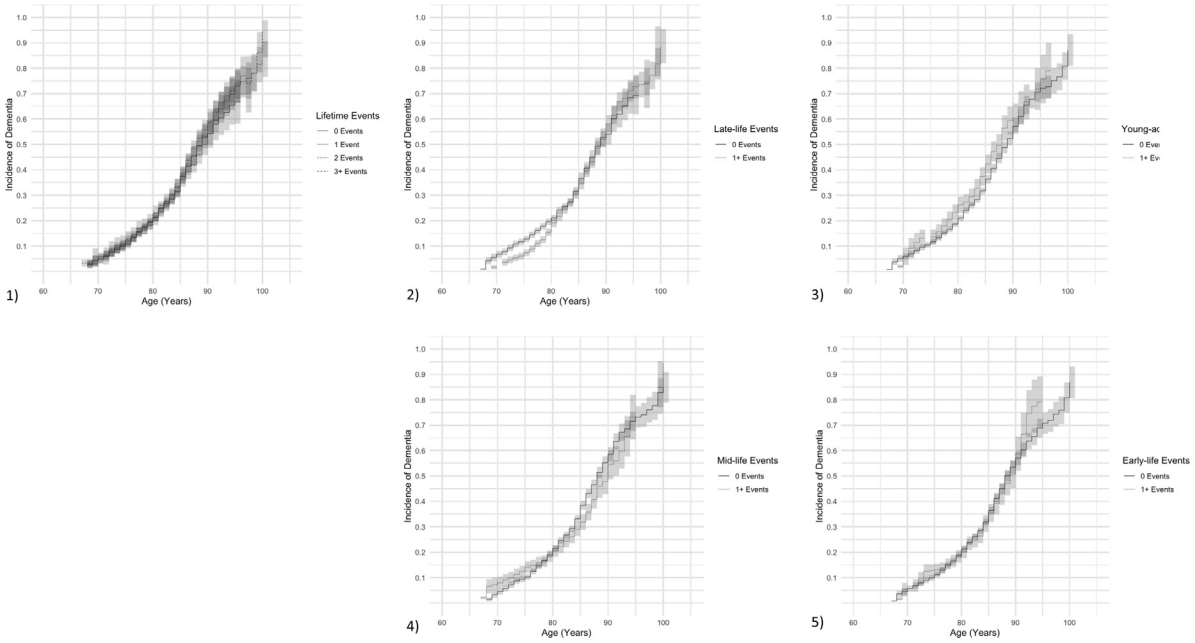

Figure 2. Cumulative predicted dementia incidence by lifecourse traumatic events.

Note: This figure depicts the cumulative predicted dementia incidence, IP weighted to account for censoring and confounding, for each form of the exposure. Panels show the incidence stratified by (1) cumulative lifecourse traumatic events (0, 1, 2, or ≥3 events); (2) early-life events; (3) young-adulthood events; (4) midlife events; and (5) late-life events. Cumulative incidence functions were estimated using the Aalen–Johansen estimator. There is a significant difference between the predicted incidence of dementia by number of lifecourse traumatic events overall or during specific life periods, except for later life events. In a small window (ages 84–97 years), there is a slightly lower predicted incidence of dementia for those who experience traumatic events in late life.

IP, inverse probability.

decades. Finally, lifetime TEs were not associated with predicted dementia incidence over a 10-year period. However, the results for all analyses should not be overinterpreted owing to several limitations discussed below.

Previous research has found mixed associations between TEs and cognitive function and decline. Several studies have found that an accumulation of stressful events or TEs is associated with lower performance in specific domains of cognitive function or mild cognitive impairment.[21,22] In the Irish Longitudinal Study on Ageing, researchers found that a history of childhood sexual abuse was associated with better global cognition, memory, executive function, and processing speed among adults aged ≥50 years.[23] The authors acknowledged that this finding was counterintuitive and hypothesize that it may be a reflection of a chronic lowering of cortisol secretion (rather than heightening) or a state of hyperarousal causing upregulation of noradrenergic activity and promotion of cognitive reserve.

Similar to these results for specific TEs, an older study by Grimby et al.[24] found that cognitive decline was not associated with stressful life events, except in the case of bereavement, where experiencing the death of a spouse or child was associated with larger declines in cognitive abilities, suggesting that graver TEs may be more impactful. Finally, Tschanz and colleagues[25] found that stressful life events' effect on cognition over 7 years varies by educational attainment and type of stressful life events. The results from this study are consistent with these findings because they show the associations of TEs overall with better cognition, depending on the event. There are many analogies where a small amount of stress can activate the immune system and be protective for health in the short term but detrimental at high levels for extended periods (e.g., exercise). This model of brief stressor-induced health protection could help to explain these findings because they are also related to differences by event type and timing. However, it is also impossible to know whether these unexpected cross-sectional associations are spurious or owing to selection biases (discussed in limitations).

This study has several strengths, including the use of a large, U.S. population–based longitudinal cohort and up to 10 years of follow-up cognitive data. Furthermore, HRS provided rare details on 10 TEs and when they occurred.

Limitations

This study also has several limitations. There is measurement error in the cognitive measures; HRS-TICS is a

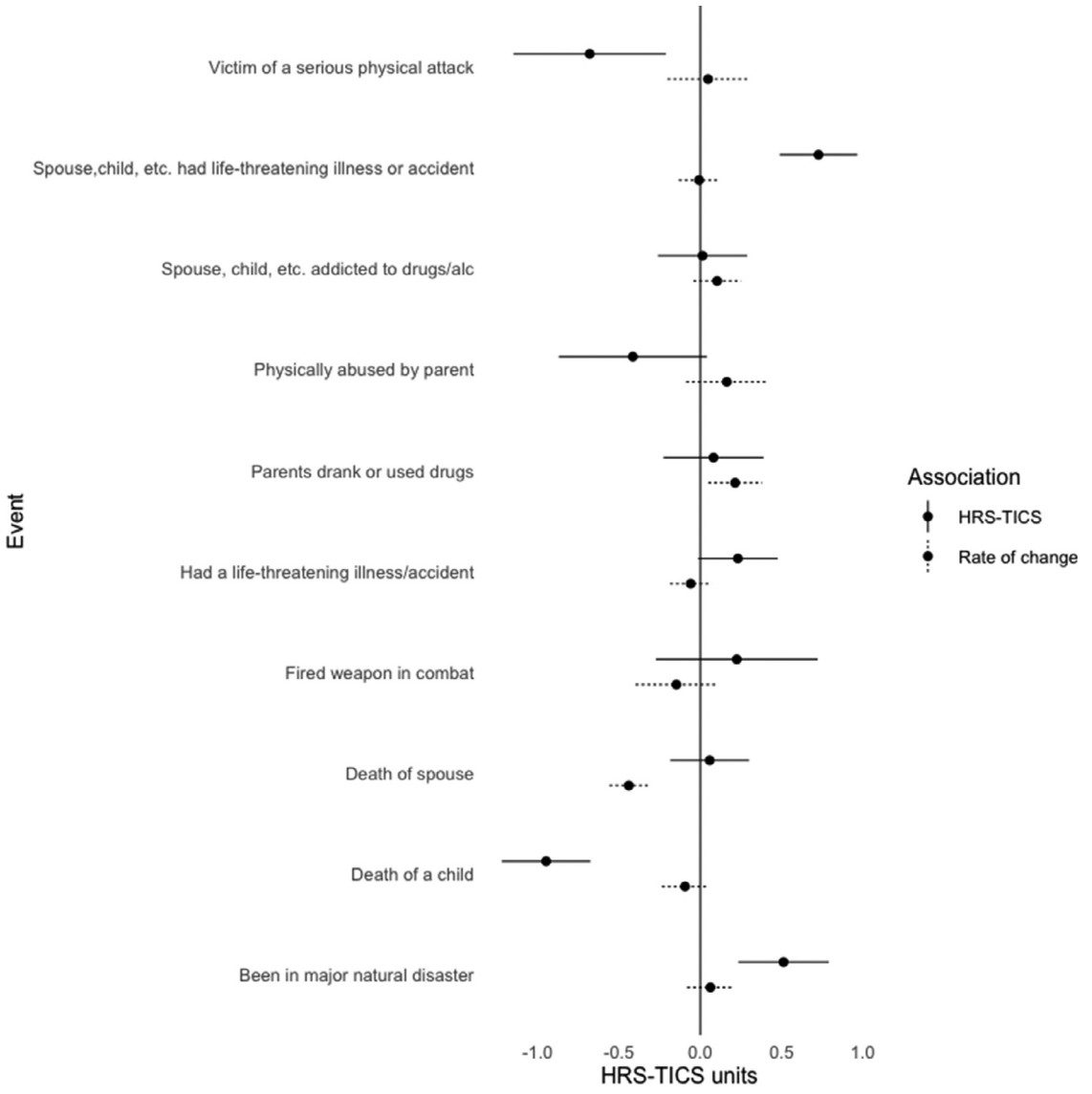

Figure 3. Fixed-effects associations between individual traumatic events and mean population cognitive function and cognitive decline.

Note: This figure shows the fixed-effects beta estimates and 95% CIs for the exposure (main effect) and exposure X age (slope) terms from linear mixed-effects models assessing the association between each specific traumatic event (specified on the y-axis) and cognitive function (HRS-TICS, specified on the x-axis).

HRS-TICS, Health and Retirement Study Telephone Interview for Cognitive Status.

limited measure of global cognition, and the algorithm used to diagnose dementia has limited sensitivity and specificity and was initially designed to diagnose prevalent dementia, not incident. Although the length of follow-up is longer than that used in most existing studies, it is possible that the maximum time of 12 years in the study and the average follow-up of 9 years (4.5 waves) in this study contributes to the null findings with predicted dementia incidence: the progression of brain pathophysiology that leads to dementia can occur over decades. There is also the potential for bias in these results owing to unmeasured confounding, and the measure of perceived stress was not included. Of particular concern is the potential for recall bias, that is, those with a better memory (resulting in a higher global cognition score) would likely more accurately report their exposure because it is measured by self-report. In this case, those with better cognitive scores would be more likely to accurately report being exposed, although the events were very significant and might be less susceptible to

recall bias than others. Moreover, a limited set of events was examined; it is possible that other TEs may be linked to cognitive outcomes. Further research on other impacts of significant stressors, such as structural racism, discrimination, and immigration experiences, among older Black, indigenous, and people of color is needed. Along with other TEs, the modifying and/or mediating nature of factors such as social support and coping skills on these relationships may be importantly influencing these results.

One of the most important limitations of this research is selection bias. The population investigated includes only individuals who survived to age 65 years and were healthy enough to enroll. This leaves a large amount of time between exposure and study enrollment, particularly for early-life and young-adulthood TEs. TEs may have had unobserved health impacts before study enrollment; those who experienced such events may have been less likely to enroll or may have been less likely to survive to eligibility. This bias would affect both the mean cognition and the cognitive decline results. Although there is little to be done about this issue in the existing cohort, including these exposures and outcomes in future longitudinal cohort studies of younger individuals would help to mitigate these limitations. Therefore, it is important to note that these exposures should not be interpreted as brain protective, for the reasons described earlier.

CONCLUSIONS

These findings suggest that later-life traumas may impact the rate of cognitive decline, but the results should be interpreted cautiously. Studies examining the impact of treatment and support for coping with traumatic experiences in later life may help to identify interventions and methods for buffering the potential impact of trauma. This study highlights that the use of composite scores of negative or stressful experiences may mask true effects on health outcomes, and a more nuanced approach to modeling the timing and impact of stressors is warranted. Although these results are not causal, knowing patients' histories with respect to TEs, understanding how these experiences may indicate a higher risk of accelerated cognitive aging, and incorporating that information into monitoring and care plans for patients could improve clinical practice.

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CREDIT AUTHOR STATEMENT

Rebecca C. Stebbins: Conceptualization, Formal analysis, Methodology, Visualization, Writing - original draft. Joanna Maselko: Conceptualization, Supervision, Writing - review and editing. Y. Claire Yang: Methodology, Supervision, Writing - review and editing. Brenda L. Plassman: Methodology, Supervision, Writing - review and editing. Jessie K. Edwards: Methodology, Supervision, Writing — review and editing. Allison E. Aiello: Conceptualization, Supervision, Writing - review and editing.

SUPPLEMENTAL MATERIAL

Supplemental materials associated with this article can be found in the online version at https://doi.org/10.1016/j.amepre.2022.05.007.

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