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The Effects of Lifetime Trauma Exposure on Cognitive Functioning in Midlife

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Accumulating evidence suggests that lifetime trauma exposure is associated with adulthood cognitive functioning. However, the nature and extent of this relation have yet to be fully explored. We used multilevel modeling to examine trauma exposure and age at first trauma exposure as predictors of the level of and change in cognitive functioning over a 9-year period. Data were from the Midlife in the United States study, a national survey that began in 1995. Data regarding trauma exposure and age at first exposure were obtained from the 2004 wave, whereas cognitive data were obtained from the 2004 and 2013 waves. The analyses were conducted using data from the 2,471 participants (age range: 28–84 years) who had complete data on all variables from the 2004 wave. Lifetime trauma exposure predicted change in executive functioning (EF), $B = -0.03$, $SE = 0.01$, $p = .015$, 95% CI [−0.05, −0.01]; and episodic memory, $B = -0.05$, $SE = 0.02$, $p = .023$, 95% CI [−0.10, −0.01], such that individuals with more trauma exposure had more decline over 9 years. Age at first exposure also predicted change in EF, $B = -0.002$, $SE = 0.00$, $p = .009$, 95% CI [−0.004, −0.001], such that individuals who were first exposed to trauma later in life had greater EF decline than individuals whose first traumatic event occurred earlier in life. Delta pseudo-R^2 values were moderate, Δpseudo$R^2 =$ .17–.39. These findings identify trauma exposure as a risk factor for cognitive decline in adulthood and highlight the elevated risk associated with adulthood trauma exposure.

Both childhood trauma exposure (Felitti et al., 1998; Kelly-Irving et al., 2013; Lee, Tsenkova, & Carr, 2014; Levine, Miller, Lachman, Seeman, & Chen, 2018; Wegman & Stetler, 2009) and lifetime trauma exposure (Elliot, Turiano, Infurna, Lachman, & Chapman, 2018; Krause, Shaw, & Cairney, 2004; O'Donovan, Neylan, Metzler, & Cohen, 2012) are associated with negative health outcomes in adulthood. A question remains as to whether lifetime trauma exposure is similarly associated with negative outcomes in adulthood cognitive functioning. Evidence that supports such an association has come from findings on cognitive impairments in posttraumatic stress disorder (PTSD; Brewin, 2014; Honzel, Justus, & Swick, 2014; Polak, Witteveen, Reitsma, & Olff, 2012) and the long-term effects of childhood trauma exposure on adulthood cognition (Gould et al., 2012; Majer, Nater, Lin, Capuron, & Reeves, 2010). However, these findings are limited by the populations and types of trauma exposure the studies have examined. Furthermore, there is conflicting evidence as to whether the age at which a person experiences a traumatic event plays a role in the occurrence and extent of long-term negative outcomes in adulthood (Krause et al., 2004; McCutcheon et al., 2010; Straussner & Calnan, 2014). In the present study, we explored the association between trauma exposure, age at first exposure, and cognitive functioning, defined herein as executive functioning (EF) and episodic memory (EM), to clarify the lasting effects of trauma exposure and age at first exposure on the level of and change in adulthood cognitive functioning.

Evidence from previous studies suggests that childhood trauma exposure is associated with poor adulthood cognitive functioning, although there is little clarity on the nature and extent of the cognitive problems. Majer et al. (2010) examined cognitive functioning in a small sample of individuals with and without a history of childhood trauma exposure in the form of emotional abuse, physical abuse, or physical neglect. Individuals who had experienced childhood trauma made more errors on a spatial working memory task; however, there were no significant group differences for tasks that assessed spatial recognition memory, pattern recognition memory, or executive function. Gould et al. (2012) identified an association between childhood trauma exposure and adulthood visual memory, spatial working memory, and executive functioning. However, the study sample was relatively small ($N = 93$) and did not include any individuals with childhood trauma exposure who did not subsequently develop depression or PTSD. Although it is difficult to draw strong conclusions from limited samples, these

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findings do suggest that trauma exposure in childhood can have lasting effects on cognitive functioning in adulthood.

Separately, there is evidence to suggest that exposure to traumatic events in adulthood affects cognitive functioning in several ways. Specifically, individuals with PTSD have been shown to demonstrate poorer performance on tasks assessing executive functioning (Polak et al., 2012), episodic memory (Brewin, 2014), and working memory (Honzel et al., 2014). It is important to note that although these findings focus on trauma-exposed individuals with a PTSD diagnosis, not all individuals exposed to traumatic events develop a subsequent stress-related disorder. Further, it is not clear whether the cognitive deficits found in individuals with PTSD persist either over time or when these individuals no longer meet the diagnostic criteria for PTSD. Therefore, in order to effectively examine the lasting cognitive effects of trauma exposure, rather than the poor cognitive performance found with PTSD, the study of trauma exposure and cognitive functioning should expand to include longitudinal data and a broader sample of individuals with lifetime trauma exposure.

Finally, a question remains as to whether age at exposure—that is, the age at which a person experiences a traumatic event—plays a role in the occurrence and extent of negative health outcomes in adulthood. Krause et al. (2004) found that exposure to traumatic events in young and middle adulthood negatively impacted health in later life more than traumatic events experienced in childhood. This finding may reflect a recency effect, such that more recent traumatic experiences affect general and mental health more than traumatic experiences that occurred further in the past. Yet, there is also evidence to suggest that trauma exposure in childhood is more harmful than exposure later in life (McCutcheon et al., 2010; Straussner & Calnan, 2014), which may be because childhood represents a unique period of development associated with increased susceptibility to risk. In fact, a recent study evaluated the differential risk of lifetime trauma exposure and adverse childhood experiences (ACE) on PTSD symptoms; the results indicated that both types of life events were independently associated with higher levels of PTSD symptoms (Frewen, Zhu, & Lanius, 2019). These divergent findings make it difficult to form a prediction regarding the effect of age at exposure on various cognitive outcomes. To shed light on this question, the present study explored the association between age at exposure and cognitive functioning in adulthood, as well as the potential moderating effect of age at exposure on the relation between trauma exposure and cognitive functioning. The aim of the present study was not to examine the differential effects of ACE and lifetime trauma exposure but rather to focus on whether the effects of trauma exposure are more pronounced if the exposure occurred earlier or later in life. In this study, only the age at which an individual first experienced a traumatic event was evaluated, herein referred to as "age at first exposure."

In the present study, we evaluated the association between lifetime trauma exposure and cognitive functioning during adulthood. Trauma exposure is defined here as threatening or physically or emotionally harmful events that cause lasting adverse effects on an individual's level of well-being or functional impairment (Pai, Suris, & North, 2017; but see American Psychiatric Association, 2013). Cognitive functioning, defined here as episodic memory (EM) and executive functioning (EF), was examined as both the level of functioning and the change in functioning over time, thus extending beyond the existing literature. Level of functioning refers to between-person individual differences in cognitive scores at one point in time, whereas a change in functioning examines the within-person differences (i.e., interindividual differences in intraindividual change) in performance over two assessment points, 9 years apart. We posited three hypotheses. First, we predicted that individuals with more trauma exposure would have lower levels of cognitive functioning and more cognitive decline in adulthood compared to those with less trauma exposure. Second, we predicted that trauma exposure that occurred later in life would be associated with more cognitive decline than exposure that occurred earlier in life, following from the evidence that adulthood trauma negatively affects health more than childhood trauma (Krause et al., 2004). Finally, we predicted that the association between trauma exposure and cognitive functioning in adulthood would be moderated by age at first exposure, such that the effect of trauma exposure on cognitive functioning would be greater for individuals with exposure that occurred later in life compared to those who experienced earlier initial trauma exposure.

Method

Participants and Procedure

Data for this study were obtained from the Midlife Development in the United States (MIDUS), a longitudinal survey that began in 1995. Participants for a national sample of over 7,000 U.S. residents aged 25–74 years were recruited using random-digit dialing, and 4,963 participants were successfully retained for Wave 2 of MIDUS, a follow-up study that ran from 2004 to 2006 (Radler & Ryff, 2010). Although the MIDUS sample was based on a probability sample, minorities and individuals with lower income levels and less educational attainment are underrepresented in the sample. Participants who were successfully recruited for Wave 2 differed from those who dropped out in that they were more highly educated, more likely to be women, more likely to be White, and had higher scores on self-report measures of health (Hughes, Agrigoroaei, Jeong, Bruzesse, & Lachman, 2018). Wave 3 of MIDUS, which took place from 2013 to 2015, successfully retained 3,294 participants, who were more highly educated, more likely to be women, more likely to be White, and had higher ratings on self-report measures of health than those who dropped out between Waves 2 and 3 (Hughes et al., 2018). Of the Wave 3 sample, 89.5% of participants described their racial background as White, 3.7% as Black or African American, 0.9% as Native American or Alaska Native Aleutian Islander/Eskimo, 0.4% as Asian, and 5.5% as other. Regarding educational attainment, 29.1% of participants

had a high school diploma or less, 29% had completed some college, 21.9% had a bachelor's degree, and 20% had completed some level of graduate school or higher. Participants in the Wave 3 sample were between 39 and 93 years of age.

Cognitive data were not available at Wave 1; thus, the current study only utilized data from Wave 2 and Wave 3 of MIDUS. Cognition was assessed in a separate telephone call after the main survey and Self-Administered Questionnaire (SAQ) were completed. After excluding participants who did not have complete data on the Wave 2 variables, a requirement for multilevel analysis, 2,471 participants remained. Of those, 1,604 had complete Wave 3 cognitive data. The mean age of the remaining participants was 55.43 years ($SD = 12.45$), the mean household income was $71,364 (USD; $SD = $60,463), and the average educational attainment was 14.28 years ($SD = 2.62$). On self-report questionnaires related to health, the mean participant rating for physical health was 3.81 ($SD = 0.93$), and the mean rating for mental health was 3.54 ($SD = 1.02$); scores of 5 indicated the best possible health rating. This research was approved by the Brandeis University institutional review board.

Measures

Trauma exposure. The SAQ from Wave 2 of MIDUS included an abbreviated version of the Life Experiences Survey (LES; Sarason, Johnson, & Siegel 1978). Participants were asked to indicate which events they had experienced and the age at which these events occurred. For each experience, participants were also asked to indicate "if [the event] affected you, positively or negatively, both initially, and in the long run," using a 5-point scale ranging from -2 (*very negatively*) to 2 (*very positively*). As the relevance of certain events in the context of trauma exposure differs across age groups, the questionnaire was structured such that respondents were first presented with a subset of events that were to be endorsed only if they occurred during the childhood or teenage years; these were followed by events that could have occurred at any time. The original LES has demonstrated adequate test–retest reliability (e.g., Cronbach's αs = .63−.64; Sarason et al., 1978). As the measure includes many events that are nontraumatic life stressors (Frewen, Zhu, & Lanius, 2019), the present study looked exclusively at 12 stressful life events considered the most potentially traumatic. Although individuals may interpret the impact of events differently, the selection of these events was informed by other studies in which the same events were used to measure lifetime trauma exposure (e.g., Turner & Lloyd, 1995; Krause et al., 2004; Elliot et al., 2018). Three of these experiences—being sent away from home, parent alcohol abuse, and parent drug abuse—were specific to childhood or teenage years. We also chose to include parental divorce in this group, as it was determined that the event carries more relevance in childhood or teenage years. The remaining eight events could have occurred at any time, and included parental death; sibling death; child death; child life-threatening illness; physical assault; sexual assault; combat experience; and losing a home to fire, flood, or natural disaster. As participants were able to record having experienced each event only once, trauma exposure was calculated as the total number of events experienced, with a possible range of 0–12.

Age at first trauma exposure. The stressful life events checklist portion of the SAQ asked participants at what age each traumatic experience occurred. Individuals reported varying amounts of trauma exposure; for the purposes of the present study, only the age of the first reported traumatic event was evaluated.

Cognitive functioning. Two summary measures developed by Lachman, Agrigoroaei, Tun, and Weaver (2014) that assess executive function (EF) and episodic memory (EM) were employed to evaluate cognitive functioning. The measures were formulated using results from the Brief Test of Adult Cognition (BTACT), which was administered over the phone at Waves 2 and 3. The BTACT was designed to examine cognition in adults of varying age and educational attainment and to be sensitive to changes associated with normal cognitive aging. The BTACT has demonstrated good test–retest and alternate forms reliability (e.g., Cronbach's αs = .28–.94 and .30–93) as well as convergent and discriminant validity (see Lachman et al., 2014, for details). The EM summary uses word lists for tasks that assess immediate and delayed recall, whereas the EF measure is broader and includes assessments of inductive reasoning, inhibition, and attention switching, using the following tasks: category fluency, number series, backward counting, stop-and-go switch, and digit span backward (see Lachman et al., 2014, for design and scoring information for each task). Factor scores were computed as the average of the z scores for the subtests, using the means and standard deviations from Wave 2 to allow for examination of change. As cognitive data were not available prior to Wave 2, the present study focused on change in cognitive function from Wave 2 to Wave 3.

In the present study, we examined both the level of and change in EF and EM. By examining participants' level of cognitive functioning, we were able to evaluate whether trauma exposure was associated with individual differences in cognitive performance. Separately, examinations of change in cognitive functioning were used to indicate whether trauma exposure resulted in larger declines in cognitive aging. Furthermore, although an individual's level of functioning may depend more on childhood cognitive abilities than on individual differences in adulthood, it is less likely that change in cognition over 9 years would depend on childhood cognition. Finally, the BTACT was designed to assess changes associated with normal cognitive aging and not dementia, per se; thus, clinical cutoffs have not been established.

Covariates. Age, educational attainment, income level, physical health, and mental health were included as covariates, as these variables are associated with both the independent and dependent variables. Educational attainment and income level

Table 1
Descriptive Statistics for Study Variables

Variable	*M*	*SD*	Range
Age (years)	55.43	12.45	28–84
Income (USD)	71,364	60,463	0–300,000
Physical health score	3.81	0.93	1–5
Mental health score	3.54	1.02	1–5
Educational attainment (years)	14.28	2.62	6–20
Trauma exposure count	1.87	1.32	0–12
Age at first exposure (years)	25.77	16.93	0–84
MIDUS Wave 2 Executive Functioning	−0.01	0.69	−5.10–2.34
MIDUS Wave 2 Episodic Memory	−0.01	0.95	−2.94–3.64
MIDUS Wave 3 Executive Functioning	−0.15	0.74	−5.63–2.02
MIDUS Wave 3 Episodic Memory	−0.04	0.99	−2.94–3.64

Note. MIDUS = Midlife in the United States study.

Table 2
Frequency of Traumatic Events Reported in the Midlife in the United States (MIDUS) Study

Variable	%
Events that occurred during childhood or teenage years	
Sent away from home	3.9
Parent alcohol abuse	23.9
Parent drug use	1.4
Parental divorce	17.3
Events that occurred at any time	
Parent died	78.3
Sibling died	37.3
Child died	14.2
Child life-threatening illness	13.6
Lost home to fire, flood, etc.	6.5
Physical assault	12.7
Sexual assault	14.9
Experienced combat	8.7

Note. Data are from MIDUS Wave 2.

were considered to be representative of an individual's socioeconomic status. Educational attainment was calculated as total years of education (range: 6 –20). Income level was based on total household income, with a range of $0 (USD) to $300,000. Physical and mental health were determined based on responses provided on a self-report health questionnaire, with possible responses for each measure ranging from 1 (*excellent*) to 5 (*poor*); responses were reverse-coded prior to analysis, such that higher scores were indicative of better health. Age was the age of the individual at the time of Wave 2 data collection.

Data Analysis

Descriptive statistics were generated for all study variables. The following were mean-centered prior to analysis: trauma exposure, age at first exposure, age, educational attainment, income level, physical health, and mental health. A series of multilevel models were fit to evaluate trauma exposure, age at first exposure, and the interaction between the two as predictors of both level of and change in cognitive functioning. The models were fit with maximum likelihood estimation in the R statistical software (Version 3.6.1; R Core Team, 2019) using the lme4 package (Bates, Maechler, Bolker, & Walker, 2015). Confidence intervals and *p* values were calculated using the lmerTest package (Kuznetsova, Brockhoff, & Christensen, 2017) with Satterthwaite's approximation for degrees of freedom (Littell, Milliken, Stroup, & Wolfinger, 1996).

The models were each run twice, first with EF as the outcome variable, then with EM. An initial model that included only time and covariates was tested. Model 1 included trauma exposure as a predictor of the intercept (Level 2) and the slope of time (Level 1). Model 2 added age at first exposure, and Model 3 added the interaction between trauma exposure and age at first exposure. Each model was specified with a Level 2 random intercept and a Level 1 residual. The slope of time was not allowed to vary. The age at first exposure variable was created only for individuals who experienced some level of trauma exposure; therefore, Models 2 and 3 were run on the subset of the sample who reported trauma exposure ($n = 2{,}815$). The best-fit model for EF and EM was determined using the Bayesian information criterion (BIC), a model fit statistic for which a smaller value indicates better predictive performance of the model.

Results

Descriptive Results

Descriptive statistics are presented in Table 1. The average number of traumatic events reported was 1.87 ($SD = 1.32$). The average age at which individuals experienced their first traumatic event was 25.77 years ($SD = 16.93$). Descriptive statistics for each traumatic event are reported in Table 2.

Table 3
Multilevel Models Predicting Level of and Change in Executive Functioning in the Midlife in the United States Study (MIDUS) From 2004 to 2014

	Model 1		Model 2		Model 3	
Variable	*B*	*SE*	*B*	*SE*	*B*	*SE*
Intercept	0.03*	0.01	0.05***	0.01	0.04**	0.01
Time	−0.25***	0.01	−0.26***	0.01	−0.26***	0.01
Age	−0.02***	0.00	−0.02***	0.00	−0.02***	0.00
Income	0.00	0.00	0.00	0.00	0.00	0.00
Mental health	0.08***	0.01	0.08***	0.01	0.08***	0.01
Physical health	0.04***	0.01	0.03*	0.01	0.03*	0.01
Education	0.07***	0.00	0.07***	0.00	0.07***	0.00
Trauma	−0.00	0.01	−0.01	0.01	−0.02	0.01
Age at Exposure[a]			0.00	0.00	0.00	0.00
Trauma × Age at Exposure[a]					−0.00	0.00
Time × Trauma	−0.02*	0.01	−0.03*	0.01	−0.04**	0.01
Time × Age at Exposure[a]			−0.00**	0.00	−0.00**	0.00
Time × Trauma × Age at Exposure[a]					−0.00	0.00
Model fit	BIC = 7,395.67		BIC = 6,272.99		BIC = 6,285.94	

Note. BIC = Bayesian information criterion.
[a]Age at first trauma exposure.
*$p < .05$. **$p < .01$. ***$p < .001$.

Of the subset of events that occurred at any time, the most commonly reported event was the death of a parent (78.3%), followed by the death of a sibling (37.3%) and sexual assault (14.9%). Of the events that occurred specifically during the childhood or teenage years, the most commonly reported event was parent alcohol abuse (23.9%), followed by parental divorce (17.3%).

Sensitivity Analyses

Sensitivity analyses were conducted to evaluate whether including new trauma (i.e., the number of additional traumatic events participants experienced between Wave 2 and Wave 3 of MIDUS) significantly affected the outcome of the model for either EF or EM, to establish whether any significant effects of trauma exposure on cognitive functioning were driven by recent traumatic experiences. The events from childhood or teenage years (e.g., being sent away from home, parent alcohol abuse, parent drug abuse, parental divorce) were not included in the new-trauma variable. Using likelihood ratio testing and employing a $p < .05$ criterion, the models for EF and EM were compared to models that included the new-trauma variable as an additional predictor. As the inclusion of the new-trauma variable did not significantly change the outcome of the model for EF or EM, we focused on the results from the models without the new-trauma variable.

Executive Functioning

An initial model was estimated with time and covariates as predictors of EF. The results showed a significant decline in EF over the 9 years between Wave 2 and Wave 3 of MIDUS, $B = -0.25$, $SE = 0.01$, $p < .001$, 95% CI [−0.27, −0.23] (see Hughes et al., 2018, for more details). Table 3 shows the results of the three multilevel linear models predicting the level of and change in EF from Wave 2 to Wave 3. The interaction between trauma exposure and age at first exposure was not significant and did not improve the predictive performance of the model, failing to support our third hypothesis (i.e., that the association between trauma exposure and cognitive functioning in adulthood would be moderated by age at first exposure). Therefore, the best-fit model was Model 2.

In line with our first hypothesis (i.e., that individuals with more trauma exposure would have lower levels of cognitive functioning and more cognitive decline in adulthood compared to those with less trauma exposure), trauma exposure significantly predicted change in EF such that individuals with more trauma exposure had higher levels of EF decline, $B = -0.03$, $SE = 0.01$, $p = .015$, 95% CI [−0.05, −0.01]. However, trauma exposure did not predict EF level, $B = -0.01$, $SE = 0.01$, $p = .326$, 95% CI [−0.04, 0.01]. Our second hypothesis (i.e., that trauma exposure that occurred later in life would be associated with more cognitive decline than exposure that occurred earlier in life) was also partially supported; age at first exposure significantly predicted change in EF, $B = -0.002$, $SE = 0.00$, $p = .009$, 95% CI [−0.004, −0.001], such that individuals whose

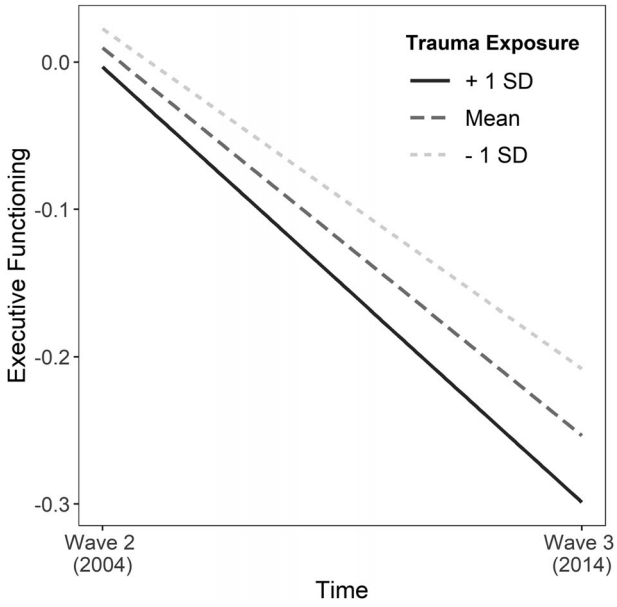

Figure 1. Change in executive functioning as a function of trauma exposure. Executive functioning is from Wave 2 and Wave 3 of the Midlife in the United States (MIDUS) study; trauma exposure is from MIDUS Wave 2.

first traumatic event occurred later in life had more EF decline than individuals whose first event occurred earlier in life. However, age at first exposure did not predict the level of EF, $B = 0.00$, $SE = 0.00$, $p = .061$, 95% CI [−0.00, 0.00]. Figure 1 depicts the association between trauma exposure and change in EF, whereas Figure 2 depicts the association between age at first exposure and change in EF. For each figure, the two MIDUS waves are on the horizontal axis and the EF level is on the vertical axis.

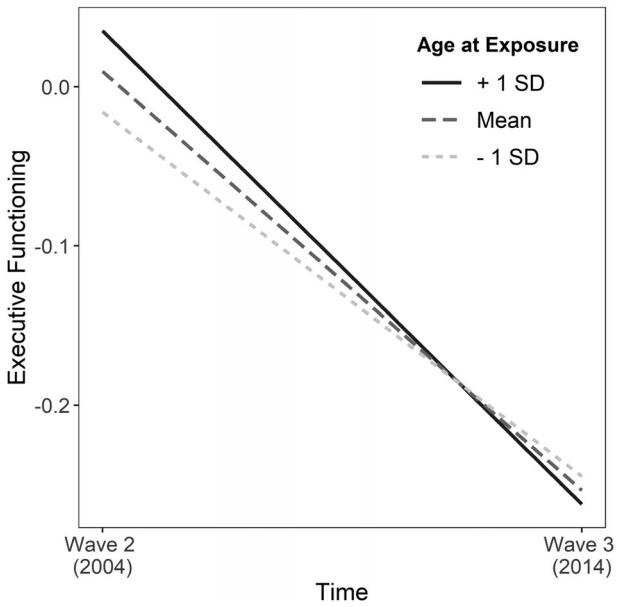

Figure 2. Change in executive functioning as a function of age at first exposure. Executive functioning is from Wave 2 and Wave 3 of the Midlife in the United States (MIDUS); age at first exposure is from MIDUS Wave 2.

Using the computational method proposed by Luke (2004) and Snijders and Bosker (1999), a delta pseudo-R^2 was calculated for each level of Model 2. The delta pseudo R^2 measures the proportion of reduction in explainable variance by the predictors when comparing the final model to a restricted model that contains only covariates. The delta pseudo R^2 values were .39 for Level 2 and .35 for Level 1, indicating that the key predictors in the model explained 39% additional variance in the mean level of EM and 35% additional variance in the intercept, above and beyond the contribution of the covariates.

Episodic Memory

An initial model was estimated with time and covariates as predictors of EM. The results showed a significant decline in EM over the 9 years between MIDUS Wave 2 and Wave 3, $B = -0.11$, $SE = 0.02$, $p < .001$, 95% CI [−0.15, −0.07]. Table 4 shows the results of the three multilevel linear models predicting the level of and change in EM from Wave 2 to Wave 3. The interaction between trauma exposure and age at first exposure was not significant and did not improve the predictive performance of the model, failing to support our third hypothesis. Therefore, the best-fit model was Model 2.

In line with our first hypothesis, trauma exposure significantly predicted change in EM, such that individuals exposed to more trauma had higher levels of EM decline, $B = -0.05$, $SE = 0.02$, $p = .023$, 95% CI [−0.10, −0.01]. However, contrary to our predictions, trauma exposure did not predict the level of EM, $B = 0.00$, $SE = 0.02$, $p = .997$, 95% CI [−0.04, 0.04]. Additionally, our second hypothesis was not supported, as age at first exposure did not predict either level of EM, $B = 0.00$, $SE = 0.00$, $p = .275$, 95% CI [−0.00, 0.00]; or change in EM, $B = -0.00$, $SE = 0.00$, $p = .105$, 95% CI [−0.00, −0.00]. Figure 3 depicts the association between trauma exposure and change in EM, with the two MIDUS waves on the horizontal axis and EM level on the vertical axis.

Using the same computational method as with the model for EF, a delta pseudo R^2 was calculated for each level of Model 2. The delta pseudo R^2 values were .23 for Level 2 and .17 for Level 1, indicating that the key predictors in the model explained 23% additional variance in the mean level of EM and 17% additional variance in the intercept, above and beyond the contribution of the covariates.

Discussion

Using a national sample of middle-aged and older adults, the present study examined the association between lifetime trauma exposure and cognitive functioning while also exploring the effect of age at first exposure. These relations were explored for individual differences in the level of and 9-year change in EF and EM. Individuals with more trauma exposure demonstrated significantly higher levels of decline in both EF and EM over

Table 4
Multilevel Models Predicting Level of and Change in Episodic Memory in the Midlife in the United States Study (MIDUS) From 2004 to 2014

	Model 1		Model 2		Model 3	
Variable	*B*	*SE*	*B*	*SE*	*B*	*SE*
Intercept	0.04*	0.02	0.06**	0.02	0.05*	0.02
Time	−0.12***	0.02	−0.13***	0.02	−0.14***	0.02
Age	−0.03***	0.00	−0.03***	0.00	−0.03***	0.00
Income	0.00	0.00	0.00	0.00	0.00	0.00
Mental health	0.09***	0.02	0.09***	0.02	0.09***	0.02
Physical health	0.00	0.02	−0.00	0.02	−0.00	0.02
Education	0.04***	0.01	0.04***	0.01	0.04***	0.01
Trauma	0.01	0.01	0.00	0.02	−0.01	0.02
Age at Exposure			0.00	0.00	0.00	0.00
Trauma × Age at Exposure[a]					−0.00	0.00
Time × Trauma	−0.05**	0.02	−0.05*	0.02	−0.07**	0.03
Time × Age at Exposure[a]			−0.00	0.00	−0.00	0.00
Time × Trauma × Age at Exposure[a]					−0.00	0.00
Model fit	BIC = 12,158.27		BIC = 10,171.61		BIC = 10,183.21	

Note. BIC = Bayesian information criterion.
[a]Age at first trauma exposure.
*$p < .05$. **$p < .01$. ***$p < .001$.

the 9 years between MIDUS Wave 2 and Wave 3. Furthermore, age at first exposure was associated with more EF decline, in that individuals whose first traumatic experience occurred later in life had more cognitive decline than those whose first traumatic experience occurred earlier in life. These results provide evidence for an association between lifetime trauma exposure and cognitive decline in adulthood, while also highlighting the elevated risk associated with adulthood trauma exposure.

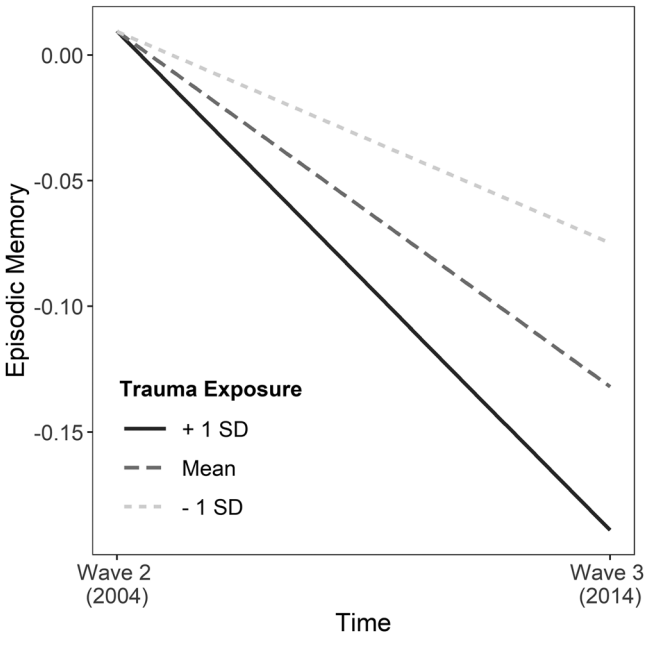

Figure 3. Change in episodic memory as a function of trauma exposure. Episodic memory is from Wave 2 and Wave 3 of the Midlife in the United States (MIDUS); trauma exposure is from MIDUS Wave 2.

The present study adds to a developing body of research examining the lasting effects of trauma exposure on health and cognitive aging. Much of the existing research on the relation between trauma and adulthood cognition focuses on either the long-term effects of childhood trauma exposure (Gould et al., 2012; Majer et al., 2010) or the concurrent cognitive impairments associated with adulthood trauma exposure and PTSD (Brewin, 2014; Honzel et al., 2014; Polak et al., 2012). The present study employed a broader approach by examining the long-term effects of lifetime trauma exposure on adulthood cognition while assessing this association using a longitudinal perspective. This longitudinal perspective was ultimately critical for the identification of the significant effects found in the present study, as the effects of trauma exposure and age at first exposure were found exclusively for change in cognition.

Trauma exposure was associated with an increased decline in both measures of cognitive functioning; however, we did not find associations between trauma exposure and level of functioning for either measure. Whereas the level of functioning focuses on individual differences in cognitive performance at a given time, change in functioning indicates the extent to which an individual is declining in cognition. We demonstrate here that the amount of trauma to which an individual has been exposed is associated with the amount of decline in cognition they experience over a 9-year period of adulthood. Individuals

with more trauma exposure demonstrated more decline associated with cognitive aging, indicating that trauma exposure has a lasting negative effect on cognitive functioning in adulthood. It is possible that trauma exposure also relates to level of functioning in adulthood, with individuals who have been exposed to more trauma demonstrating lower levels of functioning. However, we did not find evidence to support this. Because MIDUS did not include information about participants' childhood cognition, the analyses in the present study were unable to account for individual differences in early-life cognition. This lack of early cognitive data may have reduced the chances of finding effects of trauma exposure on level of functioning. Moreover, limitations of the cognitive measures used and/or the wide age range of participants may have contributed to the small effects. Although the long-term effects may be quite small and difficult to distinguish in midlife, they may become more pronounced as individuals age, as reflected in the current finding that trauma exposure is associated with increased cognitive decline.

The present study found a significant association between age at first exposure and change in EF in adulthood. Individuals whose first traumatic event occurred later in life had more cognitive decline in adulthood than those whose first event occurred earlier in life. This result aligns with what was reported by Krause et al. (2004), who found that traumatic events that occurred in young and middle adulthood had a larger negative impact on health in later life compared to traumatic events that occurred in childhood. The present study supports the existence of a recency effect such that more recent traumatic exposures have a larger effect on health and mental health than traumatic experiences that occurred earlier in life. This contrasts with the perspectives of McCutcheon et al. (2010) as well as Straussner and Calnan (2014), who identified childhood as a unique period of development associated with increased susceptibility to risk. It is notable, however, that the significant effect of age at first exposure was exclusive to EF. More work is needed to understand why the age at which an individual experiences their first traumatic event affects the rate of adulthood decline in EF but not EM.

Overall, our findings suggest that cumulative lifetime trauma exposure results in increased cognitive decline in adulthood. This finding converges with previous literature that has examined trauma and cognition in individuals with PTSD. Specifically, individuals with PTSD have been found to perform worse than individuals without trauma exposure on tasks related to EF (Polak et al., 2012), EM (Brewin, 2014), and working memory (Honzel et al., 2014). The present study extends these findings into the domain of cognitive aging, demonstrating that trauma exposure is associated with an increased decline in performance on tasks related to EF and EM. Moreover, the present study examined trauma exposure in a representative sample of adults with varying levels of trauma exposure rather than in a clinical population of individuals with PTSD.

The present study did not examine mechanisms that underlie the association between trauma and cognition. However, trauma exposure has previously been associated with poor health and preclinical biomarkers, such as metabolic syndrome and elevated inflammation (e.g., Hostinar, Lachman, Mroczek, Seeman, & Miller, 2015; Krause et al., 2004; Slopen et al., 2010), whereas poor cardiovascular health and elevated inflammation have, in turn, been linked with cognitive decline (e.g., Bourassa & Sbarra, 2017; Karlamanga et al., 2014; Sartori, Vance, Slater, & Crowe, 2012). This suggests that trauma exposure and cognition may be linked via elevated inflammation, although other possible mechanisms exist. For instance, childhood trauma has been found to increase the risk of metabolic syndrome, a precursor to diabetes that is tied to cognitive aging (Lee et al., 2014). Trauma exposure has also been found to lead to increased depressive symptoms (e.g., Fowler, Allen, Oldham, & Frueh, 2013; Wingo et al., 2010), and the presence of depressive symptoms has been shown to increase the risk of cognitive impairment and lead to accelerated cognitive decline (Köhler et al., 2010). Although the MIDUS study includes a subset of participants for whom biomarker data were collected, only a small subsample has data on both biomarkers and cognition. As MIDUS continues collecting data in future study waves, it will be more feasible to directly examine the relations among trauma exposure, inflammation, and cognitive functioning and evaluate elevated inflammation as a possible mediator.

There are notable limitations to the present study. The MIDUS sample was drawn as a representative sample, but individuals who ultimately participated were skewed in terms of educational attainment and racial diversity. The analyses conducted in the study were also limited in that the multilevel modeling approach is best suited for data with multiple assessments. Although cognitive functioning data are available from two MIDUS assessment points, data from future follow-ups can be added to the models developed for this study, thus allowing the associations identified herein to be examined more precisely and over a longer span of time. Furthermore, it is difficult to draw strong conclusions based on cognitive outcomes in a sample with a broad age range. Age was included as a covariate in our analysis, but changes in cognition may involve different pathways for individuals of different ages. Finally, it is important to note that the effects reported in the present study are relatively small. Despite this, the delta pseudo-R^2 values for each model indicate that a fair amount of additional variance in the outcome measure was explained by the variables of interest (i.e., 17%–39%).

In conclusion, most individuals will be exposed to at least one potentially traumatic experience in their lifetime, and exposure to trauma has been found to result in negative health and mental health outcomes (e.g., Elliott et al., 2018; Krause et al., 2004). The results of the present study demonstrate that lifetime trauma exposure is associated with significantly increased cognitive decline in adulthood. Furthermore, trauma exposure that occurs later in life was shown to be associated with more cognitive decline than trauma exposure that occurred earlier in life. This study has laid the groundwork for future studies to continue exploring these associations, using a multilevel modeling

approach with data from additional assessment points. Furthermore, although the mechanisms were not examined here, future studies should strive to directly evaluate possible mediators underlying the association between trauma exposure and cognitive functioning, including elevated inflammation or other cardiovascular disease risk factors, such as metabolic syndrome.

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