The Moderating Effects of Perceived Control on Relationships Between Trauma Exposure in Childhood and Cognitive Health in Adulthood

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Abstract

Objectives: Exposures to potentially traumatic events (PTE) in childhood have been linked to changes in cognitive health in later life. Perceived control is an important psychosocial resource that may serve as a potential buffer against these deleterious effects. We hypothesized that the detrimental impact of childhood PTE exposure on cognitive health would be attenuated among those with higher perceived control.

Methods: Adults ranging from 28 to 84 years old at baseline (N = 4,512) from the Midlife in the United States study (M_{age} = 55.4, 54.9% female, 82.4% white) completed surveys on potentially traumatic childhood events, perceived control, and a telephone-based battery of tests measuring executive function (EF) and episodic memory (EM) in ~2005 and ten years later in ~2015.

Results: Findings from multilevel models indicate that perceived control buffered the detrimental impacts of childhood PTE on cognitive health. More childhood PTE exposure was associated with a steeper rate of decline in EF among people with lower control (-1SD; b = -0.35, SE = 0.02, p < .001) compared with people with higher control (+1SD; b = -0.31, SE = 0.02, p < .001). In exploratory analyses, higher levels of emotional neglect were associated with worse EF only among those with lower perceived control (-1SD; b = -0.01, SE = 0.01, p =.029). Perceived control did not moderate the effects of childhood PTE on EM (ps > .05).

Discussion: A strengths-based approach is relevant for understanding the lifetime impacts of early life adversities on later life cognitive health outcomes.

Keywords: Potentially traumatic events, childhood trauma, executive function, episodic memory, perceived control

Approximately 60% of children are exposed to at least one potentially traumatic event (PTE; e.g., loss of a loved one, experiencing a natural disaster, emotional abuse, emotional neglect) by the age of 16, and around half (30%) are exposed to multiple events (McLaughlin et al., 2013). PTE exposure during childhood may lead to negative health outcomes, impacting an individual's developmental trajectory in different domains and preventing them from experiencing better health outcomes as they grow older (Waldinger et al., 2006).

In the cognitive domain, although decreasing executive function (EF) and episodic memory (EM) are typical with aging, there is variability in the rate of change and the areas of cognition that undergo change (e.g., Baltes et al., 2006; Bopp & Verhaeghen, 2005; Schaie et al., 2004). Individual differences in lived experiences, psychosocial factors, and social determinants of health, including PTE and perceived control, have the potential to influence cognitive health and aging. More lifetime PTE exposure has been associated with faster cognitive decline in both executive function (EF; planning, focusing attention, and working memory) and episodic memory (EM; long-term recall that involves the recollection of prior experiences; Lynch & Lachman, 2020). Although trauma exposure is a risk factor for cognitive decline in adulthood, it is unclear what factors may protect against the deleterious effects of childhood trauma. We examined perceived control, or the extent to which one believes that one's actions can evoke desirable outcomes (Lachman, 2006), as a potential protective factor given its role as a psychosocial resource for cognitive health (Caplan & Schooler, 2003; Cerino et al., 2020; Neupert & Allaire, 2012).

Effects of Trauma on Cognitive Health

A PTE is defined as potentially threatening or physically and/or emotionally harmful events that result in lasting adverse effects on one's level of well-being or functional impairment

(Pai et al., 2017; Lynch & Lachman, 2020). A qualitative review of clinical interviews from 122 active duty service members identified six different categories of PTE: life threat to self, life threat to others, aftermath of violence, traumatic loss, moral injury by self, and moral injury by others (Stein et al., 2012). To these can be added betrayal trauma, which is defined as a violation of one's feelings of safety and security by a trusted person or institution on whom one depends for survival (Freyd, 1994; 2001). This highlights the importance of assessing a range of PTEs to allow for a multifaceted approach to post-traumatic trajectories and phenomenologies.

In a longitudinal study of cognitively unimpaired adults ranging from 35-85 years old, more PTE exposures at any age were associated with faster decline in both EF and EM across a 10-year follow-up. Additionally, age at first PTE exposure was shown to predict change in EF such that individuals who were first exposed to PTE later in life had faster decline over 10 years than those whose first PTE exposure occurred earlier in life (Lynch & Lachman, 2020). Similarly, among a cross-sectional sample of cognitively unimpaired older adults, individuals with more trauma exposures exhibited worse performance in areas of cognition such as processing speed and executive functioning (Petkus et al., 2018). These examples demonstrate a need to examine the influence of exposure to PTEs on cognitive function across the lifespan.

Perceived Control and Trauma

Perceived control refers to the extent to which one believes that one's actions can evoke desirable outcomes, comprising two related beliefs: perceived ability to perform actions required to achieve goals, known as general mastery, and perceptions of whether certain outcomes are beyond one's control due to powerful others or by chance, known as perceived constraints (e.g., Lachman, 2006; Skinner, 1996). Perceived control is considered to be a dynamic process that is susceptible to change in response to factors such as lived experiences, interactions with others,

and age-related changes (Lachman et al., 2011). Consistent with conceptual and empirical accounts of control (e.g., Krause & Shaw, 2003; Lachman et al., 2009; Lachman & Firth, 2004; Mirowsky, 1995), past work tends to find levels of control beliefs to be lower among older adults, with longitudinal evidence of declines in perceived control across younger adults and older adults but relative stability for people in midlife (Cerino et al., 2023).

More trauma exposures have been associated with increased mortality risk for individuals with lower levels of personal mastery (Elliot et al., 2018). The association between trauma and mortality risk, however, was attenuated among people with higher mastery. Relatedly, higher levels of control have been shown to buffer against the detrimental effects of abuse on physical health and mental health (Pitzer & Fingerman, 2010). In a cross-sectional sample, experiencing abuse was related to worse physical health and higher negative affect, especially among people with lower levels of control. People who experienced abuse and had high control had similar levels of physical health and negative affect as people who had not experienced abuse (Pitzer & Fingerman, 2010). Perceived control may act as a psychosocial protective factor for health outcomes among individuals who have experienced adversity in childhood.

Perceived Control and Cognitive Health

Perceived control has been linked to cognitive health within persons over time and at the level of individual differences. Neupert and Allaire (2012) showed that on days when perceived competence was higher and locus of control was more internal, inductive reasoning performance and memory performance were higher (Neupert & Allaire, 2012). Perceived competence-inductive reasoning associations were strongest among those with low levels of perceived competence, while locus of control-memory associations were strongest among those with more internal levels of locus of control (Neupert & Allaire, 2012). In a micro-longitudinal study by

Cerino and colleagues (2020), cognitive health was better (i.e., less response-time inconsistency) on days when perceived control was higher than usual; however, these daily associations depended on age and the amount of attentional demands of the cognitive task. Specifically, reporting higher than usual perceived control was associated with worse cognitive health in an attentionally demanding task for relatively older participants. While these findings reveal some protective effects of control beliefs, they also suggest that perceived control may have different influences on individuals' cognitive health depending on their age and potential cognitive resources.

Higher perceived control is associated with a lower likelihood of cognitive decline in middle-aged and older adults (Caplan & Schooler, 2003). Specifically, older adults with higher levels of self-confidence and lower levels of fatalism (believing that life events occur due to uncontrollable external events) were less likely to experience difficulties completing cognitively demanding tasks 20 years later. Given the capacity of perceived control to serve as a psychosocial protective factor in the domain of cognitive health, it is important to determine whether it similarly operates as a psychosocial resource among individuals who have experienced adversity in childhood.

The Present Study

This study seeks to examine the extent to which perceived control moderates the relationship between childhood PTE exposure and cognitive health in adulthood. The following research questions were addressed: First, to what extent does perceived control moderate the relationship between childhood PTE exposure and cognitive function (EF, EM)? Second, to what extent does perceived control moderate the relationship between childhood PTE exposure and rate of change in cognitive function (EF, EM)? We hypothesized that:

(1) Childhood PTE exposure will be associated with lower levels of cognitive function and steeper rates of cognitive decline in later life.

(2) The detrimental effects of childhood PTE exposure on the level of cognitive function and change in cognitive function will be attenuated among those with higher perceived control.

Emotional abuse is reportedly one of the most commonly experienced adverse childhood experiences, and approximately 18% of adults retrospectively reported experiencing emotional neglect in childhood (Giano et al., 2020; Kumari, 2020). Exploratory analyses therefore examined specific effects of emotional abuse and emotional neglect.

Method

Transparency and Openness

The current study used data from the Midlife in the United States (MIDUS) study. Data are publicly available at the following website: (https://www.icpsr.umich.edu/web/ICPSR/series/203). Research questions, hypotheses, and analytic plans were pre-registered with the Open Science Framework and are publicly available at the following link: (https://osf.io/4uqb7). MIDUS was approved by the Institutional Review Board.

Participants

The analytic sample consisted of up to 4,504 (M_{age} = 55.4, *SD* = 12.3, *Range* = 28-84) participants who completed cognitive testing at Wave 2 (~2005). Of these participants, 3,291 respondents completed cognitive testing 10 years later in Wave 3 (~2015). The large sample size was likely to enhance statistical power, thereby reducing the likelihood of Type II errors. The sample was primarily female (54.9%), white (82.4%), and non-Hispanic (97.1%). The majority

of participants reported that they had completed at least some college (66.0%), and slightly less than half of the sample (45.0%) reported having experienced at least one PTE exposure before the age of 18. Two participants reported having lost a child when they were 4 years old, and another three participants reported losing a child when they were 5 years old; given that it is unlikely that these participants had children themselves at such a young age, these responses were excluded from analyses.

Method

MIDUS is a national longitudinal study that seeks to evaluate health and well-being across the adult lifespan (Ryff et al., 2014). Participants were recruited via random digit dialing. Currently, there are three waves of MIDUS data publicly available: Wave 1 collected data from a total of 7,108 participants in 1995 between the ages of 25 and 75 years, and Waves 2 and 3 followed up on retained participants every ten years in ~2005 and ~2015, respectively. As Wave 1 did not collect cognitive data, only Waves 2 and 3 were used in this study.

Measures

PTE Exposure

PTE exposure was assessed in Wave 2 of MIDUS using an abbreviated version of the Life Experiences Survey (LES; Sarason et al., 1978). The original LES demonstrated adequate test–retest reliability (total change score $\alpha = .64$; Sarason et al., 1978). Participants were asked to indicate which events they had experienced and at what age the event occurred. The 12 events used in MIDUS were previously identified as most traumatic (Elliot et al., 2018; Lynch & Lachman, 2020). PTE exposure was calculated as the total number of events experienced (0-12). Any events that occurred after the age of 18 were excluded. To measure frequencies of adulthood

PTE exposure, we restricted the LES events to only those that occurred at ages 18 and older. The 12 LES items were then summed to create an adulthood PTE sum variable.

Cognitive Functioning

The primary outcome variables in this study, episodic memory (EM) and executive function (EF), were both measured at Waves 2 and 3 using The Brief Test of Adult Cognition by Telephone (BTACT; Tun & Lachman, 2006). In the BTACT protocol, seven cognitive tests were administered after completing a brief hearing test as an inclusion criterion (Lachman et al., 2014). A composite z-score was derived for EM using performance on immediate recall and delayed recall tasks using word list learning. A composite z-score was derived for EF from performance on the following tasks: category fluency, working memory, inductive reasoning, processing speed, and attention switching. Composite scores were created by averaging participants' z-scores on each subtest, then re-standardizing the averages to create a composite z-score for each cognitive domain (DiBlasio et al., 2021). Higher composite scores indicate better EM and EF (Lachman et al., 2014). This test battery was designed for valid assessment of cognitive functioning in large community-based samples and is sensitive to differences in performance due to cognitive aging across the lifespan (Lachman et al., 2014; Lachman & Tun, 2008; Tun & Lachman, 2006; Stawski et al., 2010).

Perceived Control

Perceived control at Wave 2 was assessed using 12 items from the Midlife Developmental Inventory (Ryff et al., 2014). Participants indicated how much they agreed or disagreed with 12 statements on a 7-point Likert-type scale (1 = strongly disagree, 7 = strongly agree). Higher mean scores on a perceived control composite of mastery and constraints

(recoded) indicated higher perceived control. Internal consistency in the present sample was good (α = .86).

Covariates

Education, physical health, adulthood PTE exposures, genetic risk of PTSD, gender, race, and chronological age were included as covariates in substantive analyses given past research linking individual differences in these factors with primary study variables (e.g., Alegría et al., 2013; Frewen et al., 2019; Lachman & Firth, 2004; Lövdén et al., 2020). MIDUS measured educational attainment using a self-report of the highest level of education attained. Self-rated physical health was operationalized using one item that evaluated participants' perceptions of their physical health by asking, "In general, would you say your physical health is excellent, very good, good, fair, or poor?" (Ryff et al., 2014). Consistent with prior research, adulthood trauma — the sum of LES items indicated to have occurred at or after the age of 18 — was included as a covariate in analyses addressing primary research questions (Lynch & Lachman, 2020). As PTSD has known effects on cognitive function, polygenic risk scores providing information on genetic predisposition to PTSD were also included as a covariate. MIDUS obtained DNA tissue samples (blood and saliva) from a subsample of participants (N = 1,255) who participated in a biomarker survey in Wave 2 in order to calculate polygenic risk scores using PRSice-2 (Choi & O'Reilly, 2019). Higher values indicate a higher risk of PTSD. To account for differences in PTE exposures and risk of PTSD between individuals from different racial and ethnic groups, participants' race was included as a covariate. Similarly, given sex differences in cognitive decline (Levine et al., 2021) and gender differences in levels of perceived control (Lachman & Firth, 2004), gender (0 = men, 1 = women) was also included as a covariate.

Data Preparation

Alpha was set at .05. Baseline age, perceived control, and health were mean-centered prior to being entered into the models. We collapsed Black/African American, Native American or Alaska Native, Asian or Pacific Islander, and "other" into a dichotomous variable (0 = white, 1 = people of color) due to low cell sizes of individual racial categories. Gender was coded with men as the reference category (0 = men, 1 = women). Education was coded as a dichotomous variable (0 = high school or less, 1 = some college or more). Health was reverse-scored so that higher values indicated better self-rated health.

Analytic Plan

SAS 9.4 (SAS Institute, 2013) was used for quantitative data analyses. Univariate descriptive analyses for study variables characterized the analytic sample prior to conducting further analyses. Bivariate correlations among study variables described the interrelationships among study variables.

Given the repeated measurement structure of the MIDUS data (2 waves of assessment nested within up to 4,512 participants), we used two-level multilevel models (PROC MIXED; SAS Institute, 2013) to address our research questions. Initial unconditional/empty multilevel models for EF and EM evaluated the proportion of variation at each level of analysis (wave-level, person-level) with maximum likelihood estimation due to missing data and attrition across waves. Next, analyses were performed to address primary research questions.

Consistent with prior research using this data (e.g., Lynch & Lachman, 2020), we used multilevel models to examine links among PTE and perceived control with both the level and change in cognitive function. Wave-level (Level 1) cognitive health was regressed on a wave-level intercept and a linear trend for wave, as well as person-level (Level-2) trauma exposure, perceived control, age, gender, education, race, adulthood PTE, health, and PTSD risk. Separate

multilevel models were run with EF as the outcome, followed by EM as the outcome. Covariates were included in MLMs in a hierarchical fashion, with Model 1 only including primary main effects. Model 2 added interactions. Model 3 added sociodemographic covariates (age, gender, race, and education). Model 4 added structural covariates (adulthood PTE exposure, health, and PTSD risk) to determine whether primary interactions of interest were robust to the inclusion of sociodemographic and structural factors, respectively.

The full equation used to test the two research questions for person i and time j is described below.

$$\underline{\text{Level 1 (wave):}}\ \text{CognitiveHealth}_{ji} = \beta_{0i} + \beta_{1i}(Wave_{ji}) + e_{ji}$$

$$\underline{\text{Level 2 (person):}}\ \beta_{0i} = \gamma_{00} + \gamma_{01}(\text{ChildPTE}_i) + \gamma_{02}(\text{Control}_i) + \gamma_{03}(\text{Age}_i) + \gamma_{04}(\text{Gender}_i) + \gamma_{05}(\text{Race}_i) + \gamma_{06}(\text{Education}_i) + \gamma_{07}(\text{PhysicalHealth}_i) + \gamma_{08}(\text{PTSDRisk}_i) + \gamma_{09}(\text{AdultPTE}_i) + \gamma_{10}(\text{ChildPTE}_i * \text{Control}_i) + u_{0i}$$

$$\beta_{1i} = \gamma_{11} + \gamma_{12}(\text{ChildPTE}_i) + \gamma_{13}(\text{Control}_i) + \gamma_{14}(\text{ChildPTE}_i * \text{Control}_i) + u_{1i}$$

To address the first research question on the moderation of relationships between childhood PTE and level of cognitive health by control, we included a PTE exposure by control interaction term such that an individual's level of cognitive health is a function of the interaction between childhood PTE exposures and perceived control (parameter γ_{10} in the equation above). To address the second research question on potential control moderation of relationships between trauma exposure and change in cognitive health, we included a three-way interaction term (i.e., wave by childhood PTE exposure by control) such that an individual's rate of change in

cognitive health was a function of the interaction between childhood PTE exposure and perceived control (parameter γ_{14} in equation above).

Results

Descriptive Analyses

Supplementary Table 1 provides complete descriptive statistics for primary study variables. Supplementary Table 2 provides item frequencies for the LES. Supplementary Table 3 provides the zero-order correlations among model variables. Participants reported less than one PTE on average in childhood (M = 0.61, SD = 0.82, $Range$ = 0-5) and slightly more than one in adulthood (M = 1.29, SD = 0.95, $Range$ = 0-5). More PTE exposure in childhood was associated with better EM at Wave 2 ($r(3002) = 0.07$, $p < .001$), but not at Wave 3 ($r(2245) = 0.02$, $p = .271$). More PTE exposure in childhood was not significantly associated with EF at Wave 2 ($r(3008) = 0.01$, $p = .426$) or Wave 3 ($r(2245) = -0.03$, $p = .134$). Higher perceived control was associated with better Wave 2 EF ($r(3872) = 0.18$, $p < 0.001$) and EM ($r(3863) = 0.11$, $p < 0.001$), with similar, though weaker, associations present at Wave 3. There was no significant association between childhood PTE exposure and perceived control.

In unconditional models, 69% of the variance in EF (54% in EM) reflected between-person differences, and the remaining 31% (46% in EM) reflected within-person variation across waves, unspecified sources of variation, and error.

Research Question 1

Level of Executive Function

Childhood PTE exposures were not related to the level of EF (b = 0.01, SE = .01, $p > .05$). However, higher perceived control was related to better EF such that a one unit increase in perceived control was associated with 0.17 units higher EF performance (b = 0.17, SE = 0.02,

$p < .001$). Perceived control did not significantly moderate the relationship between childhood PTE exposures and level of EF ($b = -0.003$, $SE = 0.02$, $p > .05$). Adjusting for sociodemographic variables and structural variables did not substantively change the results. For full model results, see Table 1.

Level of Episodic Memory

Childhood PTE exposures were significantly associated with better EM such that each additional PTE exposure was associated with 0.07 units higher EM performance ($b = 0.07$, $SE = .02$, $p < .001$). Additionally, a one unit increase in perceived control was associated with 0.11 units higher EM performance ($b = 0.11$, $SE = .02$, $p < .001$). Perceived control did not significantly moderate the relationship between childhood PTE exposures and level of EM ($b = 0.01$, $SE = 0.02$, $p > .05$). Adjusting for sociodemographic variables and structural variables did not substantively change the results. For full model results, see Table 2.

Research Question 2

Modeling Change in EF and EM Across Waves

Change in cognitive function was modeled with wave of assessment. On average, EF declined by 0.36 units ($b = -0.36$, $SE = .03$, $p < .001$) and EM declined by 0.11 units ($b = -0.11$, $SE = .03$, $p < .001$) across the 10-year period.

Change in Executive Function

Perceived control significantly moderated the relationship between childhood PTE exposures and change in EF ($b = 0.03$, $SE = 0.01$, $p = .023$; Table 3). Figure 1 and Supplementary Figure 1 illustrate simple slopes at -1SD, mean, and +1SD control on a line graph communicating a decline in cognitive function (Figure 1) and bars with corresponding standard errors (Supplementary Figure 1). Each additional childhood PTE exposure was associated with a

0.35 unit decline in EF for participants with low perceived control (-1SD; b = -0.35, SE = 0.02, p < .001) compared to a 0.31 unit decline for participants with high perceived control (+1SD; b = -0.31, SE = 0.02, p < .001). Adjusting for sociodemographic variables did not substantively change the result. The interaction effect was larger but no longer statistically significant after adding structural covariates (b = 0.05, SE = 0.03, p = .143). The size of the three-way interaction term was robust to adjustment for health and PTSD risk, but was smaller after adding adulthood PTE exposures (b = 0.02, SE = 0.02, p = .160). Thus, adulthood PTE exposures may partially explain some of the interaction.

Change in Episodic Memory

Perceived control did not significantly moderate the relationship between childhood PTE exposures and the rate of change in EM in the unadjusted model (b = -0.02, SE = 0.02, p = .241). This pattern of results was consistent when adjusting for sociodemographic and structural variables. For full model results, see Table 4.

Sensitivity Analyses

We conducted sensitivity analyses that restricted analyses to participants who contributed to both waves of assessment (N = 1,989 participants for EF models, N = 1,983 for EM models). After restricting the sample, the pattern, magnitude, and statistical significance of the results remained unchanged. We conducted additional analyses that removed three outlying EF z-scores (z = 12.85; z = -5.63; z = -6.93) to assess whether the findings were a byproduct of outliers. The pattern, magnitude, and significance of the results remained unchanged. As such, the findings appeared to be robust to the influence of outliers.

Exploratory Analyses

Exploratory analyses were conducted using a subsample of participants who completed the biomarker survey at Wave 2 and completed the Childhood Trauma Questionnaire (Bernstein & Fink, 1998), following the same analytic plan as the analyses addressing primary research questions. Compared to participants from the primary sample, subsample participants (N = 1,203) were significantly more likely to be female, younger, and more educated. They also reported significantly better physical health, more childhood PTE, higher control, and had better EF and EM.

Each multilevel model was re-run by first replacing childhood PTE with emotional abuse, then by replacing childhood PTE with emotional neglect. Perceived control significantly moderated the relationship between emotional neglect and level of EF (b = 0.01, SE = 0.005, p = .022) such that more emotional neglect was only significantly related to worse EF among people with low control (-1SD; b = -0.01, SE = 0.01, p = .029). Higher levels of control buffered the detrimental impact of emotional neglect on EF. These findings were robust to the effects of sociodemographic and structural covariates. Additionally, there was a significant main effect of emotional abuse on levels of EM such that individuals with more emotional abuse exposures had higher levels of EM performance (b = 0.01, SE = 0.01, p = .031). For full model results, see Supplementary Tables 4 and 5.

Discussion

The present study found that the negative relationship between childhood PTE exposures and EF was moderated by perceived control such that the rate of decline in EF was steeper for people with lower levels of perceived control compared to people with higher levels of perceived control; however, when adulthood PTE exposures and polygenic risk scores were added into the model, the effect was no longer statistically significant. Indeed, the moderating effect was small

(b = 0.03), though this reflects the effect of each additional childhood PTE exposure. Perceived control did not moderate the effects of childhood PTE on EM outcomes, though higher levels of childhood PTE and perceived control were related to better EM performance. Further, in exploratory analyses, higher levels of emotional neglect were associated with worse EF performance only among those with lower perceived control. More emotional neglect was not related to worse EF performance among people with comparatively higher levels of control. More frequent emotional abuse was also associated with higher levels of EM.

As a complement to previous work using MIDUS data that examined relationships between lifetime PTE exposures and cognitive health, the present study focused on the specific effects of childhood PTE exposures. Lynch and Lachman (2020) found that lifetime PTE was negatively associated with the rate of change in EF, whereas the present study found that the rate of change in EF was steeper for people with lower levels of perceived control compared to people with higher levels of perceived control. Furthermore, while Lynch and Lachman (2020) did not find a relationship between lifetime PTE exposure and level of cognition, our exploratory analyses revealed a negative association between childhood emotional neglect and EF for people with low perceived control. Additionally, perceived control buffered the negative effects of childhood PTE and emotional neglect on EF performance, providing evidence that perceived control may protect against the harmful effects of childhood PTE in the domain of cognitive health in addition to physical and psychological health domains (Pitzer & Fingerman, 2010).

A growing body of literature suggests that early life exposures to PTE and similar adversities are related to alterations in the development of neural pathways and connectivity (e.g., Cassiers et al., 2018; Thomason & Marusak, 2017). A systematic review found that different subtypes of childhood trauma – such as emotional maltreatment and sexual abuse – had

differential associations with structural and functional brain activity across domains of cognitive function (Cassiers et al., 2018). Further support for this trend may come from recent work, which argues that not all childhood PTEs are equal in their influence on developmental outcomes across health domains (e.g., Barger & Oláis, 2024). Together, these findings suggest that our results may be the product of long-term differential effects of childhood PTE exposure on cognitive function. For instance, there may be neural pathways associated with perceived control that bolster EF performance in individuals with higher levels of PTE exposure. Conversely, childhood PTE exposure may strengthen pathways in areas of the brain associated with EM, such as the temporal lobe (e.g., Cook et al., 2009). Further research is needed to understand how specific PTE exposures differentially influence the development and maintenance of neural pathways and connectivity across the lifespan.

Limitations and Future Directions

The strengths of this study must be interpreted alongside its limitations. While the MIDUS study provides a large national study with a wide age range across the adult lifespan, one limitation lies in the relative homogeneity of the sample's sociodemographic composition. Participants were cognitively unimpaired, community-dwelling, and predominantly white, well-educated, and healthy. As such, future studies could replicate and extend the present study by including a more diverse sample across cognitive and sociodemographic statuses and by including a measure of childhood PTE or adversity that includes exposures to racism, prejudice, or oppression. Similarly, the BTACT was designed to detect age-related changes in cognitive health but not to detect cognitive impairment or screen for cognitive status. Thus, it is unknown whether these results would generalize to individuals with cognitive impairment (e.g., mild cognitive impairment, dementia). Future studies would benefit from the inclusion of individuals

with different cognitive health statuses to determine whether the moderating effects of perceived control are generalizable to individuals with cognitive impairment. This is especially relevant for people who have developed PTSD following exposure to PTE, as they are at higher risk of experiencing deficits in EF and EM and susceptible to increased dementia risk (Radford et al., 2017).

An additional limitation of this study lies in the measures used to operationalize childhood PTE exposures and PTSD risk. The LES was intended to capture lifetime exposures to PTE, meaning that it did not capture several adversities that may be unique to childhood such as emotional abuse and neglect. Additionally, the LES did not include a measure of the chronicity of each event, meaning that the extent to which the protective effects of perceived control are robust to the influence of repeated PTE exposures are unknown. While we attempted to address these limitations through exploratory analyses with measures assessing exposures to emotional abuse and emotional neglect, future studies would benefit from using a measure that is validated to assess childhood exposures to PTE that also includes a measure of chronicity. Furthermore, future studies may benefit from the inclusion of individuals with a lifetime diagnosis of PTSD to more accurately assess the influence of PTSD on the relationship between exposures to PTE and cognition given the known relationships among PTE, PTSD, and cognitive function (e.g., Golier et al., 2006; Qureshi et al., 2011; Petzold & Bunzeck, 2022). Lastly, the moderation analyses in this study identified *for whom* the relationships between childhood PTE and cognitive aging outcomes were most salient. They did not, however, explain *why* these associations emerged. Future research should address this with mediation analyses that examine the mechanisms underlying the relationships between more childhood PTE and steeper rates of decline in EF performance for people with low control.

Conclusion

It is crucial to understand how early life experiences shape developmental trajectories across the lifespan. By understanding the extent to which trauma exposure influences both levels of and rates of change in cognitive health, we may be able to better inform our understanding of how the harmful impacts of childhood trauma might persist across the lifespan. This study characterized the relationships between childhood PTE exposures and cognitive health later in life with explicit focus on the protective effects of perceived control within these relationships.

In elucidating the role that perceived control plays within this relationship, we have strengthened our understanding of a critical psychosocial resource that has the capacity to promote cognitive health across the adult lifespan. The results of this study indicate that perceived control may protect against the detrimental influence of childhood PTE exposure on the cognitive domain of EF. These findings are in alignment with current literature, which has identified higher perceived control as a protective factor for cognitive health (e.g., Cerino et al., 2020; Neupert & Allaire, 2012). These results have relevant implications for future research and cognitive health promotion efforts that take a strengths-based approach to understanding the lifetime impacts of early life adversities on later life outcomes.

Funding

Research was supported by the National Institute on Aging, under award numbers: P01 AG020166, M3 U19 AG051426, and the National Institute on Minority Health and Health Disparities under Award Number U54 MD012388.

Conflict of Interest

We have no interest to disclose.

Data Availability

Data are publicly available at the following website: (https://www.icpsr.umich.edu/web/ICPSR/series/203). Research questions, hypotheses, and analysis plan preregistered at https://osf.io/4uqb7

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Table 1. *Research Question 1 – Level of Executive Function*

Variables	Model 1: Unadjusted		Model 2: Interaction		Model 3: Sociodemographic Variable Adjustment		Model 4: Structural Variable Adjustment	
	Estimate (*SE*)	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**
Intercept	-.130 (.019)***	[-0.17, -0.09]	-.011 (.020)	[-0.05, 0.03]	-.084 (.031)**	[-0.14, -0.02]	.085 (.146)	[-0.20, 0.37]
Wave	—	—	-.325 (.013)***	[-0.35, -0.30]	-.329 (.013)***	[-0.35, -0.30]	-.359 (.027)***	[-0.41, -0.31]
Child PTE Sum	.009 (.0190)	[-0.03, 0.05]	.014 (.019)	[-0.02, 0.05]	-.030 (.016)	[-0.06, 0.00]	-.018 (.033)	[-0.08, 0.05]
Control	.163 (.015)***	[0.13, 0.19]	.171 (.019)***	[0.13, 0.21]	.107 (.016)***	[0.08, 0.14]	-.003 (.034)	[-0.07, 0.06]
Age	—	—	—	—	-.030 (.001)***	[-0.03, -0.03]	-.029 (.003)***	[-0.03, -0.02]
Female	—	—	—	—	-.128 (.026)***	[-0.18, -0.08]	-.094 (.054)	[-0.20, 0.01]
College	—	—	—	—	.452 (.028)***	[0.40, 0.51]	.272 (.065)***	[0.14, 0.40]
Race	—	—	—	—	-.613 (.035)***	[-0.68, -0.54]	-.183 (.197)	[-0.57, 0.20]
Health	—	—	—	—	—	—	.105 (.033)**	[0.04, 0.16]
PRS	—	—	—	—	—	—	-.021 (.178)	[-0.37, 0.33]
Adult PTE Sum	—	—	—	—	—	—	.017 (.030)	[-0.08, 0.05]
Child PTE Sum*Control	—	—	.003 (.018)	[-0.04, 0.03]	-.004 (.015)	[-0.03, 0.03]	.025 (.032)	[-0.04, 0.09]

Note. SE = standard error; PTE = potentially traumatic events; PRS = polygenic risk scores. Model 1: N = 3,031, Observations = 5,018, BIC = 11794.4.; Model 2: N = 3,031, Observations = 5,018, BIC = 11278.1; Model 3: N = 3,019, Observations = 5,003, BIC = 10122.5; Model 4: N = 528, Observations = 982, BIC = 1879.0.

*p < .05. **p < .01. *** p <.001.

Table 2. *Research Question 1 – Level of Episodic Memory*

Variables	Model 1: Unadjusted		Model 2: Interaction		Model 3: Sociodemographic Variable Adjustment		Model 4: Structural Variable Adjustment	
	Estimate (*SE*)	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**
Intercept	-.079 (.021)***	[-0.11, -0.04]	-.038 (.022)	[-0.08, 0.01]	-.409 (.035)***	[-0.48, -0.34]	-.108 (.161)	[-0.42, 0.21]
Wave	—	—	-.106 (.021)***	[-0.15, 0.07]	-.128 (.020)***	[-0.17, -0.09]	-.126 (.043)**	[-0.21, -0.04]
Child PTE Sum	.066 (.020)**	[0.03, 0.11]	.068 (.020)***	[0.03, 0.11]	-.001 (.018)	[-0.04, 0.03]	.012 (.036)	[-0.06, 0.08]
Control	.104 (.016)***	[0.07, 0.14]	.100 (.020)***	[0.06, 0.14]	.090 (.018)***	[0.05, 0.13]	-.013 (.038)	[-0.09, 0.06]
Age	—	—	—	—	-.026 (.001)***	[-0.03, -0.02]	-.027 (.003)***	[-0.03, -0.02]
Female	—	—	—	—	.507 (.030)***	[0.45, 0.56]	.618 (.059)***	[0.50, 0.73]
College	—	—	—	—	.299 (.031)***	[0.24, 0.36]	.140 (.078)	[-0.00, 0.28]
Race	—	—	—	—	-.330 (.040)***	[-0.41, -0.25]	.091 (.216)	[-0.33, 0.52]
Health	—	—	—	—	—	—	.118 (.036)**	[0.05, 0.19]
PRS	—	—	—	—	—	—	.361 (.196)	[-0.02, 0.75]
Adult PTE Sum	—	—	—	—	—	—	-.025 (.033)	[-0.09, 0.04]
Child PTE Sum*Control	—	—	.009 (.019)	[-0.03, 0.05]	-.003 (.017)	[-0.04, 0.03]	.049 (.035)	[-0.02, 0.12]

Note. SE = standard error; PTE = potentially traumatic events; PRS = polygenic risk scores. Model 1: N = 3,031, Observations = 5,008, BIC = 13584.9; Model 2: N = 3,031, Observations = 5,008, BIC = 13574.6; Model 3: N = 3,019, Observations = 4,993, BIC = 12752.4; Model 4: N = 528, Observations = 980, BIC = 2402.2.

*p < .05. **p < .01. *** p <.001.

Table 3. *Research Question 2 – Change in Executive Function*

Variables	Model 1: Unadjusted		Model 2: Unadjusted		Model 3: Sociodemographic Variable Adjustment		Model 4: Structural Variable Adjustment	
	Estimate (*SE*)	95% CI	Estimate (*SE*)	95% CI	Estimate (*SE*)	95% CI	Estimate (*SE*)	95% CI
Intercept	-.011 (.020)	[-0.05, 0.03]	-.014 (.021)	[-0.05, 0.03]	-.086 (.031)**	[-0.15, -0.03]	.086 (.146)	[-0.20, 0.37]
Wave	-.325 (.013)***	[-0.35, -0.30]	-.313 (.016)***	[-0.34, -0.28]	-.321 (.016)***	[-0.35, -0.29]	-.357 (.033)***	[-0.42, -0.29]
Child PTE Sum	.015 (.019)	[-0.02, 0.05]	.020 (.020)	[-0.02, 0.06]	-.026 (.017)	[-0.06, 0.01]	-.025 (.036)	[-0.10, 0.05]
Control	.169 (.016)***	[0.14, 0.20]	.199 (.020)***	[0.16, 0.24]	.133 (.017)***	[0.10, 0.17]	.031 (.038)	[-0.04, 0.10]
Age	—	—	—	—	-.030 (.001)***	[-0.03, -0.03]	-.029 (.003)***	[-0.03, -0.02]
Female	—	—	—	—	-.128 (.026)***	[-0.18, -0.08]	-.095 (.054)	[-0.20, 0.01]
College	—	—	—	—	.452 (.028)***	[0.40, 0.51]	.270 (.066)***	[0.14, 0.40]
Race	—	—	—	—	-.614 (.035)***	[-0.68, -0.54]	-.184 (.198)	[-0.57, 0.20]
Health	—	—	—	—	—	—	.105 (.033)**	[0.04, 0.17]
PRS	—	—	—	—	—	—	-.022 (.178)	[-0.37, 0.33]
Adult PTE Sum	—	—	—	—	—	—	.017 (.030)	[-0.04, 0.08]
Child PTE Sum*Control	—	—	-.017 (.019)	[-0.05, 0.02]	-.017 (.016)	[-0.05, 0.01]	.003 (.034)	[-0.06, 0.07]
Wave*Child PTE Sum	—	—	-.015 (.016)	[-0.05, 0.02]	-.009 (.016)	[-0.04, 0.02]	.011 (.034)	[-0.05, 0.08]
Wave*Control	—	—	-.076 (.016)***	[-0.11, -0.04]	-.067 (.016)***	[-0.10, -0.04]	-.072 (.034)*	[-0.14, -0.01]
Wave*PTE*Control	—	—	.034 (.015)*	[0.00, 0.06]	.031 (.015)*	[0.00, 0.06]	.050 (.034)	[-0.02, 0.12]

Note. SE = standard error; PTE = potentially traumatic events; PRS = polygenic risk scores. Model 1: N = 3,031, Observations = 5,018, BIC = 11270.1; Model 2: N = 3,031, Observations = 5,018, BIC = 11279.0; Model 3: N = 3,019, Observations = 5,003, BIC = 10128.5; Model 4: N = 528, Observations = 982, BIC = 1892.8.

*p < .05. **p < .01. *** p <.001.

Table 4. *Research Question 2 – Change in Episodic Memory*

Variables	Model 1: Unadjusted		Model 2: Interaction		Model 3: Sociodemographic Variable Adjustment		Model 4: Structural Variable Adjustment	
	Estimate (*SE*)	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**	**Estimate (*SE*)**	**95% CI**
Intercept	-.038 (.022)	[-0.08, 0.01]	-.048 (.023)*	[-0.09, -0.00]	-.416 (.035)***	[-0.49, -0.35]	-.124 (.162)	[-0.44, 0.19]
Wave	-.106 (.021)***	[-0.15, -0.07]	-.082 (.026)**	[-0.13, -0.03]	-.109 (.025)***	[-0.16, -0.06]	-.091 (.053)	[-0.20, 0.01]
Child PTE Sum	.067 (.020)***	[0.03, 0.11]	.085 (.023)***	[0.04, 0.13]	.012 (.021)	[-0.03, 0.05]	.039 (.043)	[-0.05, 0.12]
Control	.169 (.016)***	[0.07, 0.14]	.099 (.026)***	[0.05, 0.14]	.084 (.021)***	[0.04, 0.12]	-.011 (.046)	[-0.10, 0.08]
Age	—	—	—	—	-.026 (.001)***	[-0.03, -0.02]	-.027 (.003)***	[-0.03, -0.02]
Female	—	—	—	—	.507 (.030)***	[0.45, 0.56]	.618 (.059)***	[0.50, 0.73]
College	—	—	—	—	.299 (.031)***	[0.24, 0.36]	.141 (.072)	[-0.00, 0.28]
Race	—	—	—	—	-.330 (.040)***	[-0.41, -0.25]	.092 (.216)	[-0.33, 0.52]
Health	—	—	—	—	—	—	.118 (.036)**	[0.05, 0.19]
PRS	—	—	—	—	—	—	.362 (.196)	[-0.02, 0.75]
Adult PTE Sum	—	—	—	—	—	—	-.025 (.033)	[-0.09, 0.04]
Child PTE Sum*Control	—	—	.021 (.022)	[-0.02, 0.06]	.010 (.020)	[-0.03, 0.05]	.066 (.041)	[-0.01, 0.15]
Wave*Child PTE Sum	—	—	-.039 (.025)	[-0.09, 0.01]	-.032 (.025)	[-0.08, 0.02]	-.056 (.053)	[-0.16, 0.05]
Wave*Control	—	—	.001 (.026)	[-0.05, 0.05]	.013 (.025)	[-0.04, 0.06]	-.002 (.053)	[-0.11, 0.10]
Wave*PTE*Control	—	—	-.028 (.024)	[-0.07, 0.02]	-.028 (.023)	[-0.07, 0.02]	-.035 (.054)	[-0.14, 0.07]

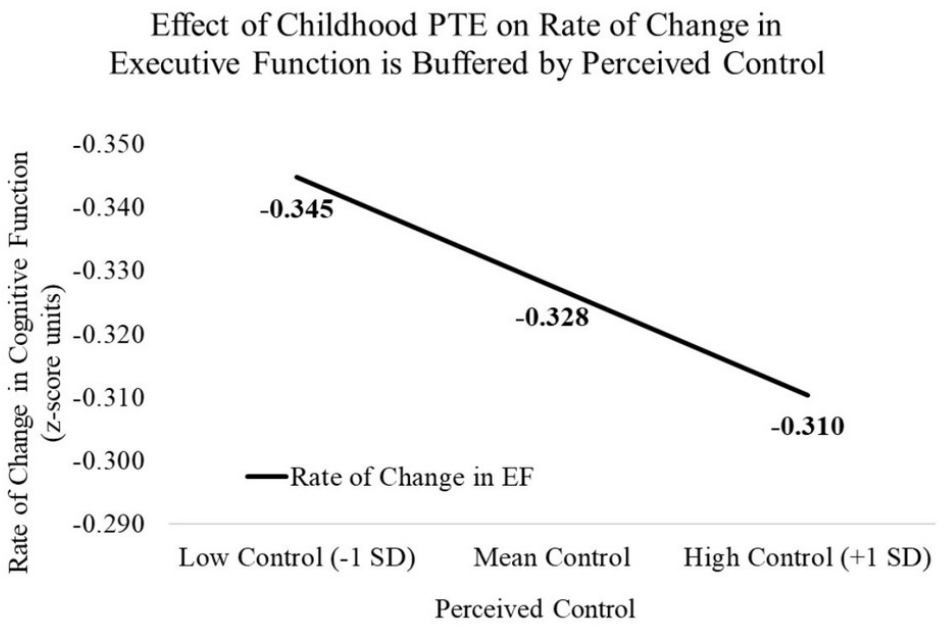
Note. SE = standard error; PTE = potentially traumatic events; PRS = polygenic risk scores. Model 1: N = 3,031, Observations = 5,008, BIC = 13566.8; Model 2: N = 3,031, Observations = 5,008, BIC = 13594.4; Model 3: N = 3,019, Observations = 4,993, BIC = 12773.5; Model 4: N = 528, Observations = 980, BIC = 2419.1.

*p < .05. **p < .01. *** p <.001.

Figure 1. *Effect of Childhood PTE on Rate of Change in Executive Function is Buffered by Perceived Control*

Note. SE = standard error; PTE = potentially traumatic events. Perceived control significantly moderated the relationship between childhood PTE exposure and the rate of change in EF (b = 0.03, SE = .01, p = .023, 95% CI: 0.01, 0.06) such that childhood PTE exposure was associated with a steeper rate of decline in EF among people with lower levels of perceived control (-1SD; b = -0.35, SE = 0.02, p < .001) compared to people with mean levels of perceived control (b = -0.33, SE = 0.01, p < .001) and higher levels of perceived control (+1SD; b = -0.31, SE = 0.02, p < .001).

Alt Text: Graphical representation showing that higher perceived control buffers the negative effect of childhood PTE on executive function decline. Rate of change attenuates from -0.345 to -0.310 as control increases.



Note. Perceived control significantly moderated the relationship between childhood PTE exposure and the rate of change in EF (b = 0.03, SE = .01, p = .023, 95% CI: 0.01, 0.06) such that childhood PTE exposure was associated with a steeper rate of decline in EF among people with lower levels of perceived control (-1SD; b = -0.35, SE = 0.02, p < .001) compared to people with mean levels of perceived control (b = -0.33, SE = 0.01, p < .001) and higher levels of perceived control (+1SD; b = -0.31, SE = 0.02, p < .001).

Alt Text: Graphical representation showing that higher perceived control buffers the negative effect of childhood PTE on executive function decline. Rate of change attenuates from -0.345 to -0.310 as control increases.

82x53mm (300 x 300 DPI)