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Childhood maltreatment and mid-life cognitive functioning: divergent psychosocial pathways linked to ethnoracial group

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Abstract

Childhood maltreatment has lasting adverse effects across multiple domains, contributing to cumulative disadvantage and accelerated aging. These outcomes may reflect not only individual vulnerability, but also ethnoracial-related differences in lived experience, highlighting how sociocultural contexts shape long-term risk. The effect of childhood maltreatment on cognitive functioning later in life varies, raising questions about the mechanisms that drive this association. Data were drawn from the Midlife in the United States (MIDUS) study. Participants were categorized into two racial groups for analysis; non-Hispanic white and ethnoracial minority. Physiological and psychosocial variables were pulled from wave II and cognitive variables were extracted from waves II and III. At wave II, the analytic sample included 692 adults (56% female, 19.5% ethnoracial minority, age: 53.43 ± 10.72 years). Serial mediation models evaluated whether depression and perceived stress linked childhood maltreatment to later cognitive decline, predicting wave III cognitive functioning while controlling for wave II cognitive decline, in the overall sample and by ethnoracial subsamples. In the full sample, the relationship between childhood maltreatment and cognitive decline was fully mediated via the indirect pathway from depression to perceived stress. Ethnoracial minority status significantly moderated the association between depression and perceived stress, yet, the subgroups estimates of the indirect pathway were not significantly different from each other. In the non-Hispanic white group, the total effect of childhood maltreatment on cognitive functioning was not significant (b = −0.0014, p = 0.283), but a significant indirect pathway existed through depression to stress (b = −0.0012, 95%CI [−0.0023, −0.0003]). However, in the ethnoracial minority group, the total and direct effects of childhood maltreatment on cognitive performance were significant (b = −0.0079, p = 0.001, and b = −0.0066, p = 0.013, respectively) with no significant indirect pathways. These findings highlight distinct psychosocial pathways that link childhood maltreatment to cognitive aging and underscore the importance of contextually relevant prevention and intervention strategies.

Keywords Childhood maltreatment · Cognitive decline · Depressive symptoms · Perceived stress · Cumulative disadvantage

Introduction

Child maltreatment is an urgent US public health concern, with prevalence rates remaining stable across recent decades (approximately 7.4 per 1000 children in 2023; U.S. Department of Health & Human Services, 2025). Entrenched socioeconomic inequalities, chronic community stress, and structural barriers to prevention may explain this persistence (Zhang et al., 2022). Exposure to any type of maltreatment including physical, emotional, or sexual abuse/neglect, heightens risk for mental and physical health problems across adulthood (De Bellis & Zisk, 2014; Struck et al., 2020; Xiao et al., 2023).

Although the psychological and physiological effects of childhood maltreatment are well established (De Bellis & Zisk, 2014; Jaworska-Andryszewska & Rybakowski, 2019; McCrory et al., 2011), its cognitive sequelae remain comparatively understudied and often not given prominence in the public health space (Hackman et al., 2010). Cognitive

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health, encompassing executive function, attention, working memory, and processing speed, is critical to adaptive functioning across the lifespan (Pechtel & Pizzagalli, 2011). Childhood maltreatment may compromise these abilities through both direct neurobiological insult (Heim et al., 2010; Stumps et al., 2026) and cumulative psychosocial processes. Over time, this process contributes to what has been termed neurocognitive "weathering"; the gradual erosion of cognitive resilience and neural efficiency due to cumulative stress exposure (Geronimus et al., 2006; Zahodne et al., 2019).

One critical pathway through which this weathering process unfolds is the development of depressive symptomatology across mid- and later life (Lorenzo et al., 2023). Childhood maltreatment robustly predicts the onset, persistence, and recurrence of depression well into adulthood (Yin et al., 2024). In midlife, depressive symptoms are consistently associated with impairment in executive functioning, processing speed, and episodic memory (Rock et al., 2014; Perini et al., 2019), and longitudinal studies indicate that cumulative depressive burden predicts accelerated cognitive decline and heightened dementia risk in later life (Kohler et al., 2018). Thus, depression may represent a key mechanism that allows early maltreatment to become psychologically and biologically embedded, contributing to long-term cognitive vulnerability.

Childhood maltreatment also dysregulates stress response systems, leaving affected individuals with heightened reactivity and greater sensitivity to subsequent stressors (Teicher et al., 2016). Remaining in a prolonged state of vigilance draws on attentional and executive resources, constraining cognitive flexibility (McLaughlin et al., 2014; Tottenham & Galvan, 2016). Over years and decades, sustained stress activation contributes to endothelial dysfunction, metabolic dysregulation, and inflammatory signaling; all of which are known to affect neural integrity and cognitive performance (Danese & McEwen, 2012; Marsland et al., 2015). These findings position cognitive decline not merely as a product of chronological aging, but as a downstream consequence of lifelong exposure to psychosocial stressors and their physiological embedding.

However, the literature remains inconsistent in quantifying these relationships. Some studies find robust associations between early maltreatment and later cognitive deficits, whereas others detect effects only among individuals with comorbid psychopathology or chronic stress exposure (Lupien et al., 2018; Tong et al., 2025). These discrepancies suggest that the pathway from childhood maltreatment to cognitive decline is indirect and context-dependent, shaped by the intervening psychological and physiological processes. In particular, perceived stress and depressive symptoms may serve as key mediators linking early adversity to cognitive outcomes. Individuals who internalize early adversity as a chronic sense of unsafety or diminished control may experience heightened stress reactivity throughout life, while those who develop depressive symptomatology may endure sustained emotional and neurochemical alterations that compromise neural health (Duman & Aghajanian, 2012; Frodl & O'Keane, 2013).

These pathways do not operate uniformly across populations. For ethnoracial minority individuals living in the United States, the same biological and psychosocial systems implicated above carry additional burdens. Structural racism, chronic discrimination, and socioeconomic marginalization function as compounding stressors that tax stress regulation, emotional processing, and inflammatory signaling—all mechanisms through which childhood maltreatment already exerts its toll (Brody et al., 2013; Geronimus et al., 2006). In this sense, racism and poverty operate as parallel and cumulative taxes on the same systems sensitized by early adversity. Many ethnoracial minority communities demonstrate meaningful resilience through cultural identity, spirituality, and collective coping that can buffer some of these effects (Jung, 2018; Upenieks, 2021), though these protective factors may differentially attenuate perceived stress and depression calling into question which is a more integral contributor to functional outcomes (Williams et al., 2024).

Emerging evidence suggests that the relationship between depressive symptoms and cognitive decline is not uniform across ethnoracial groups but instead reflects broader patterns of cumulative disadvantage (Andel et al., 2023; Beydoun et al., 2024; Brown et al., 2025). Recent longitudinal work using the Midlife in the United States (MIDUS) dataset demonstrated that depressive symptoms predict declines in cognitive functioning over time specifically among ethnoracial minority individuals, whereas this association is attenuated or absent among non-Hispanic White adults (Persin et al., 2025). This pattern is particularly notable given that ethnoracial minority populations often report lower overall prevalence of depression but greater chronicity, severity and reduced access to treatment, suggesting a disproportionate functional burden when symptoms do occur (Williams et al., 2007; Assari & Lankarani, 2016). Childhood maltreatment represents a critical upstream factor that may initiate these divergent trajectories, embedding vulnerability into both emotional and biological systems before the compounding effects of structural inequity fully accumulate (Danese & McEwen, 2012; Colich et al., 2020). Yet, the extent to which the long-term cognitive sequelae of childhood maltreatment unfold through different

psychosocial pathways across ethnoracial groups remain largely unexplored.

Despite decades of research linking childhood maltreatment to adverse health outcomes, the mechanisms underlying how early life adversity contributes to later-life cognitive decline remain only partially understood. Much of the existing literature has focused on clinical samples or isolated cognitive domains, providing limited insight into how multiple psychosocial processes interact or work in parallel across time to shape cognitive aging in community dwelling adults. Moreover, relatively few studies have examined whether these processes vary across ethnoracial groups, despite robust evidence that structural inequities, discrimination, and sociocultural factors fundamentally shape both the experience of adversity and the resources available to cope with its aftermath (Geronimus et al., 2006; Williams et al., 2019). Understanding these differential pathways is essential to explain why the same early exposure can yield distinct psychological and physiological outcomes across groups.

The current study expands on a prior study (Persin et al., 2025) by investigating how childhood maltreatment may become embedded in long-term patterns of psychological and cognitive functioning. Drawing on data from Waves II and III of the Midlife in the United States (MIDUS) study; we examined whether depressive symptoms and perceived stress mediate the relationship between childhood maltreatment and later-life cognitive decline. This approach allows for a more nuanced test of whether cognitive effects of early adversity arise primarily through emotional dysregulation or chronic stress perception or both, and if these mechanisms manifest differently between ethnoracial groups.

We hypothesized that individuals reporting higher levels of childhood maltreatment would exhibit greater depressive symptoms and perceived stress in midlife, and these psychosocial factors would, in turn, predict cognitive decline approximately ten years later (Zhang et al., 2024; Persin et al., 2025). Additionally, we anticipated that the dominant mediating pathway would differ by ethnoracial group; perceived stress being more central among non-Hispanic White participants and depression more prominent among ethnoracial minority participants (Kulshreshtha et al., 2023). By addressing these questions in a large, longitudinal, and socioeconomically diverse sample, this study contributes to a more comprehensive understanding of how childhood maltreatment becomes biologically and cognitively embedded. Ultimately, identifying these distinct pathways holds promise for informing responsive prevention and intervention efforts aimed at mitigating the long-term cognitive and emotional consequences of early life maltreatment.

Method

Study data

This secondary data project utilized the publicly available Midlife in the United States (MIDUS) study waves II (M2) and III (M3), including the Milwaukee sub-study that consists of a Black/African American population. M2 data were collected from 2004 to 2009, while M3 data were collected from 2013 to 2017. At M2, data were aggregated across the main phone survey (n = 5555 [main study (Ryff et al. 2021) + Milwaukee sub-study (Ryff et al., 2022a)]), cognitive phone testing (n = 4512 [Ryff & Lachman, 2023a]), and biomarker study (n = 1255 [Ryff et al., 2022b]) datasets, yielding a total sample size of 1152 participants. At M3, only the cognitive dataset (n = 3291 [Ryff & Lachman, 2023b]) was utilized to investigate cognitive change. For more details about the MIDUS data collection methods, study designs, and participant attrition rates, please see the information reported elsewhere (Dienberg Love et al., 2010; Hughes et al., 2018).

Participants

Participants (n = 460) were excluded from analyses due to the following criteria: incomplete M2 cognitive data (n = 136), no M3 cognitive data/lost at follow-up (n = 183), incomplete depressive symptom or perceived stress data at M2 (n = 4), and missing covariate data at M2 (n = 17). Those using antidepressants (n = 120) on average as group were significantly different compared to non-antidepressant users on all psychosocial predictors and covariate factors but not different cognitive functioning (see Supplemental Table 1 for more details); given these findings, those on antidepressants at M2 are likely representing a subgroup that is unique from the other community dwelling adults and were removed from the analyses. Thus, the final sample included 692 participants, 44.1% male, 80.5% White, and 53.4 ± 10.7 years old. The ethnoracial minority group (n = 135) was comprised of those who identified as Black/African American (n = 98), Hispanic/Latino (n = 26), Middle Eastern/North African (n = 4), Asian (n = 4), Native American/Alaskan Native (n = 2), or Other (n = 1). It should be noted that this binary coding, while necessitated by sample size constraints, does not capture the full heterogeneity of ethnoracial experience and should be interpreted accordingly. The sample characteristics (mean and frequencies) of the study variables are represented in Table 1 for the overall group and separated by ethnoracial minority status.

Table 1 Summary descriptive statistics [mean (M) and standard deviation (SD) or frequency (n) and percentage (%)] of all study variables for overall sample and by ethnoracial group status

	Overall (n = 692)		Non-Hispanic White (n = 557)		All Ethnoracial Minorities (n = 135)	
Variables	M/n	SD/%	M/n	SD/%	M/n	SD/%
M2 Ethnoracial Identity						
Non-Hispanic White	557	80.5%	557	100%	–	–
Black or African	98	14.2%	–	–	98	72.6%
Hispanic/Latino	26	3.7%	–	–	26	19.2%
Middle Eastern/North African	4	0.6%	–	–	4	3.0%
Asian	4	0.6%	–	–	4	3.0%
Native American/Alaskan native	2	0.3%	–	–	2	1.5%
Other	1	0.1%	–	–	1	0.7%
M2 Female	387	55.9%	301	54.0%	86*	63.7%
M2 Male	305	44.1%	256	46.0%	49	36.3%
M2 Education	7.6	2.5	7.8***	2.4	6.8	2.4
M2 Allostatic Load	8.4	3.2	8.4	3.2	8.5	2.7
M2 Age (years)	53.4	10.7	54.1**	10.7	50.9	10.2
M2 Cognition (BTACT)	0.25	0.92	0.37***	0.86	-0.25	0.98
M2 Childhood Maltreatment						
Total	36.8	13.2	36.1	12.8	39.5**	14.5
Emotional abuse	7.7	3.9	7.6	3.8	8.1	4.0
Physical abuse	6.8	2.8	6.6	2.7	7.6***	2.9
Sexual abuse	6.3	3.4	6.2	3.3	6.7	3.8
Emotional neglect	9.3	4.3	9.2	4.2	9.8	4.6
Physical neglect	6.7	2.6	6.5	2.5	7.4**	3.1
Minimization/Denial	0.58	0.94	0.61*	0.96	0.45	0.83
M2 Depressive Symptoms	7.3	7.0	6.8	6.7	9.6***	7.8
M2 Major Depression Diagnosis	79	11.4%	56	10.1%	23	17.0%
M2 Perceived Stress	11.5	6.0	11.0	5.8	13.3***	6.3
M3 Cognition (BTACT)	0.05	0.67	0.13***	0.64	-0.28	0.68

* $p < .05$, ** $p < .01$, *** $p < .001$. Significant group differences indicated on the group with the higher value. M2 = Mid-Life in the United States wave 2 data collection; M3 = Mid-Life in the United States wave 3 data collection; BTACT = Brief Test of Adult Cognition by Telephone; – = not applicable. For M2 Education, 7 is the equivalent of an associate's degree or 3 + years of college at a 4-year institution. For M2 Major Depression Diagnosis, participants self-reported whether or not they had been diagnosed with Major Depressive Disorder; it was not verified by a physician

Measures

Childhood maltreatment. The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1997) was used to measure child maltreatment severity. It consists of 28 items that assess childhood physical, emotional, and sexual abuse, physical and emotional neglect on a scale of 1 (*never true*) to 5 (*very often true*). The scale yields both subscales and a total score inclusive of all types of maltreatment; higher scores indicate more severe maltreatment. The scale has robust reliability and criterion validity in clinical and non-clinical samples (Bernstein et al., 1997, 2003).

Depressive symptoms. The well-validated Center of Epidemiologic Studies Depression (CES-D; Radloff, 1977) scale, a 20-item self-report questionnaire primarily utilized in research settings, assessed current depressive symptoms at M2. The total score can range from 0–60 with a higher score indicating greater depressive symptoms.

Perceived stress. The 10-item perceived stress scale (PSS-10; Cohen et al., 1983; Cohen & Williamson, 1988) was used to assess recent perceived stress levels at M2. Each item is rated on a scale from 0 (*never*) to 4 (*very often*). The total score can range from 0–40 with a higher score indicating higher perceived stress.

Cognitive functioning. The Brief Test of Adult Cognition by Telephone (BTACT) was used to estimate global cognitive function. It was designed to assess cognitive differences in various cognitive domains, including episodic verbal memory, working memory, executive function, and processing speed of non-demented adults (Tun & Lachman, 2006). The BTACT scoring consists of two subscales episodic memory and executive functioning which are combined and then converted to a z-score is assigned to the composite score to estimate globe functioning (Gurnani et al., 2015; Lachman et al., 2014). The BTACT was created to address the gap in telephone-based tests and is sensitive to normal cognitive functioning for adults without neurocognitive

diseases such as dementia while allowing for greater access for individuals for whom in-person testing is not ideal (Tun & Lachman, 2006).

Ethnoracial status. Participants were asked to endorse their racial origin(s) and afforded the opportunity to provide up to three among the following: White, Black and/or African American, Native American or Alaskan Native, Asian, Native Hawaiian or Pacific Islander, Other, or Don't Know. Any endorsement of a non-White response across the three variables was coded by their first ethnoracial minority indicated. In addition, a question asked participants whether or not they identified as having Spanish/Hispanic/Latino descent. Due to small sample sizes in other non-White identities, the ethnoracial responses were coded as 0 = non-Hispanic White identity and 1 = any ethnoracial minority identity.

Allostatic load. We constructed an estimate of multisystem dysregulation as a proxy for the accumulation of biological and physiological responses during everyday life; using a similar process previously published from the MIDUS dataset (e.g., Karlamangla et al., 2014). This allostatic load score is the sum of 26 biomarker values at M2. Dysregulation scores for biomarkers falling into the categories representing the cardiovascular (e.g., blood pressure), metabolic (e.g., cholesterol), neuroendocrine (e.g., urine catecholamines), and immune (e.g., C-reactive protein) systems were calculated using established clinical cutoff with 0 indicating individuals being within normal range and the value of 1 expressing levels of dysregulation. Other systems without well-established clinical cutoffs were incorporated into the calculation using the highest risk quartile of its distribution being coded as 1 and other quartiles coded as 0, accounting for the inverse association between several biomarkers (e.g., resting heart rate variability, grip strength, etc.) and suboptimal health outcomes.

Covariates. Demographic factors such as age (Lenehan et al., 2015), gender (Merrick & Brayne, 2024), and education (Lenehan et al., 2015) as well as allostatic load (D'Amico et al., 2020) from M2 that could influence the associations between childhood maltreatment and cognitive functioning were controlled for in the analyses. Furthermore, M2 cognitive functioning was entered as a covariate, allowing the investigation of the psychosocial factors on cognitive decline between waves M2 to M3.

Analytic plan

Statistical analyses were conducted with SPSS Statistics software (version 28). Means and standard deviation (SD) or frequency and percentage were used to summarize the sample characteristics. Independent T-tests and χ^2 analysis were used to examine group differences on continuous and categorical variables, respectively. Zero-order correlation analyses explored the relationships among the primary and secondary variables of interest in the overall sample. Mediation analyses were conducted using the PROCESS Macro (version 4.1) with 10,000 bootstrap samples, bias-corrected 95% CI, including model 91 for the full sample and model 6 for the sub-sample analyses. All continuous predictor variables (*i.e.*, X = childhood maltreatment, M_1 = depressive symptoms, M_2 = perceived stress) were mean centered prior to analysis to aid results interpretation and, if applicable, generate estimates to examine the interactions (Hayes, 2017). The data distributions for all key variables (predictor and outcomes) were examined for normalcy; if outliers were found, data were winsorized at the 5th/95th percentiles and analyses were replicated to mitigate the potential effect of the outliers without reducing the original sample size. If analyses with winsorized data were different than the raw data, the winsorized data summary statistics were reported; otherwise, the summary statistics from raw data were reported. All tests were two-tailed and set at a significance level of a = 0.05.

Results

Summary of sample characteristics and zero-order correlations

In the full sample, as expected, the non-Hispanic White group reported greater education and performed better on the cognitive testing at M2 and M3 compared to the ethnoracial minority group. The ethnoracial minority group reported more childhood maltreatment, depressive symptoms and perceived stress compared to the non-Hispanic White group. See Table 1 for more details. Among the covariate zero-order correlations, cognitive functioning at M2 and M3 was positively related to education, while negatively associated with age and allostatic load. Females reported greater childhood maltreatment compared to males. At M2, higher levels of childhood maltreatment were linked with elevated levels of depressive symptoms and perceived stress and lower levels of cognitive functioning at M3. Elevated levels of M2 depressive symptoms and M2 perceived stress were positively associated, and both psychosocial factors were statistically related to poorer cognitive functioning at M2 and M3. See Table 2 for more details.

The distributions of childhood maltreatment, depressive symptoms, and perceived stress were all left skewed as might be expected in a healthy, community dwelling adults. For childhood maltreatment, the winsorized mean ± standard error was 36.2 ± 0.43 compared to the raw mean ± standard error of 36.8 ± 0.50. For depressive symptoms, the

Table 2 Zero-order correlations among primary and secondary study variables for the overall sample

Variables	1	2	3	4	5	6	7	8	9
1. M2 Age	–								
2. M2 Ethnoracial Minority (yes)	−.12**	–							
3. M2 Sex (female)	.00	.08*	–						
4. M2 Education	−.04	−.15***	−.06	–					
5. M2 Allostatic Load	.35***	.02	−.04	−.16***	–				
7. M2 Childhood Maltreatment	−.13***	.10**	.10**	−.13***	.02	–			
8. M2 Depressive Symptoms	−.20***	.16***	.04	−.14***	−.03	.36***	–		
9. M2 Perceived Stress	−.22***	.15***	.07†	−.13***	−.08*	.35***	.74***	–	
10. M2 Cognitive Functioning	−.31***	−.27***	.02	.40***	−.11**	−.04	−.17***	−.12***	–
11. M3 Cognitive Functioning	−.40***	−.25***	−.01	.41***	−.14***	−.08*	−.12***	−.12***	.79***

n = 692. †*p* < .10, **p* < .05, ***p* < .01, ****p* < .001. M2 = Mid-Life in the United States wave 2 data collection; M3 = Mid-Life in the United States wave 3 data collection

Table 3 Direct and total effects across models

Path	Full Sample (N = 692) (b [95% CI])	Non-Hispanic White (N = 557) (b [95% CI])	Ethnoracial Minority (N = 135) (b [95% CI])
CM → Depressive Symptoms (a_1)	**.168 [.132, .204]**	**.158 [.117, .199]**	**.198 [.117, .280]**
CM → Perceived Stress (a_2)	**.036 [.012, .061]**	**.035 [.008, .062]**	.029 [−.032, .091]
Depressive Symptoms → Perceived Stress (d_1)	**.611 [.558, .664]**	**.611 [.559, .663]**	**.506 [.384, .628]**
Depressive Symptoms → Cognitive Decline (b_1)	.005 [−.001, .011]	**.009 [.002, .016]**	−.002 [−.015, .011]
Perceived Stress → Cognitive Decline (b_2)	**−.010 [−.017, −.003]**	**−.013 [−.021, −.004]**	−.007 [−.021, .008]
CM → Cognitive Decline (Direct; c′)	−.002 [−.005, .000]	−.001 [−.004, .002]	**−.007 [−.012, −.001]**
CM → Cognitive Decline (Total; c)	—	−.001 [−.004, .001]	**−.008 [−.013, -.003]**

Bolded pathways are significant. CM = Childhood maltreatment. Full sample estimates from Model 91 in PROCESS (moderated serial mediation); subgroup estimates from PROCESS Model 6. Total effect (c) is not reported for the full sample as it is conditional on ethnoracial minority status in Model 91. — = not applicable

winsorized mean ± standard error was 7.0 ± 0.23 compared to the raw mean ± standard error of 7.3 ± 0.27. For perceived stress, the winsorized mean ± standard error was 11.4 ± 0.22 compared to the raw mean ± standard error of 11.5 ± 0.23.

The pathways between childhood maltreatment and mid-life cognitive decline

Full sample

In the full sample, childhood maltreatment predicted depressive symptoms and both childhood maltreatment and depressive symptoms were significantly related to perceived stress (see Table 1). Ethnoracial minority status did not have a main effect on perceived stress (d_2: $b = .361$, SE = .415, $t = .871$, $p = .384$, 95% CI [−.453, 1.175]), but it moderated the relationship between depressive symptoms and perceived stress (d_3: $b = -.109$, SE = .051, $t = -2.132$, $p = .0334$, 95% CI [−.210, −.009]), such that the association was stronger among non-Hispanic White participants ($b = .611$, SE = .027, $t = 22.537$, $p < .0001$, 95% CI [.558, .665]) than among ethnoracial minority participants ($b = .502$, SE = .046, $t = 10.882$, $p < .0001$, 95% CI [.411, .593]). Perceived stress significantly predicted cognitive decline, the direct effect of childhood maltreatment was trending, and depressive symptoms did not directly predict cognitive decline (see Table 3).

The conditional indirect effect of childhood maltreatment → depressive symptoms → perceived stress → cognitive decline was significant for both the non-Hispanic White and ethnoracial minority groups (see Table 4). However, despite ethnoracial minority status moderating the depressive symptoms → perceived stress pathway, the index of moderated serial mediation was not significant ($b = .0002$, SE = .0001, 95% BCa CI [.0000, .0005]). These findings suggest that childhood maltreatment's negative effect on cognitive decline operates through higher levels of depressive symptoms leading to higher levels of perceived stress, regardless of ethnoracial group. Figure 1 provides the model summary of the unstandardized betas for Model 91. When repeating this analysis with winsorized data, the results were consistent with the full dataset outcomes, indicating that raw data results are robust and are not driven by extreme observations.

Non-hispanic white subsample

Among non-Hispanic White participants, childhood maltreatment predicted greater depressive symptoms, and both childhood maltreatment and depressive symptoms

Table 4 Indirect effects from child maltreatment to cognitive decline

Variables	*n*	Indirect effect (*b*)	Standard error (SE)	[95% CI]
Model 1: Full Sample				
CM → Dep. → Stress→ Cog. Decline (NH White)	**692**	**−.001**	**.000**	**[−.002, −.000]**
CM → Dep. → Stress→ Cog. Decline (Minority)	**692**	**−.001**	**.000**	**[−.002, −.000]**
CM → Dep. → Cog. Decline	692	.001	.001	[−.001, .002]
CM → Stress→ Cog. Decline	692	−.000	.000	[−.001, .000]
Model 2: Non-Hispanic White Subsample				
CM → Dep. → Stress→ Cog. Decline	**557**	**−.001**	**.001**	**[−.002, −.000]**
CM → Dep. → Cog. Decline	557	.001	.001	[−.000, .003]
CM → Stress→ Cog. Decline	557	−.000	.000	[−.001, .000]
Model 3: Ethnoracial Minority Subsample				
CM → Dep. → Stress→ Cog. Decline	135	−.001	.001	[−.002, .001]
CM → Dep. → Cog. Decline	135	−.000	.001	[−.003, .002]
CM → Stress→ Cog. Decline	135	−.000	.000	[−.001, .000]

Bolded pathways are significant. CI=confidence interval; CM=Child maltreatment; Dep=Depression; Cog=Cognitive; NH=non-Hispanic; For Model 1 (full sample, Model 91 in PROCESS), the serial pathway is conditional on ethnoracial minority status; conditional indirect effects are reported at each level of the moderator. Models 2 and 3 are unconditional PROCESS Model 6 analyses within each subsample

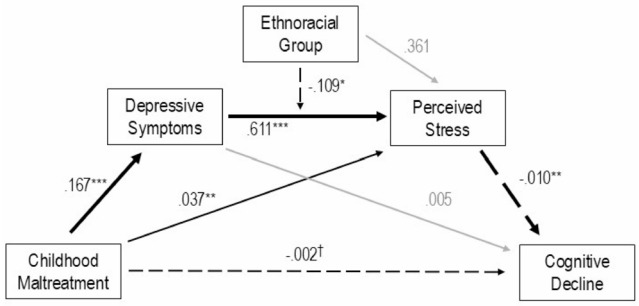


Fig. 1 Summary of PROCESS macro model 91 examining the serial mediational role of depressive symptoms and perceived stress between childhood maltreatment and cognitive decline during mid-life adulthood. Solid lines indicate positive relationships while dashed represent negative relationships. Gray lines and coefficients are nonsignificant, while black are significant. The significant indirect pathway is indicated by thickened arrows. The indirect pathway was significant for both non-Hispanic White group ($b=-.0010$, SE=.0004, 95% BCa CI [−.0019, -.0002]) and ethnoracial minority group ($b=-.0008$, SE=.0004, 95% BCa CI [−.0016, −.0001]). $^{\dagger}p<.10$, $*p<.05$, $**p<.01$, $***p<.001$

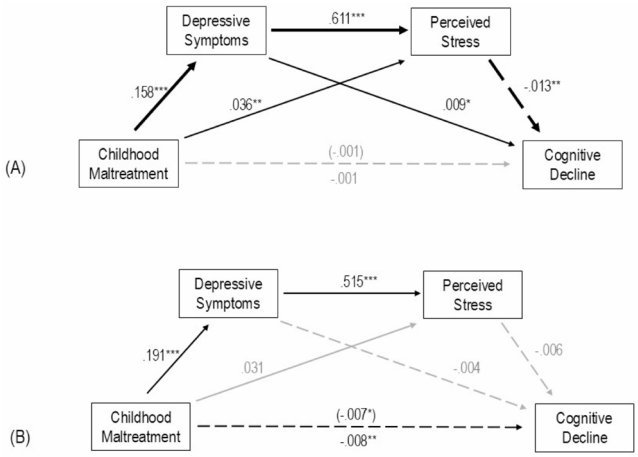


Fig. 2 Summary of PROCESS macro model 6 examining the serial mediational role of depressive symptoms and perceived stress between childhood maltreatment and cognitive decline during mid-life adulthood in non-Hispanic White group (A) and ethnoracial minority group (B). Solid lines indicate positive relationships while dashed represent negative relationships. Gray lines and coefficients are nonsignificant, while black are significant. The significant indirect pathway is indicated by thickened arrows. $*p<.05$, $**p<.01$, $***p<.001$

predicted higher perceived stress (see Table 3). Perceived stress significantly predicted cognitive decline; unexpectedly, depressive symptoms significantly predicted cognitive improvement once perceived stress was included in the model (see Table 3). Childhood maltreatment did not predict change in cognitive functioning directly, and the total effect of childhood maltreatment on cognitive decline was nonsignificant (see Table 3). The serial indirect effect through depressive symptoms and perceived stress was the only significant indirect pathway (see Table 4), suggesting that childhood maltreatment's negative effect on cognitive decline operates through higher levels of depressive symptoms leading to higher levels of perceived stress. Figure 2A provides the model summary of the unstandardized betas for Model 6 among the non-Hispanic White group. When repeating this analysis with winsorized data, the results were consistent with the full dataset outcomes, indicating that raw data results are robust and are not driven by extreme observations.

Ethnoracial minority subsample

In the ethnoracial minority sample, childhood maltreatment predicted higher levels of depressive symptoms but did not predict perceived stress. Higher levels of depressive symptoms predicted higher levels of perceived stress, but did not predict cognitive decline. Perceived stress also did not predict cognitive decline (see Table 3) while childhood

maltreatment did directly predict cognitive decline and t (c': $b = -.007$, SE = .003, $t = -2.533$, $p = .0126$, 95% CI [−.012, −.002]). The total effect was significant (see Tables 3 and 4). None of the conditional indirect effect pathways were significant; indicating that the association between childhood maltreatment and cognitive decline among the ethnoracial minority group is not accounted for by the proposed mediators. Figure 2B provides the model summary of the unstandardized betas for Model 6 among the ethnoracial minority group. The lack of significant indirect pathways, alongside the significant direct effect of childhood maltreatment on cognitive functioning, suggests that the mediation pattern observed among non-Hispanic White adults may not generalize to the ethnoracial minority group. Instead, these findings are consistent with a more direct association between childhood maltreatment and cognitive outcomes, raising the possibility that the psychosocial processes among these variables differ for this group. When repeating this analysis on winsorized data, the results were consistent with the full dataset outcomes, indicating that results are robust and are not driven by extreme observations. Additionally, given none of the indirect pathways could explain the relationship between childhood trauma and cognitive decline and the previously observed significant negative relationship between depressive symptoms and cognitive decline within in this sample (Persin et al., 2025), we examined an alternative model—depressive symptoms predicted cognitive decline with childhood trauma and perceived stress as mediators—that can be found in the supplemental file.

Childhood maltreatment subscale analyses

A post-hoc exploration examining the different subscales of the childhood trauma questionnaire was conducted. For the non-Hispanic White group, the direct and total effects had some variation. For example, the sexual abuse subscale maintained a significant direct and total effect on cognitive decline when depressive symptoms and perceived stress were in the model; neither the total childhood maltreatment score nor any other subscale did. For the ethnoracial minority group, specific subscales appear to carry the total childhood maltreatment including physical and sexual abuse as both the direct and total effects of the subscale on cognitive decline were significant, similar to the total score. Please review Supplemental Table 2 for all childhood maltreatment subscales direct and total effects separated by ethnoracial minority group. Among the non-Hispanic White group, all childhood maltreatment subscales—emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect—mirrored the indirect pathway of the total score, except for the emotional abuse subscale, where the indirect pathway through perceived stress to cognitive decline was also significant. Among the ethnoracial minority group, all the subscale analyses for indirect effects mirrored the total score; there were no significant indirect effects. See Supplemental Tables 3 and 4 for all childhood maltreatment subscales indirect effects from the subscale to cognitive decline by subgroup, non-Hispanic Whites and ethnoracial minority group, respectively.

Discussion

This study investigated the relationship between childhood maltreatment and later-life cognitive decline by testing a series of complementary models designed to clarify both mechanistic pathways and ethnoracial differences in risk trajectories. In the full sample, as hypothesized, childhood maltreatment was associated with cognitive decline indirectly through higher depressive symptoms and elevated perceived stress. Depressive symptoms did not retain a direct association with cognitive decline once perceived stress was included in the model, such that perceived stress was the only significant direct predictor of cognitive decline. In the larger, full sample analysis, this pattern was exhibited in both the non-Hispanic White and ethnoracial minority groups; even though the ethnoracial group moderated the relationship between depressive symptoms and perceived stress, the estimates of the indirect pathways were not statistically different (i.e., the lower 95% confidence interval was .0000) between the two groups.

This pattern aligns with population-based research indicating that depressive symptoms often exert their cognitive effects indirectly through stress-related processes rather than functioning as a direct neurocognitive risk factor (Juster et al., 2010; Lupien et al., 2009; Ramakrishnan et al., 2025). Prior studies emphasize that depressive symptoms are highly heterogeneous, frequently transient, and inconsistently associated with long-term cognitive decline unless embedded within broader contexts of chronic stress or cumulative adversity (Musliner et al., 2016; Zahodne et al., 2019). Furthermore, depression effects are often attenuated when stress exposure is more comprehensively modeled (Anderson et al., 2022), suggesting that stress rather than depressive symptomatology may be the operative mechanism linking psychosocial distress to downstream cognitive outcomes.

Despite the full sample model not clearly reinforcing that the non-Hispanic White and ethnoracial minority groups had different indirect pathways from childhood maltreatment to cognitive decline, the groups reported different levels on average of being exposed to childhood maltreatment as well as depressive symptoms and perceived stress; thus, due to the overall model including 4 times as many

non-Hispanic White individuals compared to the ethnoracial minority group, we ran ethnoracial subgroup analyses to determine if effects within the smaller subgroup were being overshadowed by the larger subgroup.

Among non-Hispanic White adults, childhood maltreatment influenced cognitive decline through a serial mediation pathway involving depressive symptoms and subsequent elevations in perceived stress. Unexpectedly, higher depressive symptoms were associated with cognitive *improvement* once perceived stress was included in the model. This finding is unlikely to reflect a protective effect of depression itself. One possibility is that depressive symptoms in this group capture increased symptom recognition, help-seeking behavior, or treatment engagement—processes more common among individuals with higher education, greater cognitive reserve, and greater health literacy, all of which are known buffers against cognitive decline (Ponsoni et al., 2020; Stern, 2002; Stern et al., 2020; Triolo et al., 2025).

Another consideration is the high correlation (r=.74 for the whole sample; see Table 2) between depressive symptoms and perceived stress, indicating shared variance and raising the possibility of multicollinearity or a suppression effect when both variables are included in the model. However, the data suggests that multicollinearity was not a factor as statistics do not raise concern within the whole sample or either subgroup, all variance inflation factors (VIFs) range from 1.076 to 1.271 and tolerances ranged from .787 to .929. Further, the data also do not indicate the presence of a suppression effect as the partial correlation is smaller than the zero-order correlation, r=.71 (r=.73 non-Hispanic White only) and r=.74 (r=.75 non-Hispanic White only), respectively.

It is also possible that mild or subclinical depressive symptoms co-occur with rumination, introspection, or cognitive engagement that transiently maintain performance on global cognitive measures, particularly in higher cognitive reserve individuals (Sliwinski et al., 2006; Tong et al., 2025). Importantly, depressive symptoms were not protective in isolation, and childhood maltreatment influenced cognitive decline among non-Hispanic White group exclusively through the serial pathway involving depressive symptoms and perceived stress. Alternatively, perceived stress may be more proximally related to physiological dysregulation and neurocognitive aging, such that accounting for stress absorbs variance shared with depressive symptoms that is relevant to cognitive decline (Juster et al., 2010; Lupien et al., 2009).

A different pattern emerged among ethnoracial minority adults. In this group, the association between childhood maltreatment and cognitive decline was not explained by either depressive symptoms or perceived stress. However, given prior evidence that depressive symptoms predicted cognitive decline among ethnoracial minority adults (Persin et al., 2025), a reversed model was tested to evaluate whether this association might be statistically accounted for by childhood maltreatment exposure. In this model (see Supplemental Fig. 1), childhood maltreatment fully mediated the association between depressive symptoms and cognitive decline, suggesting that, among ethnoracial minority adults, depressive symptoms may reflect downstream manifestations of early-life adversity, rather than depression operating as an independent mechanistic pathway.

Together, these stratified analyses indicate that aggregate models obscure meaningful ethnoracial heterogeneity in pathways linking early adversity to cognitive aging. Among the non-Hispanic White group, childhood maltreatment appears to influence cognitive decline through psychologically mediated stress processes, whereas among ethnoracial minority adults, childhood maltreatment may be more directly embedded in cognitive aging processes. These findings are consistent with life-course and stress-process frameworks emphasizing cumulative adversity, stress proliferation, and differential exposure rather than differential vulnerability (George & Lynch, 2003; Turner & Lloyd, 1995; Zahodne et al., 2019), underscoring the importance of modeling sociocultural context when examining pathways linking early-life adversity to later-life cognitive decline.

These subgroup differences can be further understood through the integration of cumulative advantage and disadvantage, social safety, and minority stress frameworks. The theory of cumulative advantage and disadvantage posits that early-life adversity initiates cascading processes of risk accumulation across the life course, increasing exposure to subsequent stressors and compounding their effects on health and cognition, while early advantages confer increasing protection over time (Zahodne et al., 2016). The Social Safety Model provides a mechanistic account of how these cumulative experiences become biologically embedded, suggesting that persistent perceptions of social threat, rejection, discrimination, or chronic unsafety activate stress-related neural and immune pathways that promote inflammation and disrupt neurocognitive functioning (Eisenberger & Cole, 2012; Slavich, 2020).

The Minority Stress Model (Meyer, 2003) further specifies that individuals from marginalized groups are disproportionately exposed to these conditions of social unsafety due to enduring stigma, discrimination, and structural inequities, thereby intensifying stress reactivity and downstream biological burden across the lifespan (Lewis et al., 2015). Over time, repeated activation of these stress-response systems contributes to elevated allostatic load, reflecting cumulative physiological wear-and-tear that has been linked to poorer cognitive functioning and neurostructural and neurofunctional alterations (Dobielska et al., 2022; Karlamangla

et al., 2014; Lenart-Bugla et al., 2022). Together, these integrated frameworks suggest that the more direct association between childhood maltreatment and cognitive decline observed among ethnoracial minority adults may reflect lifelong exposure to social unsafety and cumulative biological burden, whereas psychologically mediated pathway observed in among the non-Hispanic White group may be more prominent in contexts characterized by greater access to protective resources.

Several limitations should be considered when interpreting these findings, particularly with respect to sample composition, measurement constraints, and developmental context. First, although a strength of the study was the explicit examination of ethnoracial minority status, we were limited in our ability to examine heterogeneity across unique ethnoracial minority groups. The ethnoracial minority sample was notably smaller than the non-Hispanic white group around 19.5% of the total sample, and predominantly Black/African American, limiting our ability to further stratify the sample by ethnoracial subgroup. This approach treats the minority experience as a monolith, obscuring meaningful differences in place of origin, time in the United States, cultural norms, and the broader social contexts that shape stress, depression, and cognitive performance. The stress-sensitization and allostatic burden mechanisms implicated in our findings are likely to manifest differently across specific ethnoracial communities (Brown et al., 2020; Zahodne et al., 2016), and future work should stratify by subgroup and consider intersectional frameworks where sample sizes permit.

Second, this sample was relatively older (mean age = 53 years in 2004–09), which may have influenced reporting patterns, as mental health issues were more stigmatized during the formative windows (*e.g.*, 1950s) for many of the participants (Conner et al., 2010; Pescosolido et al., 2021). Additionally, retrospective reports of childhood maltreatment may be subject to recall bias in this age group, with some evidence suggesting that current psychopathology can exacerbate recollections of past adversity (Baldwin et al., 2024). Importantly, reporting patterns may also differ across ethnoracial groups, with minority adults potentially underreporting maltreatment exposure due to cultural stigma and distrust, potentially resulting in conservative estimates of adversity in the ethnoracial minority subsample and should be considered when interpreting group differences (Bontemps-Jones et al., 2025; Fontes & Plummer, 2010). Finally, perceived stress and depressive symptoms were only assessed at one timepoint, limiting the ability to capture fluctuations in psychological distress across the life course and to more precisely model their temporal relationships with cognitive decline.

The present findings have important clinical implications, particularly for the assessment and monitoring of stress- and depression-related sequelae of childhood maltreatment across diverse populations. A growing body of evidence supports the use of trauma-informed and stress-targeted interventions that address the downstream psychological and physiological consequences of early life adversity (Watkins et al., 2018). Importantly, CTQ scores in our sample fall within the subclinical range, below thresholds typically seen in psychiatric or treatment-seeking populations. This suggests that the pathways identified here are relevant even at levels of maltreatment exposure present in the broader community (Bernstein et al., 2003). Accordingly, routine screening for childhood maltreatment history and perceived stress in midlife and older adults may help identify individuals at elevated risk for cognitive decline, particularly in primary care and cognitive health settings where early intervention is most feasible.

When such individuals are identified, a range of trauma-informed approaches may be relevant. Trauma-focused cognitive behavioral therapy (TF-CBT), cognitive processing therapy and prolonged exposure therapy have all demonstrated efficacy in reducing affective distress, stress reactivity, and trauma-related depressive symptoms (Ehring et al., 2014; Watkins et al., 2018; Weber et al., 2021). Given the central role of perceived stress identified in the present study, stress-targeted approaches such as mindfulness-based stress reduction are also of particular interest. These interventions have been shown to reduce perceived stress, normalize neuroendocrine [e.g., hypothalamic–pituitary–adrenal (HPA) axis] functioning, and lower inflammatory markers among trauma-exposed adults (Black & Slavich, 2016; Holzel et al., 2011), with emerging evidence suggesting benefits for attention, executive control, and stress regulation in midlife and older adults (Fountain-Zaragoza & Prakash, 2017; Holzel et al., 2011). Whether stress reduction translates to meaningful attenuation of cognitive risk over time remains an important direction for future investigation.

Finally, the present findings underscore the importance of considering childhood maltreatment exposure even when depressive symptoms appear mild or when stress is the dominant presenting concern. Prior work indicates that maltreatment-related stress processes can exert lasting effects on physiological regulation and cognitive health independent of overt psychopathology (Slavich, 2020; Turner & Lloyd, 1995). These findings suggest that early attention to maltreatment-related stress may help interrupt pathways of cumulative disadvantage and long-term cognitive vulnerability (Karlamangla et al., 2014; Zahodne et al., 2016). Multimodal interventions that combine psychotherapy with lifestyle-based stress reduction strategies, including physical activity and sleep interventions, may further support

resilience by addressing both psychological and physiological components of stress-related risk (Creswell et al., 2012; Eisenberger & Cole, 2012; Lupien et al., 2009).

Future research should employ longitudinal designs with repeated assessments of depressive symptoms, perceived stress, and cognitive functioning to clarify temporal ordering and identify periods of heightened vulnerability across the life course (Musliner et al., 2016; Sliwinski et al., 2006). Modeling trajectories of stress and psychological distress, rather than relying on single time-point measures, will be critical for distinguishing transient distress from chronic processes that confer cognitive risk (Zahodne et al., 2016). Integrating biological markers, including inflammation, HPA axis functioning, autonomic regulation, neuroimaging indices, and allostatic load, may help elucidate the biological pathways linking childhood maltreatment to cognitive decline, particularly among ethnoracial minority adults (Juster et al., 2010; Karlamangla et al., 2014; Lupien et al., 2009). Larger and more diverse samples are needed to examine subgroup and intersectional heterogeneity (George & Lynch, 2003). Future studies should be intentionally designed to capture heterogeneity across specific ethnoracial groups by incorporating culturally responsive measures, sufficient subgroup sample sizes, and explicit attention to sociostructural contexts that shape stress exposure and health trajectories. Finally, intervention studies targeting stress-related processes within trauma-informed and culturally responsive frameworks may clarify whether cognitive risk associated with early adversity is modifiable (Slavich, 2020).

In conclusion, childhood maltreatment can influence cognitive decline during mid-life. The psychosocial pathways mediating the relationship appear to diverge whether an individual falls within the ethnoracial majority (non-Hispanic White) or minoritized group (e.g., Black/African American, Hispanic, Asian, Middle Eastern/North African, etc.), suggesting that culturally and contextually sensitive prevention and intervention strategies should be developed. Perceived stress levels remained a primary factor in cognitive decline for the non-Hispanic White group, while the effect of depressive symptoms on cognitive decline among the ethnoracial minority group was fully explained by childhood maltreatment. Unfortunately, childhood maltreatment is common and difficult to eliminate; these findings provide potential avenues for preventing or mitigating cognitive decline during mid-life associated with early-life adversity.

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Declarations

Conflict of interest The authors have no relevant financial or non-financial interests to disclose.

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