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Adverse childhood events and cognitive function among young adults: Prospective results from the national longitudinal study of adolescent to adult health

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Abstract

Background: Adverse childhood experiences (ACEs) may have lasting impacts on cognition.

Objective: To determine if ACE exposure is prospectively associated with cognition in young adults. We hypothesized that deprivation- and threat-type ACEs as well as higher cumulative ACE exposure predict poorer cognition.

Participants & setting: Participants were from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a prospective cohort investigation of U.S. adolescents followed to adulthood. Current study participants were 18–24 years old (Wave III), 24–32 years old (Wave IV), and 31–42 years old (Wave V). The maximum Wave IV sample was 12,288 adults; Wave V was 1277 adults.

Methods: History of ACEs were assessed at Wave III. Three cognitive indicators were assessed at Wave IV and Wave V using the Rey Auditory Verbal Learning Test (immediate and delayed verbal memory) and the Digit-Span Backward Task (working memory).

Results: The deprivation ACE of not-having-basic-needs met was associated with poorer working (β = 0.14, CI_{95} −0.26, −0.01), immediate (β=−0.29, CI_{95} −0.43, −0.15), and delayed memory (β=−0.27, CI_{95} −0.43, −0.12) at Wave IV; poorer immediate (β=−0.47, CI_{95}−0.79, −0.16) and delayed memory (β=−0.33, CI_{95} −0.65, −0.01) at Wave V. The threat ACE of sexual abuse was associated with poorer immediate (β=−0.40, CI_{95} −0.62, −0.17) and delayed memory (β=−0.29, CI_{95} −0.55, −0.03) at Wave IV. Higher cumulative ACEs predicted poorer delayed memory (β =−0.05, CI_{95} −0.10, −0.01) at Wave V.

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Conclusions: Higher ACEs, especially deprivation-type, were prospectively linked to poorer cognition. Early wide-scale screening/tailored treatments addressing ACEs and cognitive function may be warranted.

Keywords

Adverse childhood experiences; Early life adversity; Cognitive function; Memory; Executive function

1. Introduction

Adverse childhood experiences (ACEs) predict negative health outcomes across multiple biological, psychological, social, and, more recently, neurocognitive domains. The “ACE pyramid,” representing the ACEs-to-health framework (Centers for Disease Control & Prevention., 2020), situates disrupted neurodevelopment as one of the basic mechanisms by which early trauma and adversity lead to consequent morbidity and premature mortality. Substantial evidence from the animal literature and evolving empirical support in humans show that ACEs are linked to alterations in brain structures and poorer neurocognitive functions across a variety of brain regions (e.g., limbic, hippocampal, prefrontal) and neuropsychological tests (e.g., Stroop Task, Go/No-Go tests, Spatial or Digit Span, Rapid Visual Information Processing, Working Memory, and etcetera) (Anda et al., 2006; Bale et al., 2010; Berens, Jensen, & Nelson, 2017; Calem, Bromis, McGuire, Morgan, & Kempton, 2017; Carvalho et al., 2016; Cohen et al., 2006; Danese et al., 2017; Dannlowski et al., 2012; Davis et al., 2018; Hawkins et al., 2020; Irigaray et al., 2013; Lemche, 2018; Majer, Nater, Lin, Capuron, & Reeves, 2010; Mansueto et al., 2019; Merz & Noble, 2017; Shonkoff et al., 2012; Teicher, Tomoda, & Andersen, 2006). Importantly, ACEs are also linked to increased risk for major neurocognitive disease, such as Alzheimer’s-type dementia (Lemche, 2018). Prospective cohort studies show that exposure to early life adversity confers 2.15–4.22 increased odds of developing late onset dementia (Lemche, 2018). As summarized by Danese et al. (2017), individuals with ACEs have pervasive and clinically significant deficits in cognitive functions across a wide swath of cognitive domains: processing speed, executive function, perceptual reasoning, memory, and verbal comprehension.

Such findings linking ACEs exposure to future brain function, health, and disease is concerning given the high prevalence of ACEs (Centers for Disease Control Prevention (CDC) Kaiser Permanente (2017)). An estimated 64 % of adults endorse one or more ACEs prior to the age of 18, while 22 % report at least three ACEs (Centers for Disease Control Prevention Kaiser Permanente, 2017). ACEs are defined as traumatic events (e.g., abuse, deprivation or neglect, and household challenges) and ACE exposure has a dose-response impact on health, such that negative outcomes increase in a graded fashion as a person’s ACEs score increases (Dong, Anda, Dube, Giles, & Felitti, 2003; Dube et al., 2003; Murphy et al., 2014).

The number of people living with major neurocognitive deficits and disease is also widespread – with significant personal and societal costs. Almost 44 million people lived with dementia in 2016, an increase of 117 % from 1990 (Nichols et al., 2019).

Even more recently, subclinical or relative cognitive deficits (especially in the executive function domains such as inhibitory control and overriding dominant responses) have been observed among younger and otherwise healthy populations and has been linked with physiological dysregulation, such as elevated glucose and obesity (Hawkins, Gunstad, Calvo, & Spitznagel, 2016; Gunstad et al., 2007). As such, even relative cognitive deficits – among young samples and those without overt cognitive impairment – have garnered attention as preventative and therapeutic targets that contribute to biopsychosocial health.

The high rates and adverse outcomes of both early life adversity and deficits in brain health make it clear that these topics are burgeoning public health concerns. Investigations of ACEs-cognition relationships, particularly in younger cohorts, may provide critical information on how to prevent or to treat these inter-related risk factors for poor health before clinical manifestations of disease occur. To contribute to the evidence base of ACE exposure and subsequent cognitive function in a younger, dementia-free cohort, the present study used data from the National Longitudinal Study of Adolescent to Adult Health or Add Health (Harris, 2013).

The current investigation builds on the work of Dunn et al. (2016) who reported that the only ACE related to worse cognitive outcomes at follow-up was sexual abuse exposure reported during adolescence, and that this effect was attenuated by adding socioeconomic status (SES). We build on their contribution in several important ways by 1) adding examinations of deprivation-type ACEs via two neglect indicators as well as testing a cumulative ACE score of combined threat-type and neglect-type ACEs, 2) examining cognitive function with three cognitive function indicators: short-term memory, long-term memory, and working memory with the most recent follow-up cognitive data (Wave V), and 3) adjusting for additional confounders that may be linked to previous ACE exposure and/or poorer cognitive function: alcohol use, cigarette use, and baseline verbal ability (Wave I). Such analyses are important given the evidence that ACEs-cognition associations may differ by ACE type: threat versus deprivation (Mansueto et al., 2019; Sheridan & McLaughlin, 2014). A deprivation-type ACE is one in which expected social cognitive inputs are absent or limited (e.g., neglect), whereas a threat-type ACE is one in which unexpected risk to bodily integrity and physical health is present (e.g., physical or sexual abuse) (Sheridan & McLaughlin, 2014). We hypothesized that after adjustment for confounders 1) both deprivation- and threat-type ACEs at baseline would be linked to poorer performance on follow-up measures of cognitive performance, and 2) that higher cumulative baseline ACE exposure would also predict lower cognitive function scores at follow-ups.

2. Methods

2.1. Sample

We used data from Add Health, a prospective cohort study of a nationally representative sample of U.S. adolescents followed to adulthood. The original adolescent sample (1994–1995, 11–18 years old, Wave I) used systematic sampling methods and implicit stratification to ensure that the high schools ($n = 80$) and middle schools ($n = 52$) were representative of U.S. schools with respect to region of country, urbanicity, size, type, and ethnicity. There have been five waves of data collection. For this study, we used restricted-use data from

Wave III (defined as baseline for this study), collected from 2001 to 2002 when subjects were 18–24 years old, Wave IV, collected in 2008 when subjects were 24–32 years old, and Wave V, collected in 2016–2018 when subjects were 31–42 years old. Further details about the study design can be found elsewhere (Harris, 2013). The University of North Carolina Institutional Review Board approved all Add Health study procedures. Written informed consent was obtained.

2.2. Measures

2.2.1. Baseline predictor variables: adverse childhood experiences—

In Wave III, participants were asked to retrospectively self-report on the presence of exposure to ACEs. Physical abuse was defined as being slapped, hit, or kicked. Sexual abuse was defined as being forced to engage in sexual relations with an adult caregiver. Physical and sexual abuse have been categorized as threat-type ACEs as they represent harm to physical integrity (McLaughlin, Sheridan, & Lambert, 2014). Two forms of neglect were also assessed: 1) caregiver not meeting basic needs such as providing food or clothing or 2) being left home alone when an adult should have been present before the 6th grade. These two ACEs are clearly deprivation-type and represent the absence of appropriate environmental inputs or stimuli (McLaughlin et al., 2014). Responses fell on a six-point scale ranging from "*this has never happened"* (0) to "*10 or more times"* (5). Responses were dichotomized into "*this never happened"* (0) and "*one or more times"* (1 through 5).

We also created a composite ACEs score based on standardized "z-scores" of each of the four forms of ACEs; we transformed the variables from their original 0–5 ranging "raw" score to reflect z-scores (z-score equals observed value minus sample mean divided by standard deviation or $z = (x-\mu)/\sigma$), and then summed the four z-score transformed variables. The value in standardizing the ACEs before creating a composite score is that positive responses to less common, and potentially more detrimental (Tsuyuki et al., 2019), ACEs have greater value in the composite score.

2.2.2. Follow-up outcome variables: cognitive function

2.2.2.1. Immediate or short-term verbal memory: Verbal memory scores were determined using the Rey Auditory-Verbal Learning Test (RAVLT) at Waves IV and V. To evaluate short-term verbal memory, the interviewer reads a list of 15 common words aloud with 1-second intervals between each word. The participant was then instructed to immediately recall as many of the 15 words as possible within 90 s, or until they indicated that they could not remember any other words. The participant received one point for each correct word recalled, and higher scores indicate better immediate word recall or short-term verbal memory.

2.2.2.2. Delayed or long-term verbal memory: Long-term memory was assessed at Wave IV and V during the home interview using the delayed recall task of the RAVLT. After the first RAVLT list presentation in the immediate recall task, there was a delay after which participants were asked to recall as many of the words from the list as possible within 60 s. The participant received one point for each correct word recalled, with higher scores indicating better delayed word recall or long-term verbal memory.

2.2.2.3. Working memory: Working memory was assessed using a digit-span backwards task at Waves IV and V. The digit-span backwards task is a standardized measure that is utilized to assess working memory in the Weschler Adult Intelligence Scale (WAIS-IV). The task involved an interviewer reading strings of numbers aloud, with 1-second intervals between each number. The participant was then asked to recall the string of numbers in reverse order. The task began with a two-number string and consisted of seven levels. At each level, the participant had two trials to recall the number string backwards correctly. If the correct response was given on the first trial, the second trial of that level was not administered and the interviewer would then move to the number string at the next level. If the participant was unable to accurately recall a number string in both trials, the task was concluded. The possible range of scores was from 0 to 7, where higher scores demonstrate better number recall or working memory.

2.3. Covariates

Our covariates measures were: age, sex, race/ethnicity, household income, baseline alcohol use, baseline cigarette use, baseline depression score, and baseline verbal ability. Age (years), sex (1 = male, 0 = female), and race/ethnicity (white, non-Hispanic as referent group for Black/African American, non-Hispanic; Hispanic/Latino; Asian/Pacific Islander, non-Hispanic; American Indian/Native American; or other race/ethnicity) were based on self-report at Waves I or IV. Household income was based on parents' self-report of household income (US dollars) in the previous calendar year at Wave I. Gaussian normal regression imputation models were used to impute income for the 1638 parents who either refused to answer the income question or stated they did not know. At Wave I, adolescents were asked about alcohol and cigarette use (1 = ever used, 0 = never used). Depression was measured using a modified Center for Disease Epidemiology-Depression scale (CES-D) (Radloff, 1977). Responses to items assessing depressive symptoms are coded on a 4-point scale ranging from *"never or rarely"* (0) to *"most of the time or all of the time"* (3) with a composite score of ≥16 indicative of clinical depression. Our depression score was a dichotomized variable of persons who endorsed clinically significant depression levels (1 = yes ≥ 16, 0 = no < 16). General verbal ability was assessed using a modified version of the Peabody Picture Vocabulary Test at Wave I. In this task, the interviewer reads a word aloud and asks the participant to pick one of the four pictures in front of them that best fits this meaning. The task consisted of 87 items and raw scores were standardized by participant age. This measure was included in our analyses to control for general verbal ability and as a proxy for early IQ in adolescence (Wave I) on verbal memory and working memory in adulthood (Waves IV and V).

2.4. Statistical analyses

Data analysis was performed in 2020 using Stata 15.0 (StataCorp, College Station, TX). Multiple linear regression analyses were used to identify the association between ACEs as predictor variables and cognitive outcomes as the outcome variables, adjusting for age, sex, race/ethnicity, household income, baseline alcohol use, baseline cigarette use, baseline clinical depression score, and baseline verbal ability in each model. Missing data was handled using listwise deletion for each model. Thus, analyses using Wave IV cognitive outcomes included participants with observed data at Waves I, III, and IV (max n =

12,288, min n = 10,458), incorporating sample weighting to yield nationally representative estimates. Analyses using Wave V cognitive outcomes included participants with data at Waves I, III, and V (max n = 1277, min n = 1163). However, national sample weighting was not applied to Wave V cognitive outcome analyses given that only a subset of the cohort was assessed for cognitive outcomes in that wave.

3. Results

The mean age of the sample (n = 12,288) was 28.30 years at Wave IV, and the sample was racially and ethnically diverse (Table 1). Being left alone by a parent or guardian was the most frequently endorsed ACE at 40.82 % while sexual abuse was the most infrequent at 4.74 %. Mean scores for number recall (working memory), immediate word recall (short-term memory), and delayed word recall (long-term memory) were 4.15, 6.20, and 4.64, respectively at Wave IV and 4.20, 6.30, 4.74 at Wave V.

Prospective associations between ACE exposure and Wave IV cognitive outcomes are in Table 2 (max n = 12,288). Results from the adjusted regression analyses showed that history of neglect by not having basic needs met was associated with lower number recall/working memory (β = −0.14, CI_{95} −0.26, −0.01), lower immediate recall/short-term memory (β = −0.29, CI_{95} −0.43, −0.15), and lower delayed word recall/long-term memory scores (β = −0.27, CI_{95} −0.43, −0.12) at 24–32 years old. Sexual abuse was associated with lower immediate word recall/short term memory (β = −0.40, CI_{95} −0.62, −0.17) and lower delayed word recall/long-term memory (β = −0.29, CI_{95} −0.55, −0.03) scores at 24–32 years. With regards to covariates, older age, Black race, and lower baseline verbal ability were consistently related to lower scores across all three of the cognitive scores at 24–32 years in all models (Table 2). Lower income was also associated with lower number recall/working memory and lower immediate word recall/short term memory at 24–32 years (Table 2). Lastly, males showed lower short-term and long-term memory scores at 24–32 years while those with clinically significant depression showed lower short-term memory (Table 2). Of note, because the pattern of significance did not differ between the individual ACE exposures and the standardized ACE score, all covariate coefficients in Table 2 are from the standardized ACE score model.

Prospective associations between ACE exposures and Wave V cognitive outcomes (max n = 1277) are presented in Table 3. Neglect by not having basic needs met predicted lower immediate word recall/short term memory (β = −0.47, CI_{95} −0.79, −0.16) and lower delayed word recall/long-term memory scores (β = −0.33, CI_{95} −0.65, −0.01) at 31–42 years old. A greater number of cumulative ACEs, measured by the standardized ACEs score, predicted lower delayed word recall/long-term memory scores (β = −0.06, CI_{95} −0.10, −0.01) at 31–42 years. With regards to covariates at Wave V, lower baseline verbal ability continued to predicted all three cognitive scores at 31–42 years (Table 3). Being male, Latino, or having clinically significant depression was associated with lower short-term memory scores, whereas being male and/or Black was associated with lower long-term memory scores at 31–42 years old (Table 3). As in Table 2, all covariate coefficients are from the standardized ACE score model.

4. Discussion

In this sample of young adults, our hypothesis that both threat and deprivation-type ACEs would be linked to poorer performance on measures of cognitive performance was supported. Specifically, young adults who endorsed a history of childhood sexual abuse (threat-type ACE) or a history of not having their basic needs met (deprivation-type ACE) at baseline had poorer performance on short-term memory and longer-term memory tasks at 24–32 years old. These effects were detected after adjusting for demographics, household income, substance use, depressive symptoms, and adolescent verbal ability, a proxy for IQ. Such adjustment is clearly important given that older age and lower baseline IQ scores consistently and independently predicted poorer performance on later cognitive tests in our sample.

For the deprivation indicator, these adverse cognitive effects persisted at 31–42 years, showing that a childhood characterized by neglect (i.e., not being clean or having inadequate food or clothing) has lasting impacts on cognitive performance, up to 15–17 years after initially endorsing ACEs. Endorsement of the same neglect indicator was also associated with lower working memory scores at 24–32 years old, indicating that executive function – in addition to memory – may also be impacted by early deprivation of basic resources. Our hypothesis that cumulative ACE exposure would be associated with poorer cognitive function was also partially supported, with longer-term memory scores declining with higher ACEs exposure at 31–42 years old.

The pattern of findings observed is clearly consistent with the ACEs model, which purports that early childhood experiences have lasting effects on neurocognitive function, as well as with other empirical summaries (Carvalho et al., 2016; Irigaray et al., 2013). These results show that cognitive impacts are observable even in a young and dementia-free sample and point to the importance of following this cohort into older age to assess how ACEs are related to risk for onset of major and mild neurocognitive disorders. Additionally, our investigation advances more recent findings that the ACEs-cognition relationship is nuanced in that certain ACE types may have distinct neurobiological and psychosocial effects on cognitive function (Mansueto et al., 2019; Sheridan & McLaughlin, 2014). These distinct patterns of neurocognitive effects for threat vs. deprivation ACEs have even been found for other mental illnesses characterized by cognitive disturbances, such as psychotic disorders (Mansueto et al., 2019). Early deprivation/neglect ACEs, in particular, may be harmful to language development or other crystalized intelligence indicators (Sylvestre, Bussières, & Bouchard, 2016). Crystallized cognitive function is typically defined as knowledge that is acquired over the lifetime and available in long-term memory, so it may not be surprising that a child developing in environments that lack cognitive stimulation, educational opportunities, and/or safety would miss foundational learning opportunities (Rindermann, Flores-Mendoza, & Mansur-Alves, 2010).

Not having basic needs met is an ACE that is clearly related to and often synonymous with low SES, indicators of which have also been linked to poorer cognitive outcomes and academic achievement as well as higher levels of ACEs (Danese et al., 2017; Dunn et al., 2016; Greenfield & Moorman, 2019; Hackman, Farah, & Meaney, 2010; Slopen et

al., 2016). Although studies examining SES typically use parental occupation, education, or income as predictors, these variables likely capture the proximate factors that contribute to such downstream ACE-related outcomes of food insecurity, reduced access to hygienic resources or spaces, and other domains of scarcity. Indeed, our study also showed that other social determinants of health like lower income and marginalized racial identities (i.e., Black and Latino) were also uniquely associated with lower cognitive scores, findings that may be due to the stressful effects of system-level factors like classism, racism, and discrimination (Ozier, Taylor, & Murphy, 2019) as well as bias in neuropsychological scoring norms (Gasquoine, 2009; Norman et al., 2011). Such chronic stress may lead to greater depressive symptoms (Tafet & Bernardini, 2003), which were also associated with poorer cognitive performance in our sample. Together, these findings highlight the role of early social disparities, discrimination, and associated systems and their potentially traumatic effects on later cognitive risk and health. Previous studies on disadvantaged childhood SES suggest that it predicts the initial levels of cognition with which people enter later life but may not necessarily be predictive of cognitive decline (Greenfield & Moorman, 2019). *Post hoc* sensitivity analyses of our results confirmed such a pattern, in that the basic needs indicator did not predict change in cognition from Wave IV to Wave V (i.e., Wave V as dependent variable covarying for Wave IV cognition), despite associations with cognition at both Waves. Such findings highlight the complexities of determining the causal role of ACEs on later cognitive deficits. Some studies have suggested that existing cognitive deficits that predate a child's ACE exposure – combined with the confounding, non-specific effects of childhood SES disadvantage – explain any observed associations between his or her actual ACEs exposure and later cognition (Danese et al., 2017). However, the growing literatures on intergenerational trauma and epigenetic effects (i.e., in which trauma or ACEs in previous generations contribute to physiological alterations in future descendants) imply that it may be difficult to determine a true "time zero" or a baseline assessment period in which a child was completely free of the effects of personal trauma exposure or the ACEs of their parents, grandparents, etcetera (Scorza et al., 2019; Yehuda & Lehrner, 2018). Such data and theories call into question whether some children are "born" with historical trauma exposure and associated cognitive deficits even before they experience their own personal ACE exposure.

These emerging models of epigenetic intergenerational transmission suggest that – in addition to clear environmental inputs to cognitive health – biological pathways may also explain how ACEs promote cognitive deficits or burden, especially in the context of threat ACEs like sexual abuse and forced, early sexual initiation (Tsuyuki et al., 2019). Specifically, the toxic stress may promote neurocognitive harm through the disruption of the body's stress, inflammation, endocrine, cardiovascular, and/or metabolic systems as well as epigenetic alterations of these systems. Higher ACEs have been associated with chronic hypothalamic-pituitary-adrenal axis (HPA) dysregulation (Kalmakis, Meyer, Chiodo, & Leung, 2015), elevated cytokines and other proinflammatory markers (Baumeister, Akhtar, Ciufolini, Pariante, & Mondelli, 2016; Lacey, Pereira, Li, & Danese, 2020), blunted endocrine and cardiovascular reactivity (Voellmin et al., 2015), and obesity (Hawkins et al., 2020; Wiss & Brewerton, 2020). Such physiological sequelae impact major organ systems – including the brain – even among samples that are relatively young and after adjusting

for other risky health behaviors (i.e., substance use) and emotional problems (i.e., depressive symptoms), and a proxy for early life IQ.

Despite several study strengths, including large and diverse samples, longitudinal analyses with extended follow-up, and two ACE types, key limitations should be noted. First, our ACEs measure is based on retrospective self-report and is limited to four exposures (two threat, two neglect). These features mean that 1) we cannot address the idea that prospective and retrospective ACE assessments may identify different groups of individuals (Baldwin, Reuben, Newbury, & Danese, 2019) and 2) that we have captured only a limited array of the traumatic events possible (Finkelhor, Shattuck, Turner, & Hamby, 2013). However, these indicators were chosen to minimize measurement error given that they were all explicitly delivered to assess for ACEs exposure in the same data wave using the same using the same item stem and do broadly capture neglect and threat. Second, cognition function is comprised of multiple domains and was measured using only three indicators and not a full neuropsychological battery with multiple indicators of other cognitive abilities, such as cognitive flexibility, inhibitory control, visuospatial memory, and processing speed, which have also been linked to early life adversity (Danese et al., 2017). Lastly, although we adjusted for several covariates including demographic, socio-economic, behavioral, mental health factors as well as adolescent cognition, there is also the possibility of unmeasured confounders. For instance, measures of race-ethnicity are poor proxies that fail to account for the impact of actual racism and discrimination and other life adversities that may contribute to ACEs exposure.

In brief conclusion, higher ACEs, especially deprivation-type, were prospectively linked to poorer cognition at 24–32 and 31–42 years old. As increasing numbers of individuals face health conditions related to cognition (e.g., major and mild neurocognitive disorders), it is essential to identify the early and modifiable risk factors and treatment targets for poor cognitive health. Clinicians caring for children and adolescents may want to consider routinely screening for ACEs to aid in early identification or prevention of downstream negative health outcomes. Adult providers might consider assessing ACEs to provide more tailored interventions that address potential ACEs-driven deficits in self-regulation that may impact treatment response to ACE-related chronic diseases, such as obesity (Gunstad, Sanborn, & Hawkins, 2020). As a salient example of such intervention and policy efforts, California recently allocated $40 million to implement a statewide effort to screen patients for ACEs in 2020 (Campbell, 2020), and models of effective clinical–community partnerships are emerging in the literature (Atchison, Butler, & Damiano, 2020). While the outcomes of such policies are not yet clear, wide scale screening and tailored treatment methods addressing ACEs and cognitive function may be warranted in order to optimize prevention and intervention efforts for healthy cognitive aging and optimal brain function.

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References

Anda RF, Felitti VJ, Bremner JD, Walker JD, Whitfield C, Perry BD, & Giles WH (2006). The enduring effects of abuse and related adverse experiences in childhood. European Archives of Psychiatry and Clinical Neuroscience, 256(3), 174–186. [PubMed: 16311898]

Atchison CG, Butler J, & Damiano P (2020). Adverse childhood experiences: A model for effective 21st-century clinical–community partnerships. American Journal of Public Health, 110, 450–451. [PubMed: 32159981]

Baldwin JR, Reuben A, Newbury JB, & Danese A (2019). Agreement between prospective and retrospective measures of childhood maltreatment: A systematic review and meta-analysis. JAMA Psychiatry, 76(6), 584–593. [PubMed: 30892562]

Bale TL, Baram TZ, Brown AS, Goldstein JM, Insel TR, McCarthy MM, & Susser ES (2010). Early life programming and neurodevelopmental disorders. Biological Psychiatry, 68(4), 314–319. [PubMed: 20674602]

Baumeister D, Akhtar R, Ciufolini S, Pariante CM, & Mondelli V (2016). Childhood trauma and adulthood inflammation: A meta-analysis of peripheral Creactive protein, interleukin-6 and tumour necrosis factor-α. Molecular Psychiatry, 21(5), 642–649. [PubMed: 26033244]

Berens AE, Jensen SK, & Nelson CA (2017). Biological embedding of childhood adversity: From physiological mechanisms to clinical implications. BMC Medicine, 15(1), 135. [PubMed: 28724431]

Calem M, Bromis K, McGuire P, Morgan C, & Kempton MJ (2017). Meta-analysis of associations between childhood adversity and hippocampus and amygdala volume in non-clinical and general population samples. NeuroImage Clinical, 14, 471–479. [PubMed: 28275547]

Campbell T (2020). Screening for adverse childhood experiences (ACEs) in primary care: A cautionary note. JAMA, 323(23), 2379–2380. [PubMed: 32463425]

Carvalho JC, Donat JC, Brunnet AE, Silva TG, Silva GR, & Kristensen CH (2016). Cognitive, neurobiological and psychopathological alterations associated with child maltreatment: A review of systematic reviews. Child Indicators Research, 9(2), 389–406.

Centers for Disease Control and Prevention. (2020). Adverse childhood experiences resources: The ACE pyramid. Retrieved from https://www.cdc.gov/violenceprevention/images/acestudy/ace_pyramid_lrg.png.

Centers for Disease Control Prevention (CDC) Kaiser Permanente. (2017). The ACE study survey data [Unpublished data]. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention.

Centers for Disease Control Prevention Kaiser Permanente. (2017). The ACE study survey data [Unpublished Data]. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention.

Cohen RA, Grieve S, Hoth KF, Paul RH, Sweet L, Tate D, & Hitsman B (2006). Early life stress and morphometry of the adult anterior cingulate cortex and caudate nuclei. Biological Psychiatry, 59(10), 975–982. [PubMed: 16616722]

Danese A, Moffitt TE, Arseneault L, Bleiberg BA, Dinardo PB, Gandelman SB, & Poulton R (2017). The origins of cognitive deficits in victimized children: implications for neuroscientists and clinicians. The American Journal of Psychiatry, 174(4), 349–361. [PubMed: 27794691]

Dannlowski U, Stuhrmann A, Beutelmann V, Zwanzger P, Lenzen T, Grotegerd D, & Bauer J (2012). Limbic scars: long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. Biological Psychiatry, 71(4), 286–293. [PubMed: 22112927]

Davis MC, Lemery-Chalfant K, Yeung EW, Luecken LJ, Zautra AJ, & Irwin MR (2018). Interleukin-6 and depressive mood symptoms: Mediators of the association between childhood abuse and cognitive performance in middle-aged adults. Annals of Behavioral Medicine, 53(1), 29–38.

Dong M, Anda RF, Dube SR, Giles WH, & Felitti VJ (2003). The relationship of exposure to childhood sexual abuse to other forms of abuse, neglect, and household dysfunction during childhood. Child Abuse & Neglect, 27(6), 625–639. [PubMed: 12818611]

Dube SR, Felitti VJ, Dong M, Chapman DP, Giles WH, & Anda RF (2003). Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: The adverse childhood experiences study. Pediatrics, 111(3), 564–572. [PubMed: 12612237]

Dunn EC, Busso DS, Raffeld MR, Smoller JW, Nelson CA, Doyle AE, … Luk G (2016). Does developmental timing of exposure to child maltreatment predict memory performance in adulthood? Results from a large, population-based sample. Child Abuse & Neglect, 51, 181–191. [PubMed: 26585216]

Finkelhor D, Shattuck A, Turner H, & Hamby S (2013). Improving the adverse childhood experiences study scale. JAMA Pediatrics, 167(1), 70–75. [PubMed: 23403625]

Gasquoine PG (2009). Race-norming of neuropsychological tests. Neuropsychology Review, 19(2), 250–262. 10.1007/s11065-009-9090-5 [PubMed: 19294515]

Greenfield EA, & Moorman SM (2019). Childhood socioeconomic status and later life cognition: Evidence from the Wisconsin Longitudinal Study. Journal of Aging and Health, 31(9), 1589–1615. [PubMed: 29969933]

Gunstad J, Paul RH, Cohen RA, Tate DF, Spitznagel MB, & Gordon E (2007). Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. Comprehensive Psychiatry, 48(1), 57–61. [PubMed: 17145283]

Gunstad J, Sanborn V, & Hawkins M (2020). Cognitive dysfunction is a risk factor for overeating and obesity. American Psychologist, 75(2), 219. [PubMed: 32052996]

Hackman DA, Farah MJ, & Meaney MJ (2010). Socioeconomic status and the brain: Mechanistic insights from human and animal research. Nature Reviews Neuroscience, 11(9), 651–659. [PubMed: 20725096]

Harris KM (2013). The add health study: Design and accomplishments. Chapel Hill: Carolina Population Center, University of North Carolina at Chapel Hill.

Hawkins MAW, Gunstad J, Calvo D, & Spitznagel MB (2016). Higher fasting glucose is associated with poorer cognition among healthy young adults. Health Psychology, 35(2), 199. [PubMed: 26523354]

Hawkins MAW, Ciciolla L, Colaizzi J, Keirns N, Smith C, Stout M, … Erato G (2020). Adverse childhood experiences and cognitive function among adults with excess adiposity. Obesity Science & Practice, 6(1), 47–56. [PubMed: 32128242]

Irigaray TQ, Pacheco JB, Grassi-Oliveira R, Fonseca RP, Leite J. C.d. C, & Kristensen CH (2013). Child maltreatment and later cognitive functioning: A systematic review. Psicologia: Reflexão e Crítica, 26(2), 376–387.

Kalmakis KA, Meyer JS, Chiodo L, & Leung K (2015). Adverse childhood experiences and chronic hypothalamic–pituitary–adrenal activity. Stress, 18(4), 446–450. [PubMed: 25783196]

Lacey RE, Pereira SMP, Li L, & Danese A (2020). Adverse childhood experiences and adult inflammation: Single adversity, cumulative risk and latent class approaches. Brain, Behavior, and Immunity, 87, 820–830. [PubMed: 32201253]

Lemche E (2018). Early life stress and epigenetics in late-onset Alzheimer's dementia: A systematic review. Current Genomics, 19(7), 522–602. [PubMed: 30386171]

Majer M, Nater UM, Lin J-MS, Capuron L, & Reeves WC (2010). Association of childhood trauma with cognitive function in healthy adults: A pilot study. BMC Neurology, 10(1), 61. [PubMed: 20630071]

Mansueto G, Schruers K, Cosci F, van Os J, Alizadeh BZ, Bartels-Velthuis AA, … de Haan L (2019). Childhood adversities and psychotic symptoms: The potential mediating or moderating role of neurocognition and social cognition. Schizophrenia Research, 206, 183–193. [PubMed: 30527930]

McLaughlin KA, Sheridan MA, & Lambert HK (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. Neuroscience and Biobehavioral Reviews, 47, 578–591. [PubMed: 25454359]

Merz EC, & Noble KG (2017). Neural development in context: Differences in neural structure and function associated with adverse childhood experiences. The Wiley Handbook of Early Childhood Development Programs, Practices, and Policies, 135–160.

Murphy A, Steele M, Dube SR, Bate J, Bonuck K, Meissner P, & Steele H (2014). Adverse childhood experiences (ACEs) questionnaire and adult attachment interview (AAI): Implications for parent child relationships. Child Abuse & Neglect, 38(2), 224–233. [PubMed: 24670331]

Nichols E, Szoeke CEI, Vollset SE, Abbasi N, Abd-Allah F, Abdela J, … Murray CJL (2019). Global, regional, and national burden of Alzheimer's disease and other dementias, 1990–2016: A systematic analysis for the Global Burden of Disease Study 2016. The Lancet Neurology, 18(1), 88–106. [PubMed: 30497964]

Norman MA, Moore DJ, Taylor M, Franklin D Jr, Cysique L, Ake C, Lazarretto D, Vaida F Heaton RK, & HNRC Group. (2011). Demographically corrected norms for African Americans and Caucasians on the Hopkins Verbal Learning Test-Revised, Brief Visuospatial Memory Test-Revised, Stroop Color and Word Test, and Wisconsin Card Sorting Test 64-Card Version. Journal of Clinical and Experimental Neuropsychology, 33(7), 793–804. 10.1080/13803395.2011.559157 [PubMed: 21547817]

Ozier EM, Taylor VJ, & Murphy MC (2019). The cognitive effects of experiencing and observing subtle racial discrimination. The Journal of Social Issues, 75(4), 1087–1115.

Radloff LS (1977). The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement, 1(3), 385–401.

Rindermann H, Flores-Mendoza C, & Mansur-Alves M (2010). Reciprocal effects betwee fluid and crystallized intelligence and their dependence on parents' socioeconomic status and education. Learning and Individual Differences, 20(5), 544–548.

Scorza P, Duarte CS, Hipwell AE, Posner J, Ortin A, Canino G, … Program Collaborators for Environmental influences on Child Health Outcomes. (2019). Research Review: Intergenerational transmission of disadvantage: epigenetics and parents' childhoods as the first exposure. Journal of Child Psychology and Psychiatry, 60(2), 119–132. [PubMed: 29473646]

Sheridan MA, & McLaughlin KA (2014). Dimensions of early experience and neural development: Deprivation and threat. Trends in Cognitive Sciences, 18(11), 580–585. [PubMed: 25305194]

Shonkoff JP, Garner AS, Siegel BS, Dobbins MI, Earls MF, McGuinn L, & Care D (2012). The lifelong effects of early childhood adversity and toxic stress. Pediatrics, 129(1), e232–e246. [PubMed: 22201156]

Slopen N, Shonkoff JP, Albert MA, Yoshikawa H, Jacobs A, Stoltz R, … Williams DR (2016). Racial disparities in child adversity in the US: Interactions with family immigration history and income. American Journal of Preventive Medicine, 50(1), 47–56. [PubMed: 26342634]

Sylvestre A, Bussières E-L, & Bouchard C (2016). Language problems among abused and neglected children: A meta-analytic review. Child Maltreatment, 21(1), 47–58. [PubMed: 26620719]

Tafet GE, & Bernardini R (2003). Psychoneuroendocrinological links between chronic stress and depression. Progress in Neuro-psychopharmacology & Biological Psychiatry, 27(6), 893–903. [PubMed: 14499305]

Teicher MH, Tomoda A, & Andersen SL (2006). Neurobiological consequences of early stress and childhood maltreatment: are results from human and animal studies comparable? Annals of the New York Academy of Sciences, 1071(1), 313–323. [PubMed: 16891580]

Tsuyuki K, Al-Alusi NA, Campbell JC, Murry D, Cimino AN, Servin AE, … Stockman JK (2019). Adverse childhood experiences (ACEs) are associated with forced and very early sexual initiation among Black women accessing publicly funded STD clinics in Baltimore, MD. PloS One, 14(5), Article e0216279.

Voellmin A, Winzeler K, Hug E, Wilhelm FH, Schaefer V, Gaab J, & Bader K (2015). Blunted endocrine and cardiovascular reactivity in young healthy women reporting a history of childhood adversity. Psychoneuroendocrinology, 51, 58–67. [PubMed: 25290347]

Wiss DA, & Brewerton TD (2020). Adverse childhood experiences and adult obesity: A systematic review of plausible mechanisms and meta-analysis of crosssectional studies. Physiology & Behavior, 223, Article 112964.

Yehuda R, & Lehrner A (2018). Intergenerational transmission of trauma effects: Putative role of epigenetic mechanisms. World Psychiatry, 17(3), 243–257. [PubMed: 30192087]

Highlights

- Adverse childhood experiences (ACEs) are linked to poorer cognition in early adulthood.
- Both deprivation- and threat-type and higher cumulative ACEs predicted cognition.
- Screening and tailored treatments addressing ACEs and cognition may be warranted.

Table 1.

Descriptive characteristics of participants in the National Longitudinal Study of Adolescent Health.

	Mean ± SE or %[a]
Demographic characteristics (Wave IV, 24–32 years)	
Age, years	28.30 ± 0.12
Race/ethnicity	
White (non-Hispanic)	68.20 %
Black/African American (non-Hispanic)	15.52 %
Hispanic/Latino	11.92 %
Asian/Pacific Islander (non-Hispanic)	3.10 %
American Indian/Native American	0.50 %
Other race/ethnicity	0.76 %
Household income, US dollars (Wave I, 12–18 years)	45,974 ± 1675
Baseline substance use, mental health, and proxy for general IQ (Wave I, 12–18 years)	
Alcohol, ever use	55.57 %
Cigarette, ever use	57.71 %
Depression score (CES-D score ≥ 16)	50.05 %
Baseline verbal ability; IQ proxy (Peabody Picture Vocabulary Test)	101.59 ± 0.61
Self-reported adverse childhood experiences (Wave III, 18–24 years)[b]	
Sexual abuse	4.74 %
Physical abuse	17.40 %
Neglect: left alone by parent/guardian	40.82 %
Neglect: basic needs not met	11.79 %
Cognitive outcomes (Assessed at Wave IV, 24–32 years)[c]	
Number recall (working memory)	4.13 ± 0.04
Immediate word recall (short-term memory)	6.61 ± 0.06
Delayed word recall (long-term memory)	5.19 ± 0.06
Cognitive outcomes (Assessed at Wave V, 32–42 years)[d]	
Number recall (working memory)	4.20 ± 0.04
Immediate word recall (short-term memory)	6.30 ± 0.06
Delayed word recall (long-term memory)	4.74 ± 0.06

Note. Data is from the National Longitudinal Study of Adolescent to Adult Health (Add Health) Waves I, III, IV, & V.

[a]All means and percentages are calculated with weighted data to reflect the representative proportion in the target U.S. population.

[b]Represents the percentage of those who have ever experienced the adverse childhood experience.

[c]max n = 12,288.

[d]max n = 1,277.

Table 2.

Prospective associations between adverse childhood experiences and Wave IV cognitive outcomes in adults 24-32 years of age, adjusted for covariates.

	Number recall (working memory)	Immediate word recall (short-term memory)	Delayed word recall (long-term memory)
Self-reported adverse childhood experiences; ACEs (Wave III)	**β (95% CI)[a]**	**β (95% CI)[a]**	**β (95% CI)[a]**
Sexual abuse[b]	−0.08 (−0.27–0.11)	−0.40 (−0.62–−0.17) **	−0.29 (−0.55 – −0.03) *
Physical abuse[b]	0.07 (−0.03 – 0.17)	0.10 (−0.003 – 0.20)	0.07 (−0.07 – 0.20)
Neglect: left alone by parent/ guardian[b]	−0.05 (−0.12–0.03)	−0.01 (−0.17–0.15)	−0.04(−0.16 – 0.09)
Neglect: basic needs not met[b]	−0.14 (−0.26–−0.01) *	−0.29 (−0.43 – −0.15) ***	•0.27 (−0.43–−0.12) **
Standardized adverse childhood experiences score	0.00 (−0.02 – 0.02)	−0.02 (−0.04 – 0.01)	−0.01 (−0.04 – 0.01)
Covariates[c]			
Age	−0.04 (−0.07 – −0.01) **	−0.05 (−0.09 – 0.01) **	−0.04 (−0.08 – −0.01) *
Male sex	0.04 (−0.04 – 0.11)	−0.56 (−0.72 - −0.42) ***	−0.67 (−0.77 – −0.56) ***
Race with white as referent group			
Black	−0.19 (−0.35 – −0.03) *	−0.36 (−0.53 – −0.19) ***	−0.51 (−0.70 – −0.31) ***
Latino	−0.16 (−0.32 – 0.01)	−0.24 (−0.48 – 0.00)	−0.05 (−0.34 – 0.24)
American Indian/Native American	−0.22 (−0.65 – 0.20)	−0.23 (−0.78 – 0.31)	−0.55 (−1.19 – 0.09)
Asian/Pacific Islander	0.22(0.04 – 0.40) *	0.09 (−0.15 – 0.32)	0.15 (−0.20 – 0.50)
Other race	0.03 (−0.33 – 0.39)	−0.10 (−0.62 – 0.41)	−0.12 (−0.82 – 0.58)
Household income	0.001 (0.00 – 0.002) **	0.002 (0.00 – 0.002) **	0.001 (−0.00 – 0.002)
Baseline alcohol use	0.03 (−0.07 – 0.14)	0.11 (−0.03 – 0.25)	0.01 (−0.13 – 0.14)
Baseline cigarette use	0.04 (−.06 – 0.14)	0.01 (−0.13 – 0.14)	−0.04 (−0.17 – 0.09)
Baseline depression	−0.07 (−0.17 – 0.03)	−0.12 (−0.23 – −0.02) *	−0.08 (−0.18 – −0.01)
Baseline verbal ability	0.03(0.03 – 0.04) ***	0.04(0.03 – 0.04) ***	0.03(0.03 – 0.04) ***

Note. Data is from the National Longitudinal Study of Adolescent to Adult Health (Add Health) Waves I, III, IV, & V

* $p<.05$

** $p<.01$

*** $p<.001$.

[a] Each ACE type and the standardized ACE score were entered into separate models, each adjusted for age, sex, race/ethnicity, household income, baseline alcohol use, baseline cigarette use, baseline depression score, and baseline verbal ability

[b] Reference group has never experienced the adverse childhood experience.

[c] Coefficients for all covariates are from the models using ACFs standardized score given that pattern of significance was the same as for models with the individual ACE types.

Table 3.

Prospective associations between adverse childhood experiences and Wave V cognitive outcomes in adults 31-42 years of age, adjusted for covariates.

	Number recall (working memory)	Immediate word recall (short-term memory)	Delayed word recall (long-term memory)
Self-reported adverse childhood experiences; ACES (Wave III)	**P (95% CI)[a]**	**P(95%CI)[a]**	**P (95% CD[a]**
Sexual abuse[b]	−0.03 (−0.42 – 0.36)	−0.24 (−0.75 – 0.27)	−0.42 (−0.94 – 0.09)
Physical abuse[b]	−0.21 (−0.38 – 0.03)	−0.15 (−0.38 – 0.09)	−0.20 (−0.43 – 0.04)
Neglect: left alone by parent/ guardian[b]	−0.03 (−0.20 – 0.14)	−0.17 (−0.39 – 0.04)	−0.19 (−0.41 – 0.03)
Neglect: basic needs not met[b]	−0.22 (−0.46 – 0.02)	−0.47 (−0.79 – −0.16) **	−0.33 (−0.65–−0.01) *
Standardized ACEs score	−0.02 (−0.06 – 0.01)	−0.02 (−0.07 – 0.02)	−0.06 (−0.10 – −0.01) *
Covariates[c]			
Age	−0.04 (−0.09 – 0.01)	−0.02 (−0.09 – 0.04)	−0.02 (−0.08 – 0.04)
Male sex	−0.10 (−0.27 – 0.07)	−0.54 (−0.76 – −0.32) ***	−0.73 (−0.95 – −0.50) ***
Race with white as referent group			
Black	−0.10 (−0.33 – 0.13)	−0.24 (−0.54 – 0.06)	−0.70 (−1.00 – −0.40) ***
Latino	0.00 (−0.26 – 0.27)	−0.47 (−0.82 – −0.12) **	−0.23 (−0.58 – 0.12)
American Indian/Native American	0.38 (−0.46 – 1.23)	0.23 (−0.87 – 1.32)	0.44 (−0.66 – 133)
Asian/Pacific Islander	0.27 (−0.13 – 0.67)	0.13 (−0.40 – 0.65)	0.05 (−0.47 — 0.57)
Other race	−0.31 (−1.15 – 0.54)	0.35 (−0.75 – 1.46)	−0.55 (−1.65 – 0.55)
Household income	0.00 (−0.00 – −0.00)	0.00 (−0.00 – −0.00)	0.00 (−0.00 – −0.00)
Baseline alcohol use	0.05 (−0.15 – 0.26)	−0.07 (−0.33 – 0.20)	0.08 (−0.18 – 035)
Baseline cigarette use	0.05 (−0.15 – 0.25)	−0.01 (−0.27 – 0.25)	−0.06 (−0.32 – 0.20)
Baseline depression	−0.20 (−0.38 – −0.02) *	−0.26 (−0.49 – −0.02) *	−0.23 (−0.46 – 0.01)
Baseline verbal ability	0.03 (0.03 – 0.04) ***	0.03 (0.02 – 0.04) ***	0.02 (0.02 – 0.03) ***

Note. Data is from the National Longitudinal Study of Adolescent to Adult Health (Add Health) Waves I, III, IV, & V.

* $p < .05$,

** $p < .01$,

*** $p < .001$.

[a] Each ACE type and the standardized ACE score were entered into separate models, each adjusted for age, sex, race/ethnicity, household income, baseline alcohol use, baseline cigarette use, baseline depression score, and baseline verbal ability.

[b] Reference group has never experienced the adverse childhood experience.

[c] Coefficients for all covariates are from the models using ACEs standardized score given that pattern of significance was the same as for the individual ACE types.