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# Are Adverse Childhood Experiences Associated with Worse Cognitive Function in Older Adults?

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**Abstract**

**Objectives:** Adverse childhood experiences (ACE) are associated with an increased risk for dementia, but this relationship and modifying factors are poorly understood. This study is the first to our knowledge to comprehensively examine the effect of ACE on specific cognitive functions and measures associated with greater risk and resiliency to cognitive decline in independent community-dwelling older adults. **Methods:** Verbal/nonverbal intelligence, verbal memory, visual memory, and executive attention were assessed. Self-report measures examined depression, self-efficacy, and subjective cognitive concerns (SCC). The ACE questionnaire measured childhood experiences of abuse, neglect, and household dysfunction. **Results:** Over 56% of older adults reported an adverse childhood event. ACE scores were negatively associated with income and years of education and positively associated with depressive symptoms and SCC. ACE scores were a significant predictor of intellectual function and executive attention; however, these relationships were no longer significant after adjusting for education. Follow-up analyses using the PROCESS macro revealed that relationships among higher ACE scores with intellectual function and executive attention were mediated by education. **Conclusions:** Greater childhood adversity may increase vulnerability for cognitive impairment by impacting early education, socioeconomic status, and mental health. These findings have clinical implications for enhancing levels of cognitive reserve and addressing modifiable risk factors to prevent or attenuate cognitive decline in older adults.

**Keywords:** Abuse, neglect, cognitive reserve, education, modifiable risk factors, late-life

Considerable research demonstrates that adverse childhood experiences (ACE) negatively impact physiological, behavioral, and emotional health well into adulthood (Danese et al., 2009; Dube, Felitti, Dong, Giles, & Anda, 2003; Felitti et al., 1998). Mounting evidence suggests ACE may also have long-lasting negative effects on cognition (Geoffroy, Pereira, Li, & Power, 2016; Gould et al., 2012; Hart & Rubia, 2012; Lupien, McEwen, Gunnar, & Heim, 2009). Developmental research provides support that pathophysiological changes, particularly hypothalamic-pituitary-adrenal (HPA) axis dysregulation and markers of allostatic load, can adversely impact the development and structural integrity of the brain (Colich, Rosen, Williams, & McLaughlin, 2020; Hart & Rubia, 2012; Luecken, 2006; Lupien et al., 2009; Teicher, Samson, Anderson, & Ohashi, 2016). Studies also implicate mental health factors (Danese et al., 2009) and hindrances to educational attainment (Almquist & Brännström, 2018; Lansford et al., 2002; Merrick et al., 2019) as routes by which childhood adversity can weaken cognitive and intellectual function. However, despite a large body of research in children and adults, there is limited research regarding the long-term effects of early-life adversity in older adults.

Several direct and indirect routes link childhood adversity with cognitive dysfunction. Directly, adversity can lead to changes in brain structure and function (Kim & Diamond, 2002). Namely, HPA axis dysregulation stimulates the adrenal glands to overproduce glucocorticoids which cross the blood-brain barrier and bind to neuronal receptors. Prolonged exposure to glucocorticoids is thought to cause a neuronal loss in specific brain regions (Lupien et al., 2009), which may lead to volumetric reductions in the hippocampus, anterior cingulate cortex, ventromedial prefrontal cortex (PFC), dorsomedial PFC, and parietal subregions, as well as volumetric increases in the amygdala across age groups (Ancelin et al., 2021; Hart & Rubia, 2012; Lupien et al., 2009; Teicher et al., 2016). In addition, cortical thinning in areas such as the ventromedial PFC, frontoparietal, default,

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and visual networks, can also exist in children and adolescents with a history of adversity (Colich et al., 2020). Taken together, maltreatment may lead to disruptions in the development of brain areas, particularly the prefrontal cortex and hippocampus (Petchtel & Pizzagalli, 2011). These regions are also sensitive to cognitive aging processes (Salthouse, 2010). Thus, it is plausible that older adults with ACE may experience a "double hit" for the risk of cognitive decline.

Indirectly, links between ACE and cognitive dysfunction may occur through socioeconomic disadvantages, decreased opportunities for educational attainment, and increased risk for psychopathology (Almquist and Brännström 2018; McLaughlin, DeCross, Jovanovic, & Tottenham, 2019; Merrick et al., 2019; Turecki, Ota, Belangero, Jackowski, & Kaufman, 2014). Large cohort studies suggest a dose-dependent relationship between the number of adverse events and socioeconomic status (SES) in adulthood (Font & Maguire-Jack, 2016; Merrick et al., 2019). This is a problematic link given the importance of higher educational attainment in bolstering greater levels of cognitive reserve, thereby reducing the risk for dementia (Stern, 2006). In addition, consistent associations exist between psychopathology and early adversity. Children raised in aversive households are more likely to experience emotion regulation difficulties which may later persist into dispositional negative affect (Taylor, Way, & Seeman, 2011). These vulnerabilities may help explain the increased prevalence of depression across the life span in those with high ACE (Danese et al., 2009; Rao et al., 2010). Moreover, depression may mediate the relationship between maltreatment and later cognitive dysfunction in adults (Toyoshima et al., 2020). Within older adult populations, there is a high correlation between depression and subjective cognitive concerns (SCC; Jessen et al., 2014). Together, these factors may heighten the risk for cognitive impairment and decline (Mendonça, Alves, & Bugalho, 2016; Rabin, Smart, & Amariglio, 2017). Furthermore, resiliency factors such as mastery, self-esteem, and problem-solving abilities tend to be less prevalent in individuals with ACE (Taylor et al., 2011), which may negatively impact the building of cognitive reserve (Boyle et al., 2012).

Throughout childhood and adulthood, cross-sectional evidence suggests worse performance on measures of attention, working memory, executive function, and processing speed among individuals with a history of early adversity (Geoffroy et al., 2016; Gould et al., 2012; Majer, Nater, Lin, Capuron, & Reeves, 2010; Nikulina & Widom, 2013; Nolin & Ethier 2007; Sheridan, Peverill, Finn, & McLaughlin, 2017; Vasilevski & Tucker, 2016). Steeper rates of cognitive decline have been observed in older adults with both adverse childhoods and current depressive symptomology (Korten, Penninx, Pot, Deeg, & Comijs, 2014). On a global cognitive screener measure, there are suggestions of specific effects of ACE on memory but not on other cognitive functions in older adults (Kobayashi et al., 2020). However, others have found that early traumatic events are associated with worse performance on measures of processing speed, attention, and executive functioning in anxious and depressed older adults (Petkus et al., 2018). Ritchie and colleagues (2011) comprehensively investigated cognitive function with specific types of abuse and found that poor environmental conditions are associated with worse verbal retrieval and visuospatial memory; however, there was evidence of better performance on measures of verbal fluency. Similarly, others have found an association between better word retrieval performance and childhood sexual abuse (Feeney, Kamiya, Robertson, & Kenny, 2013). There is also evidence that adults and older adults with childhood adversity demonstrate relatively similar cognitive abilities and rates of decline (Barnes et al., 2012; Dunn et al., 2016).

Finally, ACE may also impact intellectual development in children (Hart & Rubia, 2012), which could influence cognitive reserve. However, this relationship is inconsistent in adolescent and older adult populations (Ritchie et al., 2011; Vasilevski & Tucker, 2016).

ACE are associated with an increased risk for dementia, but this relationship and modifying factors are poorly understood. To address gaps in the literature, this study is the first to our knowledge to comprehensively examine the effect of ACE on specific cognitive functions and measures associated with greater risk and resiliency to cognitive decline in independent community-dwelling older adults. We aimed to determine the effect of ACE on cognitive function in community-dwelling older adults while adjusting for relevant demographic variables of age and education. We hypothesized ACE would associate with worse performance on measures of intellectual function, memory, and executive attention. We also examined interrelationships among ACE, depression, SES, SCC, and self-efficacy, as these factors have been implicated in both early life adversity and cognitive deficits. Follow-up analyses tested whether education mediated the relationship between specific cognitive functions and ACE.

## METHOD

### Participants

Participants were recruited as part of the Maine-Aging Behavior Learning Enrichment (M-ABLE) Study at the University of Maine. This study used community-based participatory research (CBPR) methods to enhance the recruitment of a socioeconomically diverse community-dwelling older adult sample. To reduce participation barriers, study visits occurred at easily accessible locations within the community and were offered on the weekends. The present study was interested in obtaining a representative sample of independent community-dwelling older adults with a range of cognitive functions (normal to MCI). Study inclusion criteria were intentionally broad to improve the generalizability of findings to more diverse older adults. Inclusion criteria included: ages 55–90 years old, willing to undergo neuropsychological assessment, and willingness to provide income information. Exclusion criteria included moderate

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to severe cognitive impairment (Montreal Cognitive Assessment scores < 18; Nasreddine et al., 2005), severe depression (Geriatric Depression Scale scores > 10; Sheikh & Yesavage, 1986), moderate to severe neurological impairments (e.g., moderate-to-severe traumatic brain injury), recent stroke (defined as in the past year), neurodegenerative disorder (e.g., Parkinson's disease or Alzheimer's disease), or physical limitations (e.g., loss of visual field or unable to hold a pencil) that prohibited cognitive testing. Intellectual disability, dementia disorder, or any untreated or severe psychiatric conditions (e.g., psychotic disorders) were also excluded. Participants were compensated up to 70 U.S. dollars for the completion of the study. To attenuate time of day effects on cognitive performance, most appointments were scheduled in the morning, starting between 8:00 a.m. and 9:00 a.m.

## Ethics of human subjects

Participants were screened for eligibility and underwent informed consent procedures approved by the University of Maine Institutional Review Board. All procedures performed in this study were in accordance with the ethical standards of the 1964 Helsinki Declaration and its later amendments. Trained research assistants administered neuropsychological assessments.

## Measures

### *Clinical characteristics*

Participants provided demographic information (age, sex, years of education, income level) and clinical/medical history via interview. Clinical/medical history was collected via the National Alzheimer's Coordinating Center (NACC) Uniform Data Set (UDS) Subject Health History form (Form A5; Morris et al., 2006). This form measures various medical conditions that participants endorse as either "absent," "actively present," "remotely present," or "unknown." Current depressive status was determined by the "active" depression variable (endorsing a major depressive episode within the past two years). Other psychiatric disorders (e.g., generalized anxiety disorder, post-traumatic stress disorder) were represented by the endorsement of an "active" response on the psychiatric disorder variable. A cardiovascular risk composite score was created by summing the variables of cardiac arrest, atrial fibrillation, congestive heart failure, angina, and other evidence of coronary disease. The cerebrovascular risk composite score represented the sum of the stroke, transient ischemic attack, or evidence of other cerebrovascular disease variables.

Anthropometrics including waist circumference (centimeters), height (inches), and weight (pounds) were also collected. Body mass index (BMI) was derived with a formula to convert English to metric measurements. Sex-specific waist circumference cutoff measurements were used in conjunction with BMI to form relative disease risk (RDR) categories (National Heart, Lung, Blood Institute, National Institute of Diabetes, Digestive, & Kidney Diseases, 2013). Participants with a BMI in the obese or overweight range were classified as high RDR, while those within normal-to-underweight ranges were classified as normal RDR based on these guidelines.

### *Measures of psychological risk and resilience*

The ACE questionnaire retrospectively collects information on experiences of abuse, neglect, and household dysfunction occurring in one's life before the age of 18 (Felitti et al., 1998). The ACE is a dichotomous (*yes* or *no*) ten-question inventory, composed of 17 sub-items with demonstrated good to excellent reliability (Felitti et al., 1998). Total scores range from 0 to 10, with higher scores indicating more adverse events. Domains include physical abuse, sexual abuse, neglect, and household dysfunction. Questions of physical and sexual abuse assess the nature of the abuse, neglect questions ascertain the degree of emotional and physical neglect, while household dysfunction questions determine the presence of substance abuse, marital discord, and family mental illness. Risk is commonly approximated by the total ACE score, considering most individuals exposed to one type of adversity are also exposed to a second type of adversity (Felitti et al., 1998). Therefore, the current study used the continuous variable of the total ACE score as a measure of abuse.

The National Institute of Health-Toolbox for the Assessment of Neurological and Behavioral Function Quality of Life in Neurological Disorders (NIH-TB Neuro-QOL, Version 2.0) Cognitive Function, Depression, and Self-Efficacy self-report questionnaires were administered to assess modifiable risk factors associated with risk and resiliency for cognitive decline (Gershon et al., 2013). The 18-item Cognitive Function questionnaire evaluated SCC within the past seven days (National Institute of Neurological Disorders and Stroke [NINDS], 2015). Participants rated perceived levels of difficulty in memory, attention, decision-making, and everyday functions (e.g., reading, planning tasks, or remembering names) according to a 5-point scale ranging from 1 (*cannot do*) to 5 (*none/no difficulty*). Total scores represent the sum of all items and range from 18 to 90, with lower scores indicating greater SCC. Scale reliability in the current sample was excellent ($\alpha = .905$). The Depression questionnaire is a 24-item scale measuring how often participants experience depressive symptoms (National Institute of Neurological Disorders and Stroke (NINDS), 2015). Each of the 24 items is rated on a 5-point scale, ranging from 1 (*never*) to 5 (*always*). The ratings are summed, yielding a total score ranging from 24 to 120, in which higher scores reflect greater depressive symptomatology. Participants selected responses that best described their mood, thoughts, and behaviors during the past seven days. Scale reliability in the current sample was excellent ($\alpha = .946$). The General Self-Efficacy scale (National Institute of Neurological Disorders and Stroke (NINDS), 2015) contains four items that evaluate confidence in the

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ability to problem-solve and handle unexpected situations, ranging from 1 (*I am not confident at all*) to 5 (*I am very confident*). Summed ratings yield a total score ranging from 4 to 20, where higher scores indicate higher confidence levels regarding perceived self-efficacy. Scale reliability for the current sample was excellent ($\alpha = .904$).

### *Neuropsychological assessment*

*Intellectual function.* The 70-item noncontextual word reading test, a subtest of the Wide Range Achievement Test-Fourth Edition (WRAT-4), served as a measure of verbal intellectual functioning (Wilkinson & Robertson, 2006). The Peabody Picture Vocabulary Test—Fourth Edition (PPVT-4), a measure of receptive vocabulary, and the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV) Block Design subtest, a measure of visuospatial construction, were used to assess nonverbal intelligence (Dunn & Dunn, 2007; Wechsler, 2008).

*Verbal memory.* The Rey Auditory Verbal Learning Task (RAVLT) served as a measure of episodic memory and verbal learning (Schmidt, 1996). The RAVLT is composed of two 15-item word lists with five successive recall trials of List A (Trials 1–5), an interference trial (List B), and 20-minute delayed recall and recognition trials. The sum of scores from Trial 1 through Trial 5 reflected immediate recall, while the total number of words freely recalled 30 min after initial registration of the word list represented delayed recall.

*Visual memory.* The Brief Visuospatial Memory Test—Revised (BVMT-R) measured visuospatial learning and memory (Benedict, 1997). The task contains three learning trials and a 25-minute delayed recall trial. The immediate memory score reflected the sum of the learning trials, while the delayed memory score represented the amount of information remembered after a 25-minute delay.

*Executive attention.* The Trail Making Test B (TMT-B; Reitan, 1958) total time in seconds assessed set-shifting ability. The WAIS-IV Digit Span task total score examined auditory working memory. Auditory digit sequences were repeated in forward, reverse, and numeric order, and the sum of these three tasks represented the total score (Wechsler, 2008). The WAIS-IV Coding subtest and the total seconds on the Trail Making Test A (TMT-A) served as estimates of visual scanning and rote attention ability (Reitan, 1958; Wechsler, 2008). TMT-A and B were appropriately scaled such that larger numbers reflected slower (worse) performance, and smaller numbers reflected faster (better) performance.

## Analyses

Descriptive statistics were generated for demographic variables, neuropsychological test scores, and ACE scores to determine whether assumptions of normality were violated and to obtain characteristics of the sample. Data were visually inspected and examined for outliers, skew, and kurtosis. Significant outliers ($z$-scores exceeding ± 3.29 standard deviations from the mean) were winsorized. Neuropsychological variable raw scores were converted into normally distributed scaled scores via Rankit's method (Soloman & Sawilowsky, 2009). Ranked scores were transformed into normally distributed z-scores. Scores were then converted to scaled scores with a mean of ten and a standard deviation of three.

Tests chosen for the composite tests were selected based on prior theoretical framework regarding gold standard measures for assessing cognitive domains, and appropriate composite item reliability (Heaton et al. 2014, Scott, Sorrell, & Benitez, 2019; Weintraub et al., 2014). An Intellectual Function composite was created by forming an average of the standardized and scaled scores of the WRAT-4, PPVT-4, and WAIS-IV Block Design tests and was again transformed and rescaled to have a mean of ten and a standard deviation of three. This procedure was repeated to form Verbal Memory (average of RAVLT immediate recall and delayed recall scores), Visual Memory (average of BVMT-R immediate memory and delayed memory scores), and Executive Attention (average of TMT-A, TMT-B, WAIS-IV Coding, and WAIS-IV Digit Span Total scores) composites. Cronbach's alpha assessed the internal consistencies of each composite. Scale reliability of the Intellectual Function ($\alpha = .772$), Verbal Memory ($\alpha = .863$), Visual Memory ($\alpha = .945$) and Executive Attention ($\alpha = .751$) composites ranged from acceptable to excellent and were deemed adequate for study aims.

The composites served as dependent variables in a series of hierarchical regressions to assess the unique contribution of ACE scores on domains of intellectual function, verbal memory, visual memory, and executive attention while adjusting for relevant demographic variables of age and education. Adjusted $R^2$ and standardized beta values are reported for the final regression models. Spearman's rank correlations explored relationships among ACE scores and clinical, demographic, and psychological measures. Mediation analyses were performed using Hayes' PROCESS macro for SPSS (Hayes, 2017), posited to be a more advanced approach than traditional causal steps analyses (e.g., Baron & Kenny, 1986). The PROCESS approach is a regression-based method that allows for the simultaneous evaluation of the direct and indirect effects, as well as bootstrapped 95% Confidence Intervals for these estimates that serve as measures of effect size (Hayes, 2017). Bonferroni corrections (.05/4 = .0125) were applied to regression and mediation analyses to account for multiple comparisons.

Statistical power was computed a priori using G * Power 3.1 and was sufficient to test a linear multiple regression using three predictors (Faul, Erdfelder, Buchner, & Lang, 2009). Pearson $r$ values served as effect sizes for correlations; $r$ values of .10, .30, and .50 corresponded to small, medium, and large effect sizes, respectively (Cohen, 1988). For hierarchical regressions, $f^2$ values of .02, .15, and .35, represented small, medium, and large effect sizes, respectively (Cohen,

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**Table 1.** Clinical and demographic characteristics of sample

| *Variables* [range] | Total ($N = 121$) |
|---|---|
| Age [57–87 years] | 70.69 (6.47) |
| Education [10–20 years] | 15.63 (2.69) |
| % Female | 73.60 ($n$=89) |
| MoCA [18–30] | 26.21 (2.61) |
| GDS [0–6] | 1.04 (1.36) |
| ACE [0–8], *Mdn* = 1.0 | 1.49 (1.95) |
| BMI [15.2–47.5] | 29.08 (6.29) |
| WC, cm [69–137] | 101.19 (15.30) |
| RDR | 61.20% ($n = 74$) |
| *Risk factors* | |
| Depression | 13.20% ($n = 16$) |
| Diabetes | 6.60% ($n = 8$) |
| Hyperlipidemia | 43.80% ($n = 53$) |
| Hypertension | 45.50% ($n = 55$) |
| Cardiovascular | 23.10% ($n = 28$) |
| Cerebrovascular | 4.10% ($n = 5$) |

1988). All tests of significance were two-tailed. Statistical analyses were performed using SPSS Version 27.

## RESULTS

### Descriptive statistics on demographics and clinical characteristics

Table 1 presents descriptive statistics for the demographic and clinical characteristics of the sample. There was a larger proportion of women, as well as a broad range of years of education and annual income level (overall range: <$10,000–>$100,00, *Mdn:* $50,000–$59,000). Global cognition scores fell within the mild cognitively impaired to normal cognition ranges. Overall, depression scores were in the normal range. Hypertension and hyperlipidemia were the most endorsed vascular risk factors. Average BMI was in the overweight range, and over half of the sample fell in the High RDR category when considering the relative contributions of waist circumference and BMI.

### Adverse childhood experiences

Table 2 presents descriptive information on the prevalence of ACE events. Over 56% of participants experienced at least one adverse childhood event. Men and women did not statistically differ in ACE scores, $F$ (1, 119) = .18, $p$ = .670. Similarly, there were no sex differences in type of abuse, as men and women experienced relatively equal levels of physical/verbal abuse [$\chi^2$ (1, $N = 121$) = .33, $p$ = .568], sexual abuse [$\chi^2$ (1, $N = 121$) = 1.51, $p$ = .219], neglect [$\chi^2$ (1, $N = 121$) = 2.08, $p$ = .149], and household dysfunction, $\chi^2$ (1, $N = 121$) = 2.78, $p$ = .095. Household dysfunction represented the most endorsed domain, followed by physical abuse/verbal abuse. Parental substance abuse was the most endorsed type of adverse event.

**Table 2.** Frequency of ACE responses by categories and items

| ACE categories ($N = 121$) | Frequency % ($n$) |
|---|---|
| **Physical abuse/verbal abuse** | **28.90($n = 35$)** |
| 1. Swear, insulted, and/or put down | 27.30 ($n = 33$) |
| 2. Pushed, grabbed, slapped, hit so hard you were injured | 16.50 ($n = 20$) |
| **Sexual abuse** | **12.40 ($n$ = 15)** |
| 1. Touch/fondled you or had you touch/fondle them in a sexual way; attempted/had oral, anal, or vaginal intercourse with you | |
| **Neglect** | **21.50 ($n$ = 26)** |
| 1. Felt unloved, unimportant, unsupervised, and/or unsupported by family | 17.40 ($n = 21$) |
| 2. Did not have medical care when needed, enough to eat, wore dirty clothes, had no one to protect you; parents were too drunk or high to take care of you | 7.40 ($n = 9$) |
| **Household dysfunction** | **43.80 ($n$ = 53)** |
| 1. Parents were separated or divorced | 19.0 ($n = 23$) |
| 2. Mother or stepmother often physically abused or threatened with violence/weapon | 4.90 ($n = 6$) |
| 3. Lived with someone abusing substances | 23.90 ($n = 29$) |
| 4. Household member depressed, mentally ill, or attempted suicide | 19.80 ($n = 24$) |
| 5. Household member went to prison | 0.80 ($n = 1$) |
| **Total ACE score** | |
| 0 | 43.80 ($n = 53$) |
| 1 | 20.70 ($n = 25$) |
| 2 | 14.00 ($n = 17$) |
| 3 | 5.80 ($n = 7$) |
| ≥4 | 15.70 ($n = 19$) |

### Neuropsychological performance

A series of hierarchical regression analyses were used to determine whether the number of ACE events associated with verbal/nonverbal intelligence, verbal memory, visual memory, and executive attention while adjusting for relevant age and education differences in neuropsychological test scores in the models. A rudimentary model was performed to test whether ACE was associated with intellectual function, visual/verbal memory, and executive function while adjusting only for age. These results revealed that ACE score was a significant predictor of intellectual function ($\beta = -.24$, $p = .010$) executive attention ($\beta = -.21$, $p = .012$), and a trend towards visual memory ($\beta = -.17$, $p = .058$). ACE scores were not significantly associated with verbal memory ($\beta = -.07$, $p = .449$). Intellectual function and executive attention results remained significant after Bonferroni correction; however, the visual memory trend dissipated. Analyses next adjusted for both age and education in the model. Results indicated ACE scores were no longer a significant predictor of intellectual function or executive attention ($ps > .05$) once the significant effect of education was adjusted for in the model (Table 3).

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**Table 3.** Summaries of hierarchical regression analyses

| Composite | $R$ | $R^2$ | Adjusted $R^2$ | $R^2$ change | $F$-change | $p$-value | $f^2$ |
|---|---|---|---|---|---|---|---|
| Premorbid intellectual function | .58 | .34 | .32 | <.001 | .01 | .927 | .52 |
| Verbal memory | .40 | .16 | .14 | .00 | .17 | .684 | .19 |
| Visual memory | .43 | .18 | .16 | .01 | .68 | .410 | .22 |
| Executive attention | .52 | .27 | .25 | .01 | 1.58 | .211 | .37 |

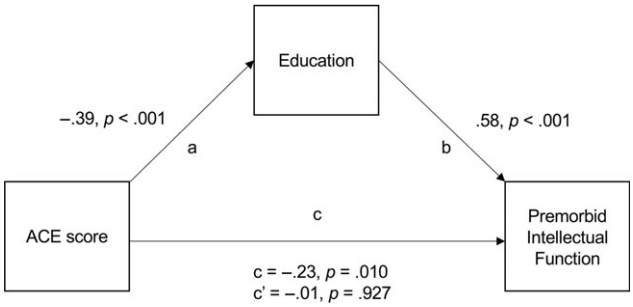


**Fig. 1.** Education mediates the relationship adverse childhood experiences and premorbid intellectual function. Higher ACE scores were significantly associated with less years of education (path a) and lower premorbid intellectual function (path c). However, this relationship was no longer significant (path c') once adjusting for the significant effect of years of education on premorbid intellectual function (path b).

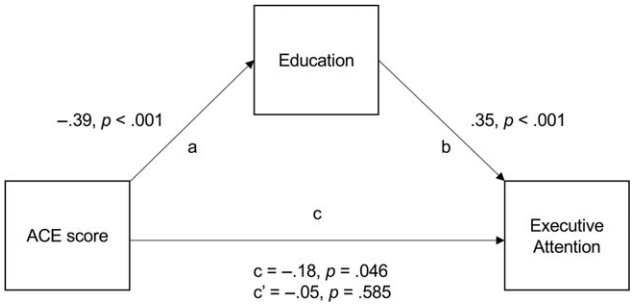


**Fig. 2.** Education mediates the relationship adverse childhood experiences and executive attention. Higher ACE scores were significantly associated with less years of education (path a) and lower executive attention (path c). However, this relationship was no longer significant (path c') once adjusting for the significant effect of years of education on executive attention (path b).

To explore the possibility of a mediating role of education between ACE and cognitive abilities, a mediation path analysis was performed using the PROCESS macro-Version 3 (Hayes, 2017). Figures 1 and 2 present the conceptual relationships. As previously indicated, higher ACE scores predicted lower levels of education (path a: $\beta = -.56$, $p < .001$) within both models. These results indicated approximately 16.30% of the variance in education was explained by ACE scores ($\beta = -.56$, 95% CI [−.79, −.32], $p < .001$). There was a direct effect of higher ACE scores on lower levels of intellectual function (path c: $\beta = -.10$, $p = .010$). However, when the significant effect of education on intellectual function (path b: $\beta = .18$, $p < .001$) was included in the model, the relationship between ACE score and intellectual function (path c': $\beta = -.003$, $p = .927$) was no longer significant. These results provide support that education mediated the relationship between ACE scores and intellectual function ($\beta = -.10$, 95% CI [−.14, −.06]; this indirect effect accounted for approximately 23.22% of the variance in intellectual function. A direct effect of ACE scores was also found on executive attention (path c: $\beta = -.06$, $p = .046$) when education was not adjusted for in the model. However, when the significant effect of education on executive attention (path b: $\beta = .08$, $p < .001$) was included in the model, the relationship between ACE score and executive attention (path c': $\beta = -.02$, $p = .585$) was no longer significant. These results provide support that education mediated the relationship between ACE scores and executive attention ($\beta = -.05$, 95% CI [−.07, −.02]; this indirect effect accounted for approximately 12.50% of the variance in executive attention. While the direct effect of ACE on intellectual function survived Bonferroni correction, the direct effect of ACE on executive attention did not.

## Relationships among ACE with risk/resilience factors

Table 4 presents the interrelationships among ACE and risk/resilience factors. Higher ACE scores were significantly associated with fewer years of education, lower annual income, and higher levels of SCC and depressive symptoms (Table 4). Higher levels of depression symptoms were associated with fewer years of education, lower annual income level, less sense of self-efficacy, and higher levels of SCC. Higher perceptions of self-efficacy were associated with fewer SCC.

## DISCUSSION

The present study is the first to our knowledge to comprehensively examine the effect of ACE on specific cognitive functions and measures associated with greater risk and resiliency to cognitive decline in independent community-dwelling older adults. ACE scores were significantly associated with lower intellectual function and worse executive attention, yielding large effect sizes. Subsequent analyses revealed that these relationships were fully mediated by years of education. However, after adjusting for multiple comparisons, the direct effect of ACE on executive attention was no longer

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**Table 4.** Associations among total ACE scores, risk and resilience factors

| Measure | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| 1. ACE | – | | | | | |
| 2. Education | −.359** | – | | | | |
| 3. Income | −.229* | .373** | – | | | |
| 4. Depression[1] | .274** | −.191* | −.264** | – | | |
| 5. SCC[2] | −.256** | .140 | −.247* | −.408** | – | |
| 6. Self-Efficacy[3] | −.112 | .094 | .088 | −.355** | .370** | – |

*Note.* [1]NeuroQOL (Version 2.0) Depression Questionnaire; [2]Higher scores reflect less subjective cognitive concern (SCC); [3]Patient Reported Outcomes Measurement Information System (PROMIS) Self-Efficacy Scale; *p<.05; **p<.001.

significant. Results are thus interpreted within the context of large effect sizes and limitations of the conservative nature of Bonferroni corrections (i.e., risk of increased type II errors; Perneger, 1998). Significant associations, with small to medium effect sizes, also emerged among ACE with lower SES, greater depression symptoms, and greater SCC. Taken together, childhood adversity may serve as a distal basis by which risk factors build and interact to influence the risk of cognitive decline.

## SES and ACE

A potential route by which ACE may increase the risk of dementia is through lower levels of early education, thereby hindering development for greater cognitive reserve. This is in line with a 15-year longitudinal study suggesting educational disparities have the most prominent influence on functional limitations and negative health outcomes during early to middle old age (House, Lantz, & Herd, 2005). Relatedly, longitudinal evidence demonstrates that educational attainment is a powerful mediator between early adversity and various poor social, economic, and health-related outcomes (Almquist & Brännström, 2018). Even when adjusting for relevant family and child characteristics, evidence indicates that school attendance rate and perceptions regarding the likelihood of attending college are negatively impacted by childhood maltreatment (Lansford et al., 2002).

Theoretical models suggest that children raised in environments of deprivation, characterized by lack of cognitive stimulation or opportunities for learning, may exhibit worse attention and executive function abilities (McLaughlin & Sheridan, 2016; Sheridan et al., 2017). Further, research suggests that higher educational attainment is an important socioeconomic contributor towards preserving memory function in older adults, while low annual income is associated with risk for cognitive decline (Marden, Tchetgen Tchetgen, Kawachi, & Glymour, 2017). Thus, heightened risk for late-life cognitive dysfunction may exist through lack of opportunities for learning and enrichment in those with early adversity (Marden et al., 2017; McLaughlin & Sheridan, 2016; Sheridan et al., 2017). While inherent genetic vulnerability for lower intellectual abilities may exist (Deary, Spinath, & Bates, 2006), educational attainment appears to play a consistent mediating role between childhood adversity and poor health, social, economic, and cognitive outcomes. Future research should target ways to increase educational opportunities in efforts to attenuate enduring negative effects of early life disadvantages.

## Clinical implications

Individuals with more than one ACE reported higher levels of SCC. While these concerns can occur irrespective of objective cognitive deficits, they are often predictive of future cognitive decline (Mendonça et al., 2016; Rabin et al., 2017). However, SCC may also reflect a negative attention bias and is highly correlated with subclinical levels of depression (Jessen et al., 2014; Rabin et al., 2017). These findings warrant further investigation in older adults with ACE to improve understanding of the interrelationships among heightened levels of depression, perceived cognitive difficulties, and objective impairment.

Within this study, current subclinical depression symptoms, but not a clinical history of depression, were significantly associated with greater ACE and current perceptions of lower self-efficacy. Notably, subclinical depression has emerged as a strong predictor of cognitive function in older adults (MacAulay et al., 2020). Research suggests early life adversity strongly predicts risk for developing mental illness such as depression, anxiety, and substance abuse disorders (Turecki et al., 2014). It is plausible that predispositions to psychopathology are directed by genes and shaped by early experiences creating an additive vulnerability (Schwartz, Wright, & Valgardson, 2019; Turecki et al., 2014). Even in circumstances in which adversity has dissipated, early life stress can have enduring negative effects (Pechtel & Pizzagalli, 2011) that are beyond that produced by more proximal negative life events (Korten et al., 2014). These enduring effects may speak to the complexity of gene-environment interactions and epigenetic influences such as prenatal and intergenerational exposure to adversity that increase the risk for vulnerability factors shared by both early adverse experiences and mental illness (Schwartz et al., 2019). These effects may also impart a greater likelihood of negative response bias in those individuals reporting high numbers of ACE. As such, investigating the potential cascading effects of early adversity on future psychopathology is warranted.

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## Strengths and Limitations

A strength of this study included the use of CBPR approaches, leading to increased enrollment of socioeconomically diverse participants. Recruitment efforts targeted older adults, and the time of testing was controlled to provide optimal times for cognitive performance in this population. The use of a comprehensive neuropsychological battery to test specific cognitive domains also highlights a strength of this study. Single global measures of cognitive function are commonly used to represent cognitive status. However, these measures can lack the sensitivity and specificity needed to detect subtle cognitive differences appropriately. Global measures may also preclude detection of singular domain impairments when other domains are within normal limits.

Although the present study extends the literature by investigating the effects of childhood adversity on cognition in socioeconomically diverse older adults, limitations include a racially and ethnically homogenous sample, reflecting the 94.7% non-Hispanic white population estimate for the state of Maine (U.S. Census Bureau, 2018). Studying these factors within a racially and ethnically diverse sample is needed to determine if racial and ethnic disparities potentiate accumulative effects. Future research should also examine the role of education quality as it has been shown to predict cognitive abilities beyond years of education alone (Crowe et al., 2013).

## Conclusion

In summary, the current study found that the relationship between objective cognitive performance and childhood adversity was mediated by years of education. These results indicate that childhood adversity may indirectly impact cognitive functioning in older adults by reducing factors related to building and maintaining cognitive reserve, such as greater educational attainment. Interestingly, greater levels of SCC were reported in those with adverse childhoods. In line with prior research, greater ACE were also associated with greater levels of subclinical depression and lower socioeconomic status (e.g., less education and less annual income). Taken together, individuals with adverse backgrounds appear to be more vulnerable to experiencing numerous known risk factors for cognitive decline. An important goal will be to understand the additive effects of these risks toward late-life cognition in older adults. In addition, identification of resilience factors is also warranted, considering that specific coping styles, personality traits, or cognitive processes may elucidate approaches that can be used to foster self-esteem and reduce behavioral risk factors in this population. Future research should emphasize identifying mechanisms that may serve a mediating or moderating role in the relationship between ACE and late-life cognition.

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## CONFLICT OF INTEREST

The authors have no conflict of interest to disclose.

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