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Early Life Adversity Predicts Reduced Hippocampal Volume in the Adolescent Brain Cognitive Development Study

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Abstract

Purpose: Cross-sectional studies in adults have demonstrated associations between early life adversity (ELA) and reduced hippocampal volume, but the timing of these effects is not clear. The present study sought to examine whether ELA predicts changes in hippocampal volume over time in a large sample of early adolescents.

Methods: The Adolescent Brain Cognitive Development Study provides a large dataset of tabulated neuroimaging, youth-reported adverse experiences, and parent-reported financial adversity from a sample of children around the United States. Linear mixed effects modeling was used to determine the relationship between ELA and hippocampal volume change within youth (n = 7036) from ages 9–10 to 11–12 years.

Results: Results of the models indicated that the number of early adverse events predicted bilateral hippocampal volume change ($\beta = -0.02$, $t = -2.02$, $p < .05$). Higher adversity was associated with lower hippocampal volume at Baseline ($t = 5.55$, $p < .01$) and at Year 2 ($t = 6.14$, $p < .001$).

Discussion: These findings suggest that ELA may affect hippocampal development during early adolescence. Prevention and early intervention are needed to alter the course of this trajectory. Future work should examine associations between ELA, hippocampal development, and educational and socioemotional outcomes.

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Supplementary Data

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Keywords

Adolescent; Adversity; Hippocampus; ABCD; Neurodevelopment

The hippocampus plays a major role in the formation and reconstruction of memories and is strongly associated with learning. The hippocampus has a high density of glucocorticoid receptors and is, therefore, especially sensitive to stress [1]. Glucocorticoids, including the stress hormone cortisol, are released following activation of the body's stress response system, the hypothalamic-pituitary adrenal axis, and bind to glucocorticoid receptors in the hippocampus. Acutely, stressors (in early life or otherwise) result in glucocorticoid release. However, dysregulation of glucocorticoid release resulting from early-life adversities is associated with chronic overactivation of the hypothalamic-pituitary adrenal axis during childhood and glucocorticoid dysregulation in adulthood [2]. In the hippocampus, heightened glucocorticoid exposure can result in dendritic atrophy and the suppression of neurogenesis (i.e., the formation of new neurons) [3], suggesting a process by which early life adversity (ELA) leads to volumetric changes in this brain area. Indeed, in adults reporting histories of childhood maltreatment, researchers have consistently found reduced hippocampal volumes compared to adults without a history of childhood maltreatment [4,5].

Findings from studies of children and adolescents are inconsistent, with moderate evidence of reduced hippocampal volume following ELA [6]. Humphreys and colleagues [7] identified a relationship between very early (<5 years) stressful life events and reduced bilateral hippocampal volume in adolescents. This finding was not replicated by stressful life events occurring after age 5 [7]. Similarly, a longitudinal study found ELA during pre-school years, but not school-age years, were associated with decreased hippocampal volume in adolescents [8]. Another longitudinal study linked child maltreatment to increased left hippocampal volume in adolescents and decreased hippocampal volume growth indirectly mediated by psychopathology during early adolescence [9].

Variability of results regarding the effects of stressors on hippocampal volume in adolescence may be due to different windows of vulnerability to stress, sampling periods in adolescence, or overall study design. Few studies have used a longitudinal design to explore these factors. Indeed, even among those with a longitudinal design, few have included more than one neuroimaging session. A longitudinal approach utilizing two neuroimaging timepoints allows us to establish temporal precedence, as we have measures of hippocampal volume at more than one time point. Further, we can better characterize trajectories of within-individual change in hippocampal development as a function of ELA. Inferences regarding developmental change using cross-sectional designs are limited by confounds such as period and cohort and cannot be used to examine within-individual change [10]. In the current study, we aim to fill these gaps by using a within-subjects longitudinal design to examine the influence of ELA on youth hippocampal volume over two years. We hypothesized that ELA would predict a smaller increase in child hippocampal volume from Baseline to their second follow-up, two years later.

Methods

This secondary data analysis was conducted on data from the 5.0 Data Release of the Adolescent Brain Cognitive (ABCD) Study, a multisite, longitudinal research project conducted in the United States (21 research sites - abcdstudy.org, https://doi.org/10.15154/8873-zj65).

Participants

The ABCD Study enrolled 11,875 youth at ages 9- and 10-years old and will follow the cohort for 10 years [11]. Data Release 5.0 includes data on the complete sample for three annual time points: Baseline (ages 9–10 years), Year 1 follow-up (ages 10 – 11 years), and Year 2 follow-up (ages 11–12 years). (Note that none of the variables with values corrected in the 5.1 Data Release were used in the current study.) Youth and parents completed self-report assessments each time point, and youth completed structural magnetic resonance imaging (MRIs) at both Baseline and the Year 2 follow-up assessment, see Figure 1. All study procedures were approved by the central IRB at the University of California, San Diego.

Neuroimaging

Structural MRIs were completed at Baseline (ages 9–10, mean age 9.9 years, n = 11,867) and during the Year 2 follow-up (ages 11–12, mean age 11.9 years, n = 8,092 with structural MRI). For full details on the imaging acquisition protocol, including harmonization across sites and scanners (n = 29), please see [12–14]. Subjects were removed from the current analysis if 1) they did not have a quality control measure score ([15]; 239 subjects, n = 7853); 2) both their scans did not pass the quality control measure ([15]; 559 scans, 424 subjects, n = 7,429); 3) their two scans were less than 18 months or more than 30 months apart (155 subjects, n = 7,274) or 4) they did not have complete variables to calculate ELA and covariates to complete the model (238 subjects, n = 7,036). All structural neuroimaging processing was completed by the ABCD Data Analysis and Informatics Core using FreeSurfer version 5.3.0 (aseg, http://surfer.nmr.mgh.harvard.edu/) according to standardized processing pipelines as described by Hagler et al. [15], including automated subcortical segmentation [16]. Tabulated data was acquired from the NIMH Data Archives for this secondary analysis. Total hippocampal volume was summed from the left and right regions of interest provided in the abcd_smrip10201.txt data package. To account for sites with multiple scanners, scanner serial number was used in lieu of site ID as a covariate.

Early life adversity

Adverse childhood experiences are commonly measured with a 10-item scale [17], but this scale was not collected in the ABCD Study. An ELA sum score was modeled after the more conservative approximation score introduced by Karcher and colleagues [18], see Table S1. Measured in the Year 1 follow-up, this 12-item ELA sum score includes traumatic life experiences and chronic financial instability. Traumatic life events include five youth self-reported items regarding household dysfunction from the PhenX Life Events Questionnaire (“One of the parents/care-givers went to jail?”, “Parents separated or divorced?”, “Someone in the family died?”, [child] “Was a victim of crime/violence/assault?”, “Family member

had a mental/emotional problem?"). Added to the adverse experiences were seven parent-reported financial adversity variables: inability to afford food or phone service, missed rent/mortgage payments, evictions, utilities disconnected due to nonpayment, inability to afford medical or dental care. These items were summed to create a composite score representing ELA.

Statistical analyses

Because there are only two timepoints and the independent variable (ELA) cannot be randomly assigned [19], we chose to use a difference score as our estimate of hippocampal growth. Within-subject hippocampal volume change scores were created by subtracting the Baseline volumetric measurement from the Year 2 follow-up volumetric measurement. Linear mixed-effects models were conducted to test the relationships between total hippocampal volume change and the ELA sum score. The model included the child's age in months at Year 2, caregiver-reported race/ethnicity, sex at birth, time period between the two MRI scans (in months), baseline total intracranial volume, as well as caregiver education. Caregiver education was included as an approximation for socioeconomic status. Race/ethnicity were included in the model to account for the disproportionate rate of ELA among youth of different racial and ethnic backgrounds [20]. ABCD reports race and ethnicity as a single variable with five levels [21]. Random effects (intercepts) for family nested within scanner ID were also included in each model. Linear mixed-effects models were conducted in R [22] using lme4 [23] and lmerTest [24]. To further understand any effects of laterality, the same models were run separately for the right and left hippocampi as exploratory analyses.

Results

A final sample of 7,036 participants were included in the analyses. Demographic information and participant characteristics are available in Table 1. The sample had a mean ELA score of 1.57 ($SD = 1.37$), with 1,243 participants having an ELA score 1 SD above the mean (i.e., a score of three or more on a scale of 1–12). Linear mixed effects modeling for bilateral hippocampal volume change revealed a main effect for ELA, $\beta = -0.02$, $b = -3.69$, $SE_b = 1.83$, $t = -2.02$, $p = .043$. Time (in months) between Baseline and Year 2 scans ($t = 5.64$, $p < .001$), age at Year 2 ($t = -4.35$, $p < .001$), and Asian race ($t = 2.27$, $p = .023$) were also significant predictors of hippocampal change; see Table 2. Exploratory analyses were conducted to examine effects of ELA by hemisphere (Table S1).

Models of hippocampal change by hemisphere demonstrated a significant main effect of ELA for the right hippocampus ($\beta = -0.03$, $b = -2.51$, $SE_b = 1.12$, $t = -2.25$, $p = .024$), but no significant effect for the left hippocampus, ($\beta = -0.01$, $\beta = -1.30$, $SE_b = 1.24$, $t = -1.05$, $p = .29$). This pattern indicates that effects of ELA were primarily driven by the right hippocampus; see Table S2.

Post hoc analyses

In order to determine specific effects of ELA, we performed descriptive post hoc analyses to compare bilateral, left, and right hippocampal volume for youth with ELA scores of ≥ 3

and those with an ELA score of 0; see Table S3. These scores represent 1 standard deviation above and below the mean, rounded to the nearest whole number. As described above, the ELA scale ranged from 0–12. Mean hippocampal volume of the full sample at Baseline was 8,185.65 mm^3 (*SD* = 785.92).

Descriptive analyses of Baseline bilateral hippocampal volume revealed that those with an ELA score of ≥ 3 had a mean volume of 8,039.10 ± 792.19 mm^3, whereas the subset with ELA scores of 0 evidenced a mean volume of 8,220.27 ± 810.64) mm^3. This pattern indicates that youth with higher ELA scores had significantly smaller hippocampal volume at Baseline, t (2405.6) = 5.55, $p < .001$; see Figure 2. At Year 2, those with an ELA score of ≥ 3 had a mean volume of 8148.36 ± 808.53, whereas those with ELA scores of 0 had a mean volume of 8351.83 ± 820.91. Again, those with higher ELA scores had significantly smaller hippocampal volume at Year 2 follow-up, t (2405.6) = 6.14, $p < .001$. A descriptive comparison of mean volume change differences between those with an ELA score of 0 and those with an ELA score ≥ 3 indicates 17% less growth in the high ELA group.

Discussion

The present findings demonstrate the potential effects of early adversity on adolescent hippocampal development. During early adolescence, when hippocampal volume typically increases slightly [25], ELA was associated with reduced change in hippocampal volume, wherein youth with ≥3 ELAs had 17% less growth than those youth with none. Exploratory post hoc analyses revealed that these effects were largely driven by the right hippocampus and were not significantly different for males and females. These results provide new insight into the effects of ELA on human brain development.

Studies of adults have consistently found smaller hippocampal volumes in those exposed to early adversity [26]. Moreover, cross-sectional studies in both children and adolescents have also reported negative associations between ELA and hippocampal volume [27–29], suggesting onset of these effects during childhood. The current study adds to the body of evidence supporting that ELA impacts hippocampal volume through slower volumetric change in early adolescence. Consistent with our findings, a meta-analysis found volumetric differences associated with ELA specifically in the right hippocampus [30]. The body of evidence on childhood maltreatment suggests that the effects of childhood adversity on hippocampal volume may be strongest during puberty [5,31]. The current study aligns with these findings, providing evidence that early adversity is affecting the hippocampus during early adolescence, a time of prime development in regions important for emotion reactivity and regulation [32].

Early adversity is associated with emotion dysregulation and psychiatric disorders in both adolescents and adults [33,34], including in the ABCD sample [14,35–38]. A proposed neurobiological process that underlies these findings is through heightened glucocorticoid release as a result of chronic stress. There is evidence to suggest these glucocorticoids inhibit neurogenesis in the hippocampus [3]. One aspect of the hippocampus's role in memory function is the emotional aspects of episodic memory [39]. As psychiatric disorders such as major depressive disorder have been associated with alterations in autobiographical

memory retrieval [40], it is possible that this is a mechanism through which early adversity affects socioemotional outcomes. Indeed, previous studies have reported reduced hippocampal volume as a mediator between childhood adversity and later mental health outcomes [41,42]. Another possibility, however, is that neurobiological effects such as reduced hippocampal volume may represent adaptive responses to a stressful environment. Additional longitudinal studies that include measures of mental health and related outcomes, particularly in late adolescence and adulthood, are needed to disentangle these effects.

The current study has the advantage of studying a large adolescent sample longitudinally at standardized ages. One limitation of this study, however, is there is not detailed information about the timing of the early life experiences. A cross-sectional study of early adolescents found effects for early adversity on hippocampal volume only for those with adverse experiences prior to age 5, suggesting a sensitive period for these events [7]. While the timing of events occurring prior to Baseline in the ABCD Study were not measured, future data releases will allow us to determine if adverse experiences that occur during adolescence (i.e., new life events occurring between research sessions) affect subsequent hippocampal development. Relatedly, the Life Events questionnaire was administered at Year 1 (after the Baseline scan), and thus some items included in the ELA variable may have occurred following the Baseline scan. It is also important to note the current study uses a cumulative risk approach to examine ELA (i.e., dichotomizing each adverse experience and summing the dichotomous scores), which assumes the underlying mechanisms by which these experiences influence outcomes are the same or similar. Thus, this approach may mask the differential effects of severity, chronicity, and/or different types of adversity on developmental outcomes [43,44]. While we were constrained to this approach due to our dataset, future researchers might consider using a dimensional approach that assesses the severity and frequency of different types of adverse experiences on hippocampal development. Additionally, the effect sizes in our study were small, and further research is needed to determine whether these hippocampal differences impact cognition, behavior, and/or mental health. These small effect sizes are in line with previous ABCD findings, however, and researchers have suggested that a “recalibration” of effect size interpretation may be needed to account for the typically small effects found in this study [45]. Our findings of both reduced hippocampal volumes at Baseline and less growth over the two-year study period indicate that these small effects may accumulate over time [21,46], resulting in possible behavioral and clinical impacts in later adolescence and adulthood. Future research is needed to determine the persistence of these effects throughout the lifespan and possible associations with socio-emotional and educational outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding Sources

Data used in the preparation of this article were obtained from the Adolescent Brain Cognitive Development (ABCD) Study (https://abcdstudy.org), held in the NIMH Data Archive (NDA). This is a multisite, longitudinal study designed to recruit more than 10,000 children age 9–10 and follow them over 10 years into early adulthood. The ABCD Study is supported by the National Institutes of Health and additional federal partners under award numbers U01DA041048, U01DA050989, U01DA051016, U01DA041022, U01DA051018, U01DA051037,

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IMPLICATIONS AND CONTRIBUTION

This study reveals a significant association between early life adversity and reduced hippocampal volume change during the early adolescent developmental period between ages 9 and 12 years. Future studies should examine if this relationship persists across middle and late adolescence and whether it may be predictive of socioemotional and educational outcomes.

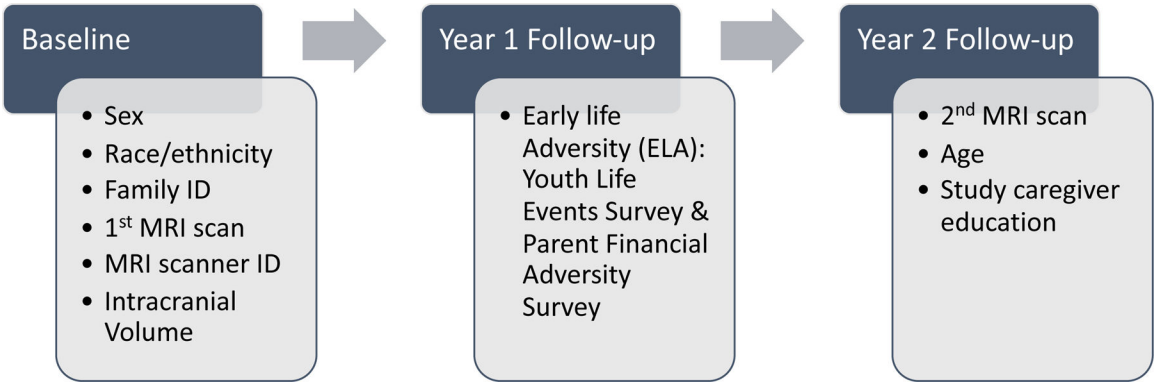

Figure 1.
Timeline of ABCD Assessments and data acquisition.

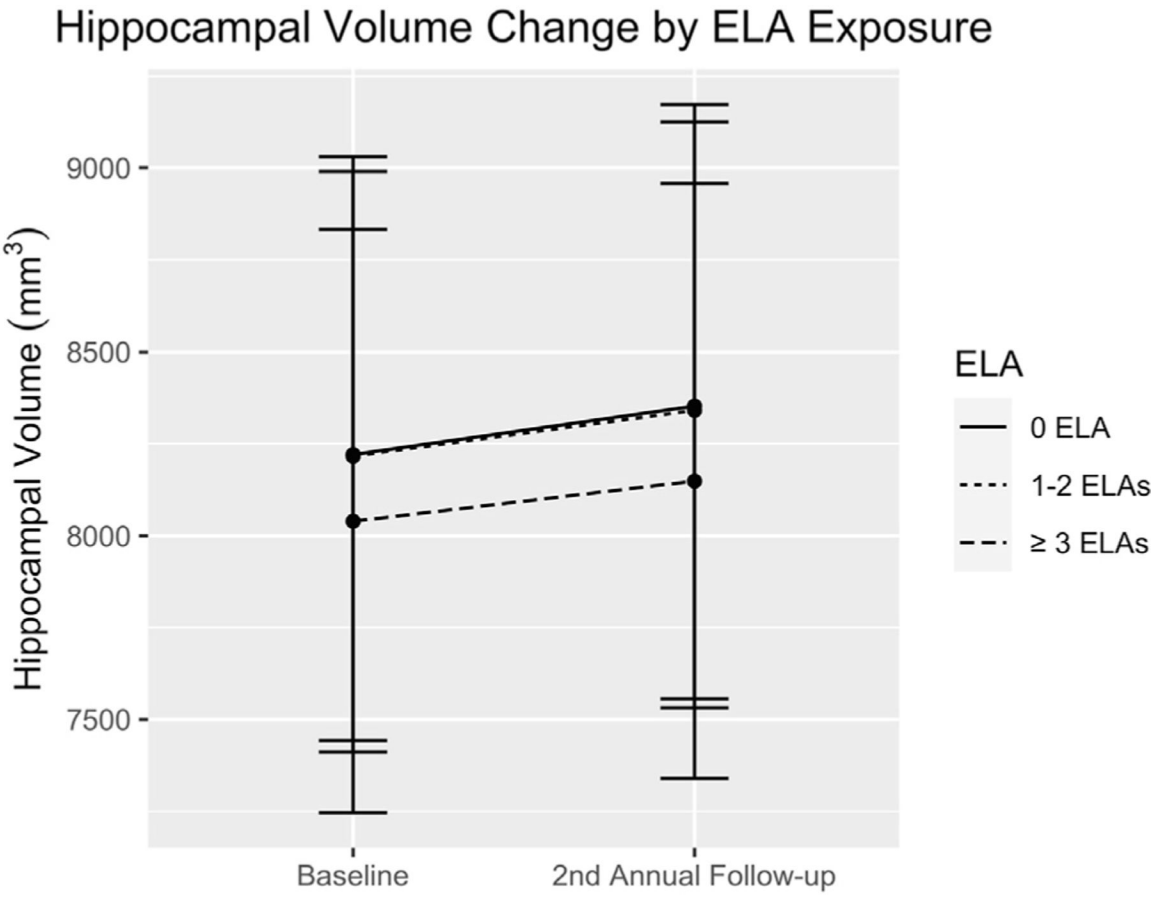

Figure 2.
Hippocampal volume changes between Baseline and Year two by ELA exposure. Note: Error bars represent standard error of the mean.

Table 1.

Participant characteristics and adversity scores.

Early Life Adversity	***Mean***	***SD***
	1.57	1.37
ELA Endorsed	*N*	*%*
0	1178	16.74
1	3098	44.03
2	1517	21.56
3	654	9.30
4	273	3.88
5	162	2.30
6	83	1.18
7	38	0.54
8	27	0.38
9	4	0.06
10	2	0.03
11	0	0.00
12	0	0.00
Race/Ethnicity		
White	3965	56.35
Black	878	12.48
Hispanic	1338	19.02
Asian	135	1.92
Other	720	10.23
Combined Household Income		
< $5,000	187	2.66
$5,000 – $ 11,999	203	2.89
$12,000 – $15,999	133	1.89
$16,000 – $24,999	289	4.11
$25,000 – $34,999	365	5.19
$35,000 – $49,999	571	8.12
$50,000 – $74,999	962	13.67
$75,000 - $99,999	1027	14.60
$100,000 – $199,999	2068	29.39
> $200,000	723	10.28
Refuse to answer	241	3.43
Don’t Know	267	3.79
Study Caregiver Education Level		
8th Grade or Less	81	1.15
Some High School, No Degree	313	4.45
High School Diploma or GED	650	9.24
Some College, No Degree	2078	29.53

Early Life Adversity	*Mean*	*SD*
Bachelor's Degree	2115	30.06
Master's Degree	1391	19.77
Professional or Doctorate Degree	408	5.80

Table 2.

Linear mixed-effects model results: Bilateral hippocampal volume.

	b	SE	β	t	p	R^2
Hippocampal Volume						.084
ELA	−3.69	1.83	−.02	−2.02	<.05	
Sex (male)	4.47	5.58	.01	0.80	.42	
Time Between Scans	7.28	1.29	.07	5.64	<.001	
Intracranial Volume	0.00	0.00	.02	1.11	0.27	
Age at Year 2	−1.44	0.33	−.05	−4.35	<.001	
Caregiver Education	1.36	2.23	.01	0.61	.54	
Race/Ethnicity (Black)	0.82	8.64	.00	0.10	.92	
Race/Ethnicity (Hispanic)	6.98	7.69	.03	0.91	.36	
Race/Ethnicity (Asian)	41.81	18.40	.20	2.27	<.05	
Race/Ethnicity (Other)	−1.22	8.53	−.01	−0.14	.89	