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Archival Report

Dimensions of Early-Life Adversity Are Differentially Associated With Patterns of Delayed and Accelerated Brain Maturation

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ABSTRACT

BACKGROUND: Different types of early-life adversity (ELA) have been associated with children's brain structure and function. However, understanding the disparate influence of distinct adversity exposures on the developing brain remains a major challenge.

METHODS: This study investigates the neural correlates of 10 robust dimensions of ELA identified through exploratory factor analysis in a large community sample of youth from the Adolescent Brain Cognitive Development Study. Brain age models were trained, validated, and tested separately on T1-weighted (n = 9524), diffusion tensor (n = 8834), and resting-state functional (n = 8233) magnetic resonance imaging data from two time points (mean age = 10.7 years, SD = 1.2, age range = 8.9–13.8 years).

RESULTS: Bayesian multilevel modeling supported distinct associations between different types of ELA exposures and younger- and older-looking brains. Dimensions generally related to emotional neglect, such as lack of primary and secondary caregiver support and lack of caregiver supervision, were associated with lower brain age gaps, i.e., younger-looking brains. In contrast, dimensions generally related to caregiver psychopathology, trauma exposure, family aggression, substance use and separation from biological parent, and socioeconomic disadvantage and neighborhood safety were associated with higher brain age gaps, i.e., older-looking brains.

CONCLUSIONS: The findings suggest that dimensions of ELA are differentially associated with distinct neurodevelopmental patterns, indicative of dimension-specific delayed and accelerated brain maturation.

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Early-life adversity (ELA) such as exposure to abuse, violence, neglect, and chronic poverty, among others, can have widespread effects on youth neurodevelopment (1) and increase risk for mental disorders (2,3). Previous studies investigating how ELA influences neural development have adopted varied theoretical frameworks, reflecting the complex and multifaceted nature of the field (4,5). Traditionally, research has focused on a single type of adversity, reporting associations between heightened amygdala activation and early exposure to violence (6), and lower volumes of gray matter and low income (7) and exposure to institutionalization (8). A limitation of this approach, however, is that most children are exposed to numerous types of adversities concurrently (9). The cumulative risk approach overcomes this by aggregating different forms of adversity into a singular risk factor. Early cumulative risk has been prospectively associated with lower total gray matter volume, cortex volume, and right superior parietal and inferior parietal cortical thickness (10). While the cumulative risk approach is useful for identifying children at greatest risk for intervention and can thus serve as a vital public health tool, aggregating risk factors may obscure potentially diverging effects of different adverse experiences on the developing brain.

More nuanced perspectives that have emerged differentiate between adverse experiences related to threat versus deprivation (11), drawing support for the neural basis of this distinction from studies on fear learning and sensory deprivation. For children exposed to threat, studies have reported lower cortical thickness, surface area, and volume of the amygdala and hippocampus (12–14) and ventromedial prefrontal cortex (PFC) (15,16), in addition to reduced resting-state amygdala–ventromedial PFC connectivity (17). The observed effects may be indicative of accelerated maturation and are consistent with life history and evolutionary biology theories proposing accelerated development as an adaptation to harsh or stressful environments (4,18–20). Similarly, another conceptual model, the stress acceleration hypothesis, argues that adversity may expedite neural development as a means of compensating for the absence of species-expectant maternal buffering of emotional reactivity (21).

For children exposed to deprivation, such as institutional rearing, lack of social and cognitive stimulation, and other forms of parental absence, research has revealed altered structure and function in the frontoparietal network, amygdala-hippocampal-PFC connectivity (11,22), and reductions in

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cortical gray matter and total brain volume, in addition to widespread cortical thinning (8,23,24). Moreover, electroencephalography studies have shown associations between spectral profiles indicative of delayed patterns of functional and cortical maturation and neglect (25), poverty (26), and parental stress (27).

While previous research has contributed to an understanding of how different features of ELA are associated with unique brain outcomes (28), real-world occurrences of adversity are multifaceted and often co-occur in a complex manner, making it a considerable challenge to precisely account for the heterogeneity in ELA. To address this, several data-driven methods have been applied (29–33), albeit on small or homogeneous samples. Recent research by Brieant *et al.* (34) capitalized on big data from the Adolescent Brain Cognitive Development (ABCD) Study and identified 10 dimensions of adversity co-occurrence pertaining to conceptual domains reflecting 1) caregiver psychopathology, 2) socioeconomic disadvantage and lack of neighborhood safety, 3) secondary caregiver lack of support, 4) primary caregiver lack of support, 5) child report of family conflict, 6) caregiver substance use and biological parent separation, 7) family anger and arguments, 8) family aggression, 9) trauma exposure, and 10) caregiver lack of supervision. Yet, to date, the neural correlates of these dimensions of ELA have not been investigated.

Brain age prediction offers a framework that combines multiple imaging features and yields an individualized marker of brain maturation (35). Brain age involves estimation of biological age based on brain magnetic resonance imaging (MRI) characteristics, which may differ from an individual's chronological age (36). This difference, termed the brain age gap (BAG), could reflect deviation from typical neurodevelopmental patterns, and has been validated in several neurodevelopmental studies (37–40). Previous literature has also validated the stability of brain age models across early adolescence, with evidence of BAG scores tracking with metrics of maturation (40). Studies have also linked lower BAG in youth to attention-deficit/hyperactivity disorder, lower socioeconomic status, higher anxiety and depression, and greater psychopathology symptom severity (37,41–44). In the context of ELA, in which different dimensions of adversity may be associated with unique brain outcomes (4,28,45–49), brain age can probe individualized markers of delayed or accelerated maturation by means of younger- or older-looking brains.

To this end, using ABCD Study data, we aimed to test for associations between MRI-based estimates of brain maturation and 10 previously characterized dimensions of ELA co-occurrence (34). Based on theoretical accounts and the reviewed empirical studies, we hypothesized ELA dimensions of caregiver psychopathology, socioeconomic disadvantage and lack of neighborhood safety, child report of family conflict, caregiver substance use and biological parent separation, family anger and arguments, family aggression, and trauma to be associated with older-looking brains and accelerated maturation between the two time points (TPs). We hypothesized ELA exposures of secondary caregiver lack of support, primary caregiver lack of support, and caregiver lack of supervision to be associated with younger-looking brains and delayed maturation over time.

METHODS AND MATERIALS

Sample and Ethical Approval

The ABCD Study (50) comprises children and adolescents part of an ongoing longitudinal study. Participants were excluded using the ABCD Study exclusion criteria listed elsewhere (51). Data used in the present study were drawn from the ABCD curated annual release 5.0, containing data from baseline up until the second-year visit (https://data-archive.nimh.nih.gov/abcd). All ABCD Study data are stored in the NDA (National Institute of Mental Health Data Archive) Collection #2573, which is available for registered and authorized users (Request #7474, principal investigator: LTW). The 5.0 release is defined in the NDA Study 1299 and has been assigned the DOI http://dx.doi.org/10.15154/8873-zj65. The Institutional Review Board at the University of California San Diego approved all aspects of the ABCD Study (52). Parents or guardians provided written consent, while the child provided written assent. The current study was conducted in line with the Declaration of Helsinki and was approved by the Norwegian Regional Committee for Medical and Health Research Ethics (REK 2019/943).

Demographic Information and Data Quality Assurance

The initial sample consisted of ∼11,800 participants (52% male) at mean age 10.75 years (SD = 1.18, age range = 8.92–13.83 years), with baseline and 2-year follow-up observations of T1-weighted imaging (T1) (observations = 19,048), diffusion tensor imaging (DTI) (observations = 17,672), and resting-state functional MRI (rs-fMRI) (observations = 16,495) data. Quality control procedures followed a standard protocol described in Hagler *et al.* (53). Briefly, participants with excessive head motion or poor data quality were excluded from the curated data release by the ABCD Study team. Additional quality assurance was carried out following extraction of data using the recommendations for data cleaning provided by the ABCD Study team (using data structure abcd_imgincl01). Following quality assurance, the final sample included 19,047 T1 observations, 17,668 DTI observations, and 16,466 rs-fMRI observations used for the current study. Demographic information for each brain MRI modality–specific sample can be found in Table S1 in Supplement 2, while the T1 sample is illustrated in Figure 1.

MRI Acquisition and Processing

Neuroimaging data were acquired at 21 different sites (using 31 scanners) and processed by the ABCD Study team. A 3T Siemens Prisma, GE 750, or Philips scanner was used for data acquisition. Protocols used for data acquisition and processing are described in detail elsewhere (50,53) and available in Section S1 in Supplement 1. Three modalities of brain structural and functional measures were used in the present study: structural gray matter measures (T1), diffusion white matter microstructural measures (DTI), and resting-state functional connectivity measures (rs-fMRI) (53). Cortical surface reconstruction and subcortical segmentation was performed with FreeSurfer v7.1.1 (54,55). White matter microstructural measures were generated using AtlasTrack, a probabilistic atlas-based method for automated segmentation of white matter

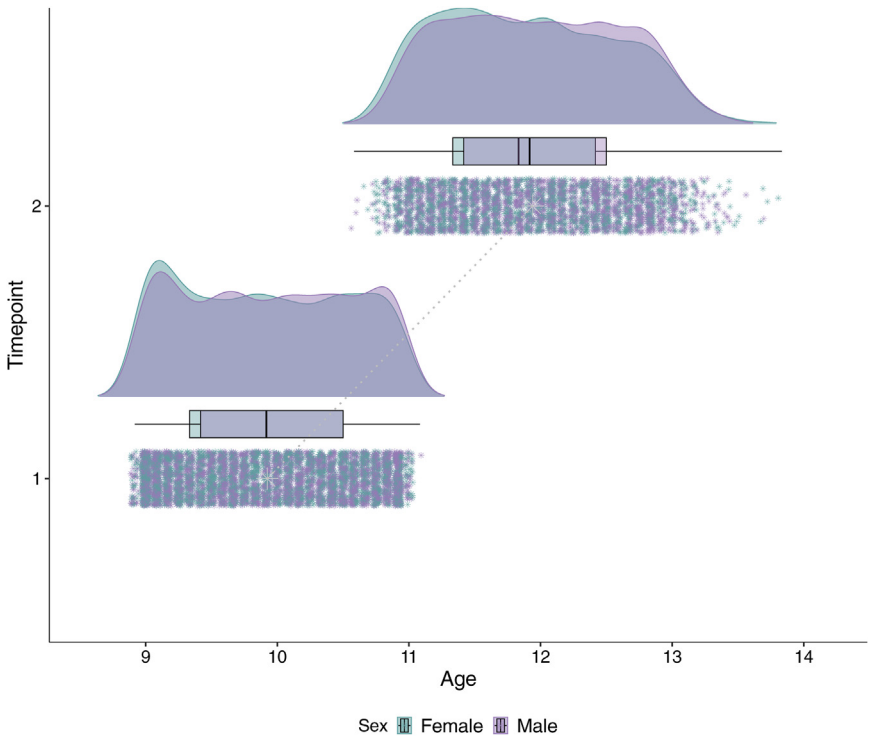

Figure 1. Age distribution split by sex and time point. Time points 1 and 2 represent baseline and 2-year-follow-up data, respectively, from the Adolescent Brain Cognitive Development Study cohort. Female data are shown in teal blue; male are shown in lavender. The dotted gray line is drawn between mean age at time points 1 and 2.

fiber tracts (56). Measures of functional connectivity were computed using a seed-based, correlational approach (57), in which average time courses were calculated for cortical surface-based regions of interest using a functionally defined parcellation based on resting-state functional connectivity patterns (58) and subcortical regions of interest (55).

For T1, we extracted tabulated total and regional measures of cortical surface area, thickness, volume, sulcal depth, intensity-gray-white contrast, and subcortical volume (397 measures). For DTI, full and inner (multi)shell tissue properties including functional anisotropy and mean diffusivity, longitudinal (or axial) diffusivity, and transverse (or radial) diffusivity were extracted for total and regional features (576 measures). For rs-fMRI, functional connectivity within and between parcellations from Gordon network, including subcortical data (58), were extracted (416 measures). Following procedures of quality assurance (see Demographic Information and Data Quality Assurance), harmonization of multiscanner effects was carried out using *longCombat* (59) (see Section S2 and Figures S1–S3 in Supplement 1).

Brain Age Prediction

Brain age prediction was carried out using the XGBoost regression model (60). Parameters were tuned using 10-fold cross-validation, stratified by age. The models were fitted using the best estimators and optimized models were applied to the (hold-out) test sample. r^2, root mean square error, and mean absolute error were calculated to evaluate prediction accuracy in the test set. For each brain modality (T1, DTI, rs-fMRI), 50% of the data were used as the hold-out test sample and 50% were used for model training and validation. Here, data were split, ensuring an equal distribution of cross-sectional and longitudinal data across training and testing samples, whereby no 2 data points from the same individual were separated.

Consistent with a recent brain age article using the ABCD Study sample (61), confounding effects from complex family-related factors were minimized using a group shuffle split with family ID as the group indicator to ensure that no siblings were split across training and test sets. A detailed overview of T1, DTI, and rs-fMRI training and test samples, including demographic information, are provided in Section S3 and Figures S4–S6 in Supplement 1; Table S1 in Supplement 2. A complete list of all the extracted measures used for brain age prediction per modality is provided in Tables S2–S4 in Supplement 2. Feature importance scores for each model are provided in Figures S7–S9 in Supplement 1. To adjust for commonly observed age bias (overestimated predictions for younger participants and underestimated predictions for older participants) (62), we applied a statistical correction previously described (63). The difference between an individual's predicted brain age and their chronological age (BAG) was calculated (BAG = predicted age – chronological age) for each of the models, providing T1-, DTI-, and rs-fMRI–based BAG values for all participants.

Early-Life Adversity

The current study utilized 10 previously obtained factor scores from 60 measures of ELA as detailed in Brieant *et al.* (34). Briefly, Brieant *et al.* identified 139 potential ELA items from the

ABCD Study baseline measures, which encompass a spectrum of ELA constructs including caregiving disruption, caregiver psychopathology, maltreatment, and neighborhood safety/violence, among others. These variables were sourced from child and parent reports, as well as researcher assessments, and originated from modified versions of validated scales. To identify dimensions, 60 ELA variables that were binary, polytomous, and continuous in nature were entered into an exploratory factor analysis conducted in Mplus version 8.7 (64), resulting in 10 dimensions (F1 to F10; see Table S5 in Supplement 2 and Figure S10 in Supplement 1 for correlation matrix). To obtain factor scores, an exploratory structural equation model was carried out specifying the number of factors identified in the exploratory factor analysis. Further details of the variable selection process, identification of ELA dimensions, and calculation of factor loadings can be found in Brieant *et al.*

Statistical Analysis

All analyses were carried out using R version 4.2.1 (65). To investigate the association (main effect) between each ELA dimension (F1:F10) and deviation from expected age patterns (i.e., BAG), and whether the effect of each ELA dimension on BAG varies across TPs (interaction effect), Bayesian multilevel models were carried out using the *brms* R package (66,67). Here, multivariate models are fitted in familiar syntax to comparable frequentist approaches such as a linear mixed-effects model using *lme4* (68). We assessed the relationship between each ELA dimension at baseline and residualized (age bias corrected) BAG, in which modality-specific BAG (T1, DTI, rs-fMRI) was first entered as the dependent variable and each ELA dimension (F1:F10) and interaction term (TP:F1–F10) between TP and ELA dimensions (F1:F10) were separately entered as independent fixed-effects variables with sex entered as a covariate, and with subject ID as the random effect.

To prevent false positives and to regularize the estimated associations, we defined a standard prior around zero with an SD of 0.1 for all regression coefficients, reflecting an expectation of effects being small but allowing for sufficient flexibility in estimation. To ensure robust convergence and adequate sampling from the posterior distributions, each model was run with 8000 iterations, including 4000 warmup iterations, across 4 chains. For each coefficient of interest, we report the mean estimated value of the posterior distribution (*b*) and its 95% credible interval (the range of values that with 95% confidence contains the true value of the association), and calculated the Bayes factor (BF)—provided as evidence ratios—using the Savage-Dickey method (69). Briefly, BF can be interpreted as a measure of the strength of evidence (extreme, very strong, strong, moderate, anecdotal, none) in favor of the null or alternative hypothesis. For a pragmatic guide on BF interpretation, see Table S6 in Supplement 2.

RESULTS

Descriptive Statistics

Descriptive statistics can be found in Table S1 in Supplement 2. Table 1 summarizes descriptive and model validation statistics pertaining to each model. Figure 2 shows predicted age as a function of chronological age. Figure S11 in Supplement 1 shows a correlation matrix including each modality-specific predicted brain age and BAG.

Table 1. Descriptive and Model Validation Statistics

	T1	DTI	rs-fMRI
r^2	0.34	0.43	0.17
RMSE	0.96	0.89	1.08
MAE	0.79	0.71	0.89
r (95% CI)	0.59 (0.57–0.60)	0.66 (0.65–0.67)	0.41 (0.39–0.43)

DTI, diffusion tensor imaging; MAE, mean absolute error; RMSE, root mean square error; rs-fMRI, resting-state functional magnetic resonance imaging; T1, T1-weighted imaging.

Bayesian Multilevel Modeling

Bayesian multilevel modeling tested the association between each ELA dimension and modality-specific BAG. The full results are available in Tables S7–S9 in Supplement 2 and are visualized below in Figure 3. For estimated credible intervals, see Figures S12 and S13 in Supplement 1.

T1 BAG Relations. For T1 BAG, the test revealed evidence of a positive association between ELA dimension F2 (socioeconomic disadvantage and neighborhood safety) and T1 BAG (BF $<$ 0.01, β = 0.13), indicating that this dimension was associated with older-looking brains. However, the effect did not accelerate over time, as indicated by no interaction effects of TP (BF = 3.27, β = 0.01). Further, the tests revealed evidence of negative associations between ELA dimensions F3 (secondary caregiver lack of support) (BF = 0.9, $\beta = -0.05$) and F4 (primary caregiver lack of support) (BF = 0.17, $\beta = -0.08$) and T1 BAG, indicating that dimensions related to caregiver emotional neglect are associated with younger-looking brains. These effects did not indicate delayed maturation over time (F3: BF = 1.21, $\beta = -0.04$; F4: BF = 2.21, $\beta = -0.03$). In terms of interaction effects of TP, we found a negative association between F10 (lack of supervision) and T1 BAG (BF = 0.08, $\beta = -0.09$) but no main effect (BF = 2.65, $\beta = -0.02$), indicating that unsupervised youth diverge more from normative age patterns throughout the course of the study period.

DTI BAG Relations. For DTI, the tests revealed evidence of a positive association between F2 and DTI BAG (BF = 0.27, β = 0.06) but no interaction effects of TP (BF = 2.86, β = 0.02), aligning with the T1 BAG findings, and indicating that those living in more disadvantaged and less safe environments may have older-looking brains. In terms of interaction effects of TP, the tests revealed evidence supporting a positive association between ELA dimension F1 (caregiver psychopathology) and DTI BAG (BF = 0.09, β = 0.07) but no main effect (BF = 4.09, β = 0.01), indicating that the brain ages of these youth diverged more from normative age patterns over time.

rs-fMRI BAG Relations. For rs-fMRI, the tests revealed evidence of a positive association between F2 and rs-fMRI BAG (BF $<$ 0.01, β = 0.18) but no interaction effect of TP (BF = 1.71, β = 0.03), aligning with findings from DTI and T1 BAG. The tests also revealed positive associations between F1

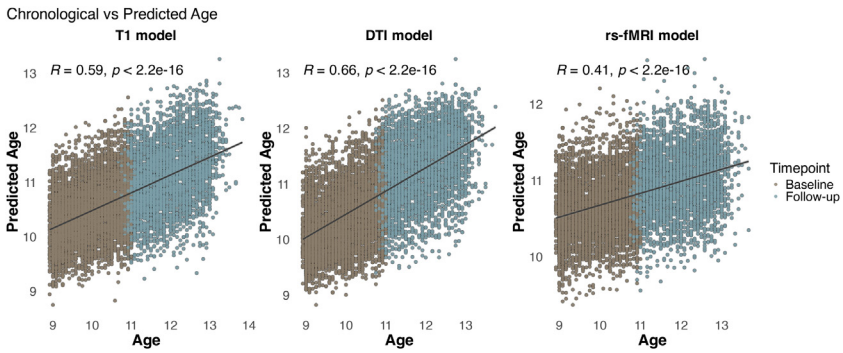

Figure 2. Predicted age as a function of age. Scatterplots demonstrating the correlation between chronological and predicted ages for three brain imaging modalities: T1-weighted imaging (T1), diffusion tensor imaging (DTI), and resting-state functional magnetic resonance imaging (rs-fMRI). Each plot illustrates the Pearson correlation coefficient (*r*) and statistical significance (*p* value), with data points representing individual samples at baseline and follow-up time points. The plots reveal varying degrees of performance accuracy across modalities, with the T1 and DTI models showing higher correlations compared with the rs-fMRI model.

(BF = 0.63, β = 0.07), F6 (caregiver substance abuse and separation from biological parents) (BF = 0.03, β = 0.15), F8 (family aggression) (BF = 0.66, β = 0.08), and F9 (trauma exposure) (BF = 0.07, β = 0.15) and rs-fMRI BAG, with a positive interaction effect for F6 (BF = 0.51, β = 0.09) but none for the other aforementioned dimensions (F1: BF = 2.07, β = 0.01; F8: BF = 1.64, β = 0.03; F9: BF = 1.74, β = 0.01). These positive associations largely indicate that dimensions linked to a threatening environment relate to older-looking brains, with effects also accelerating over time for F6. Further, the tests revealed a negative association between F4 and rs-fMRI BAG (BF = 0.27, β = −0.10), with no interaction effect of TP (F4: BF = 1.05, β = −0.08), in line with findings from T1 BAG. In terms of other interaction effects of TP, we found evidence supporting negative associations between both F3 (BF = 0.62, β = −0.08) and F5 (family conflict) (BF = 0.58, β = −0.08) and rs-fMRI BAG but no main effects (F3: BF = 1.19, β = −0.05; F5: BF = 1.19, β = −0.05).

DISCUSSION

Research indicates that approximately half of all children will experience at least one form of adversity by the time they reach adulthood (2,70). Different forms of ELA co-occur and may uniquely impact child brain structure and function. This work sought to delineate the potentially unique brain outcomes of 10 co-occurring dimensions of ELA in a longitudinal sample of 9- to 14-year-old youth. Our main findings indicate that ELA dimensions of caregiver lack of support and supervision are associated with younger-looking brains and that ELA dimensions of caregiver psychopathology, socioeconomic disadvantage and neighborhood safety, caregiver substance abuse and separation from biological parents, family aggression, and trauma exposure are associated with older-looking brains. Our findings largely align with and extend the current literature on the differential impact of different co-occurring patterns of environmental exposures and adversity on brain development (45,49).

Links to Accelerated Brain Maturation

Families living in lower socioeconomic status neighborhoods are exposed to more harm, such as interpersonal violence (71), and are more likely to have concerns about neighborhood safety (72). Positive associations between the dimension representing socioeconomic disadvantage and neighborhood safety (F2) and BAG were found with all 3 brain age models, and are consistent with research showing socioeconomic disadvantage and neighborhood violence linked to smaller cortical volumes and greater cortical thinning (73–75). Positive associations between exposure to trauma (F9) and BAG is also consistent with previous research showing that children exposed to trauma are more likely to be misclassified as adults by means of older DNA methylation age compared with chronological age, and earlier pubertal maturation (76–78).

For caregiver psychopathology (F1), we found both positive BAG associations as main effects and as interaction effects of TP, suggesting that parent psychopathology-related deviations from expected age-patterns accelerate over time. Our findings are challenging to interpret in the context of mixed results from previous research. ABCD Study findings have revealed smaller volume in the right putamen (79), left hippocampus (80), and bilateral hippocampi (81) in relation to parental psychopathology. These findings reflect age patterns of hippocampi and putamen usually not seen until late adolescents and young adulthood—whereby subcortical volumes are expected to reduce over time (see Figure S14 in Supplement 1) (82,83)—indicating accelerated maturation in our sample, and thus aligning with the directionality of our results. However, studies using other datasets have found that parental history of depression is associated with larger volume in the bilateral amygdala (84), and others have reported no effects (85). ELA dimensions that indicated the potential presence of household hostility including family aggression (e.g., throwing things, hitting) (F8) revealed a positive main effect for rs-fMRI BAG but no effects for family arguments (e.g., expressing anger, fighting, raising voices) (F7), with the latter finding going against our hypothesis.

Co-occurrence of caregiver substance abuse and separation from biological parents in one dimension (F6) might reflect child custody issues related to caregiver substance use disorders or arrests (34,86). Moreover, this dimension also has factor loadings from domestic violence. Our results revealed positive rs-fMRI BAG associations both as a main effect and an interaction effect of TP. Drawing from concepts of stress acceleration, parental deprivation accelerates the functional development of the medial PFC in children, such that amygdala–medial PFC interactions are more adult-like following deprivation experiences (21). Children in this group may have accelerated maturation as a means of adapting from

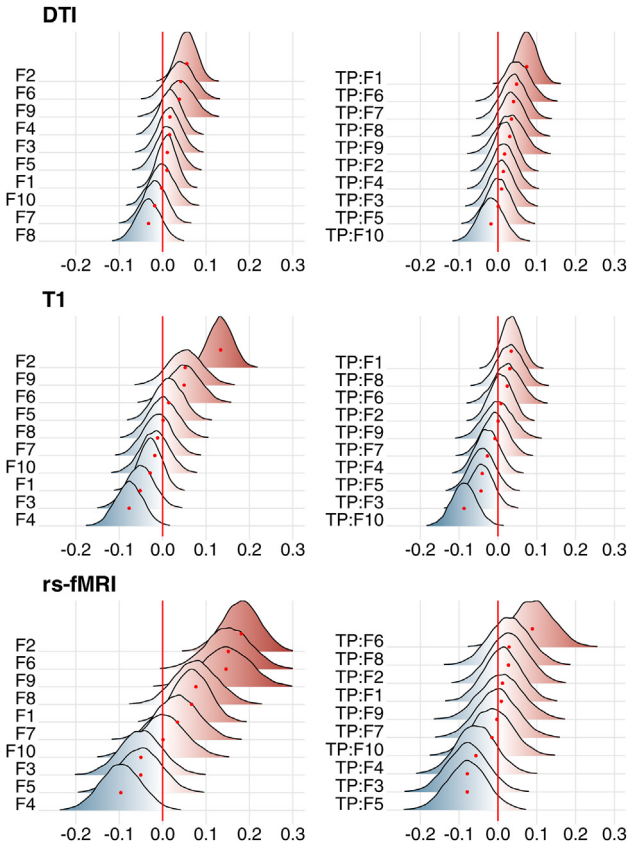

Figure 3. Associations between early-life adversity (ELA) dimensions and brain age gap for diffusion tensor imaging (DTI), T1-weighted imaging (T1), and resting-state functional magnetic resonance imaging (rs-fMRI). The figure shows posterior distributions of the estimates of the standardized coefficient. Estimates for each ELA dimension on brain age gap (main effect) on the left, and ELA dimension interaction effect of time point (TP) (TP:ELA) on brain age gap on the right. The color scale follows directionality of evidence, with positive (red) values indicating evidence in favor of positive associations (greater adversity linked to older-looking brains) and negative (blue) values indicating evidence in favor of negative associations (greater adversity linked to younger-looking brains) for each ELA dimension. The width of the distributions represents the uncertainty of the parameter estimates. F1 indicates caregiver psychopathology, F2 indicates socioeconomic disadvantage and neighborhood safety, F3 indicates secondary caregiver lack of support, F4 indicates primary caregiver lack of support, F5 indicates youth report of family conflict, F6 indicates caregiver substance use and separation from biological parent, F7 indicates family anger and arguments, F8 indicates family aggression, F9 indicates trauma exposure, and F10 indicates lack of supervision.

a state of parent-regulated to self-regulated emotional processing due to absent or inconsistent parental care (4,19–21). Further research is needed to understand the underlying mechanisms at play. In summary, dimensions of ELA co-occurrence related to older-looking brains support research reporting accelerated brain maturation in children exposed to potentially more hostile or dangerous environments.

Links to Delayed Brain Maturation

Neurobiological studies of brain development have long assumed a deficit model in which lack of input to the development of a child will result in delay of certain skills (87). In the child brain, this may be reflected by a delay in pruning and thus larger brain volumes and younger-looking brains. Our results support this, with lack of primary (F4) and secondary (F3) caregiver support as well as lack of caregiver supervision (F10) all revealing negative associations with T1 and rs-fMRI BAG, with main effects for F3 and F4, and interaction effects for F3 and F10. Importantly, these factors each share elements of emotional neglect.

Previous research investigating more severe forms of neglect (physical and emotional) have found that children reared in institutions demonstrate electroencephalogram patterns suggestive of a delay in cortical maturation in frontal, temporal, and occipital regions (25). However, a wealth of studies also report conflicting results indicative of advanced maturation in similar samples (8,24). A caveat of research carried out on neglect is that children are often in environments enriched for several co-occurring ELAs, making it difficult to rely on the stability of the neglect scores while considering other stressors. Importantly, our results specifically capture emotional forms of neglect in terms of lack of household emotional support in a community with relatively lower levels of risk, which may explain divergence from prior studies on extreme physical neglect or institutionalization.

Last, a negative interaction effect of TP for rs-fMRI BAG and family conflict (F5) was also found, indicating that youth exposed to family conflict diverge more from normative age patterns (i.e., delayed maturation) over time. This contradicted our hypothesis and findings from previous research also utilizing the ABCD Study cohort, which reported high family conflict associations with smaller cortical surface areas of the orbitofrontal cortex, anterior cingulate cortex, and middle temporal gyrus (88). In summary, ELA dimensions related to emotional neglect were associated with younger-looking brains and delayed maturation.

Strengths and Limitations

Our study adds new insight into the neural correlates of co-occurring ELA dimensions in a large-scale longitudinal community sample. Using a sample not enriched for adversity exposure has the added value of demonstrating that even less severe ELA exposure is associated with changes in the developing brain, facilitating broader generalization. However, there is trade-off in that our findings cannot necessarily be generalized to interpret neural correlates of children exposed to more severe forms of adversity, as these groups may not be well represented in the ABCD Study. Future research is required to address this without losing adequate power. There remain also additional challenges such as accounting for differences in chronicity of adversity events, interindividual differences in resilience, and overlap in adversity types. More future directions are discussed in Section S4 in Supplement 1.

Conclusions

The current study supports notions that brain MRI outcomes related to ELA are differentially associated with accelerated and delayed brain maturation. Neurodevelopmental processes influenced by experiences of trauma, parental psychopathology, socioeconomic disadvantage and neighborhood safety,

caregiver substance abuse and separation from biological parents, and family aggression are at least partially distinct from those influenced by experiences of emotional neglect, with brain age deviations indicating differential maturational patterns.

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ARTICLE INFORMATION

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