SUPPLEMENT

Associations Between Early-Life Stress Exposure and Internalizing Symptomatology During the COVID-19 Pandemic: Assessing the Role of Neurobehavioral Mediators

Supplement 1: Participant inclusion criteria

All participants were required to (a) be between the ages of 18-30; (b) be free of current treatment with psychotropic medication; (c) display an IQ >80; (d) be free of lifetime history of head trauma resulting in loss of consciousness for more than five minutes; (e) be right-handed; (f) be free of contraindication for MRI scanning; and (g) be free of chronic medical illness and neurological disorder. $N = 64$ participants met the primary inclusion criteria for this study, which included the completion of: (1) two resting-state functional MRI scans; (2) a detailed clinical interview regarding their life history of stress exposure; (3) a questionnaire battery regarding self-reported symptomatology and emotion regulation strategies; and (4) a battery of additional questionnaires sent to participants following the onset of the COVID-19 pandemic.

A total of $N = 191$ individuals completed Phase 1 measures (i.e., completed interview assessing ELS exposure, questionnaires related to self-reported internalizing symptomatology, resting-state fMRI scans). Participants were paid $25/hour for completing the first part of the study and $50 upon completion of the scan visit. Individuals who participated in Phase 1 were sent an email inviting them to complete COVID-related measures in Phase 2 of the study. A total of $n = 84$ participants responded and completed the Phase 2 measures (i.e., questionnaires related to COVID-19 pandemic and emotion regulation). Of these participants, $n = 20$ participants were excluded from the sample in the current registered report due to having incomplete ELS, self-reported internalizing symptomatology, and/or resting-state fMRI data from Phase I of the study,

resulting in the final sample reported in the registered report ($N = 64$). Participants who completed the follow-up study during the COVID-19 pandemic were electronically sent an Amazon gift card valued between $5 and $15, depending on the percentage of the battery they completed.

In order to assess attrition bias, independent samples t-tests and chi-square tests were run to examine whether participants who completed all Phase 1 and Phase 2 measures ($N = 64$) and participants who did not complete all Phase 2 measures ($N = 98$) differed significantly on demographic variables (income, age, sex assigned at birth, race/ethnicity [number of non-Hispanic White participants relative to non-White participants]) or ELS exposure (average reported severity across all ELS exposures). Family income level, sex assigned at birth distribution of sample, racial/ethnic identity distribution of sample, highest level of education completed distribution of sample, and average ELS severity scores were not significantly different among individuals that completed all measures and those that did not complete all Phase 2 measures (all p-values > .05). The average age of participants differed ($p = .009$, $d = .39$) between those that completed all measures from Phase 1 and Phase 2 ($M_{age} = 21.94$, $SD = 3.35$) and those that did not complete all Phase 2 measures ($M_{age} = 23.19$, $SD = 3.03$). Age was entered as a covariate in all analyses, and we note this potential source of attrition bias as a limitation.

Supplement 2: Study timing

ELS and resting-state functional connectivity data were obtained from participants over a period of several years (Phase 1), prior to the onset of the COVID-19 pandemic. The elapsed time between Phase 1 and Phase 2 varied among participants (M = 16, SD = 10, range = 3-40 months). To account for this variability, the number of months between fMRI scan and completion of pandemic-related questionnaires were included as a covariate in all analyses. Questionnaire data collected during the COVID-19 pandemic were obtained in mid-to-late May 2020. Though mid-to-late May saw a significant decrease in daily deaths and infections in the general U.S. population, relative to the peak in April 2020, by May 15th, it was estimated that the U.S. had suffered over 90,000 total COVID-19 related deaths (1). Additionally, between May 15th and May 18th 2020, 87% of Americans reported that they were practicing social distancing (i.e., staying home and avoiding others; 2).

Supplement 3: Measures (additional information)

University of California, Los Angeles Posttraumatic Stress Disorder Reaction Index (UCLA PTSD RI)

An extended version of the University of California, Los Angeles Posttraumatic Stress Disorder Reaction Index (UCLA PTSD RI) (3) was developed to assess an individual's trauma history. The extended version of the UCLA PTSD RI contains 25 modules, each designed to query a specific type of adverse experience (e.g., physical abuse, neglect). Participants were asked to report on the number of ages at which they were exposed to each type of adverse experience. Participants were also asked to report on the subjective severity of each event (with each event operationalized as exposure to a specific event type at a specific age; e.g., sexual abuse at age 8) using an 8-point Likert scale ranging from 0 (not at all) to 8 (extremely). ELS severity for each participant was calculated by averaging the severity scores reported across all events endorsed prior to the age of 12.

Emotion Regulation Questionnaire (ERQ)

The Emotion Regulation Questionnaire (ERQ; 4) was used to assess individuals' tendency to use two distinct emotion regulation strategies: reappraisal (6 items) and suppression (4 items). Participants rated their agreement with statements describing two strategies (e.g., "When I'm faced with a stressful situation, I make myself think about it in a way that helps me stay calm."), on a 7-point Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree). Ratings from each scale are summed, yielding a total score ranging from 6-42 for the reappraisal scale and from 4-28 for the suppression scale. Across both scales, higher scores indicate greater use of that emotion regulation strategy. The ERQ has displayed good construct validity and 3-month test-retest reliability (4). The scale scores for both reappraisal and suppression strategies

were used in the current study to assess reliance on prototypically-adaptive and maladaptive emotion regulation strategies, respectively.

Beck Depression Inventory-II (BDI-II)

The Beck Depression Inventory (BDI-II; 5) was used to assess self-reported depressive symptomatology. The BDI-II is a 21-item self-report measure of depression symptom severity. Participants rated items describing depressive symptoms on a 4-point Likert scale, from 0 (e.g., "I do not feel like a failure.") to 3 (e.g., "I feel I am a total failure as a person."). The ratings across all items are summed, yielding a total score ranging from 0-63, with higher scores indicating higher levels of depression symptomatology. The BDI-II has demonstrated both high concurrent and discriminative validity (5,6), as well as excellent test-retest reliability (7). Because we were unable to monitor clinical risk in the context of an online questionnaire, Item 9, which assesses self-harm and suicidality (i.e., "Suicidal thoughts or wishes."), was omitted from the Phase 2 protocol. Total standardized scores (z-scores) on the BDI-II and the SCARED-A were summed to create a singular metric for COVID-related internalizing symptomatology. This is in line with previous work that has used a composite score of well-established scales to create a single metric of internalizing symptomatology for model simplification purposes (8–10).

Screen for Child Anxiety Related Emotional Disorders-Adult (SCARED-A)

The Screen for Child Anxiety Related Emotional Disorders-Adult (SCARED-A; 8) was used to assess self-reported anxiety-related symptomatology. The SCARED-A is an adaptation of the original Screen for Child Anxiety Related Emotional Disorders-Child (SCARED-C; 6), an instrument used to assess various dimensions of child anxiety. The SCARED-A has been found to have good internal consistency, test-retest reliability, and discriminant validity (12). This modified version of the SCARED is similar in content to the child version, albeit rephrased to

better match an adult's perspective. The SCARED-A consists of 71 items that are rated on a 3-point Likert scale ranging from 0 (almost never) to 2 (often), with total scores ranging from 0-142. The SCARED-A captures the same subscales as the SCARED-C: panic disorder (13 items), generalized anxiety disorder (9 items), social phobia (9 items), separation anxiety disorder (12 items), obsessive compulsive disorder (9 items), post-traumatic stress disorder (4 items), (specific) phobia consisting of three types, namely animal phobia (3 items), blood injection-injury phobia (7 items), and situational environmental phobia (5 items). As noted above, total standardized scores (z-scores) on the BDI-II and the SCARED-A were summed to create a singular metric for COVID-related internalizing symptomatology.

The Epidemic – Pandemic Impacts Inventory (EPII)

The Epidemic – Pandemic Impacts Inventory (EPII; 10) was used to assess the impact of the COVID-19 pandemic on multiple domains of personal and family life. The EPII contains a series of questions about personal and familial impacts of the pandemic, across 10 different domains of life: work and employment, education and training, home life, social activities, economic, emotional health and well-being, physical health problems, physical distancing and quarantine, infection history, and positive change. Participants are asked to endorse whether a specific impact (e.g., "Increase in sleep problems or poor sleep quality") pertains to themselves or to someone that they live with during the pandemic. At the end of each list of questions assessing each domain listed above, we added a single question assessing the general degree of distress participants felt with regard to this specific domain (e.g., "In general, what is the level of distress you have experienced relating to employment and financial impacts due to the COVID-19 outbreak?"), which participants answered using a 7-point Likert scale ranging from 1 (Mildly distressing) to 7 (Highly distressing), that was modeled after a line of questions included in the

COVID-19 and Perinatal Experiences (COPE) study (14). Given that the EPII was developed in rapid response to the recent COVID-19 outbreak, psychometric information for this measure is not yet available. In order to reduce the number of covariates in our model, a composite score representing general COVID-related impacts was calculated by summing these 10 items queried at the end of each domain. This is in line with past work that has combined scores in individual domains of the EPII to formulate a composite score (15,16). Additionally, all five questions from the "economic" domain of the EPII were used to generate a total score for economic-related impacts. An endorsement of "yes" (indicating that the statement applies to themselves) for any of the five questions in this domain was coded as 1, for a scaled score of economic impacts ranging from 0-5.

Supplement 4: MRI acquisition protocol (additional information)

Mock scan

All participants completed a 20-minute mock scan session in a dedicated simulator at the scanning facility (bore diameter = 55-60 mm). The mock scanner is located at the [masked for review] Department of Psychology and is a replica of the 3.0 Tesla Siemens Prisma scanner at the [masked for review]. It is outfitted with speakers for simulating scanner sounds, a motorized table to mimic the motion of entering a scanner, and a head coil. Due to the fact that head motion is a significant concern in neuroimaging studies (17), participants completed motion training with behavioral shaping. During the training session in the simulator, a modified Wii remote was attached to the participant's head with a strap to monitor motion and provide feedback to the participant in the form of a vibration whenever the participant exceeded a set motion threshold (protocol developed by Niles Oien at University of Colorado, Boulder; full description of motion compliance training is provided in the appendix of Heller, 2017). Following motion compliance training, participants listened to pre-recorded scanner noises.

Resting-state scan

Participants completed two resting-state fMRI scans, which lasted five minutes each. Participants were positioned using foam padding and a weighted blanket to minimize motion and were fitted with OptoAcoustics noise-cancelling headphones to maximize reduction of external scanner noises and to enable participants to hear instructions during the scan (19). Participants were also fitted with a pulse monitor and respiratory belt to measure cardiac and respiratory cycle data during the scan session. Additionally, an emergency alarm ball was placed near the participant's left hand to be used if the participant needed to signal an emergency, and a microphone enabled participants to speak to the experimenter at any point during the scan.

During the resting-state scans, participants were instructed to focus on a white crosshair on a black screen.

MRI acquisition parameters

High-resolution anatomical images were acquired for each participant with a T1-weighted 3D gradient echo MPRAGE sequence in the axial plane (TR/TE = 2500 ms/2.88 ms, inversion time TI = 1060 ms, flip angle = 8°, FOV = 256 mm, matrix size = 256×256, slices = 176, 1.0mm isotropic) for transformation, co-registration, and localization of functional data into Montreal Neurological Institute (MNI) space. A whole-brain high-resolution T2-weighted fast spin echo was also acquired for detection and quantification of white matter lesions and cerebral spinal fluid (3200 ms TR; 565 ms TE; variable flip angle; 256 mm FoV; 176 slices in sagittal plane; 256 x 256 matrix; 2x parallel imaging; 1.0 x 1.0 x 1.0 mm resolution). Two fieldmap images were acquired in opposing phase-encoding directions (TR/TE = 8860 ms/80 ms, FOV = 216mm, multiband factor = 6, echo spacing = .56ms, 2.4mm isotropic). Functional scans were acquired using a multiband echo-planar imaging (EPI) sequence with (TR = 800 ms; TE = 30 ms; 60 axial slices; 52° flip angle, multiband factor = 6, echo spacing = .56ms, 2.4mm isotropic).

Imaging Preprocessing

Raw neuroimaging data were converted to Brain Imaging Data Structure (BIDS; 13) structure using heudiconv (www.github.com/nipy/heudiconv) and preprocessed with the Human Connectome Project (HCP) Minimal Preprocessing Pipeline (MPP; 16). The resting-state data underwent standard preprocessing under the MPP, which included gradient distortion correction, EPI field map preprocessing and distortion correction, motion correction, nonlinear registration to the MNI template (MNI 152 space), and grand-mean intensity normalization. EPI fMRI images were corrected using spin echo EPI scans. The FSL toolbox “topup” (22) was used to

estimate the distortion field, which was then aligned to the distorted gradient echo EPI single-band reference image using the FSL toolbox "FLIRT." The result was then concatenated with the topup-derived distortion field, and the EPI single-band reference image was distortion-corrected using spline interpolation (see Glasser et al., 2013 for additional details on HCP minimal preprocessing pipeline). Allowable rotation and translation movements were restricted to less than 1mm for participants. All individual-level analyses for the current study included regressors for residual head motion in each direction and their temporal derivatives.

Supplement 5: Analytic plan (additional information)

We implemented an anatomical component-based noise correction procedure (aCompCor) to denoise signal from cerebral white matter and cerebrospinal fluid, which was anatomically derived from each participant's T1-weighted structural scan that had been preprocessed into MNI space as described above (23). Scan volumes identified as outliers on the basis of motion or global signal were scrubbed to minimize BOLD variability. Acquisitions with framewise displacement above 0.9mm or global BOLD signal changes above 5 standard deviations were flagged as potential outliers and scrubbed (24), consistent with standard practices in the CONN framework (25). Participants with >50% volumes scrubbed were excluded from analyses. Temporal band-pass filtering was applied to remove frequencies below 0.01 Hz to remove low-frequency signal drift and above 0.1 Hz to remove high-frequency noise from respiratory and cardiac cycles or sudden motion (26). Cardiac and respiratory cycle data were loaded into the CONN toolbox at the denoising step. Based on these raw data, Retroicor (27) was used to generate predicted sine and cosine components of respiratory and cardiac effects to be included as potential confounds in linear regression models. Mean head motion was included as a covariate in first-level analyses in CONN. Following denoising, resting-state functional connectivity between the two ROIs was calculated as the Fisher Z-transformed correlation coefficient of their time courses in the CONN toolbox. Correlation coefficients from the two resting-state scans were averaged into a single value representing frontoamygdala connectivity for each participant.

Supplement 6: Power considerations

Because our sample for the current report was comprised of participants who completed data collection in Phase 2 ($N = 64$), we conducted a post-hoc power calculation to estimate achieved power for the indirect effects in our models using Monte Carlo simulations (28). Our simulations were run with one independent variable and two serial predictors, used 5,000 replications with 20,000 draws per replication, were specified to have a sample size of $N = 64$, and were run with a 95% confidence level. As population input parameters, we assumed medium-sized effects ($r > .4$) among our mediators, ELS, and psychopathology, consistent with effect sizes observed in prior work that has examined the effects of ELS on neurobehavioral development (29,30). This assumed effect size is also consistent with prior work that has used serial mediation models to examine mediators in associations between ELS and mental health outcomes (31). Achieved power was estimated to be > 0.80 for all indirect effects.

Supplement 7: Descriptive and correlation analyses (additional information)

Participant attributes are shown in **Table 1**. Descriptions and correlations of the variables in our primary models are shown in **Table 2**. None of the variables in our primary models were significantly correlated. **Table S1** depicts correlations for all independent, control, and dependent variables in our primary and supplementary models. Pre-pandemic internalizing symptomatology was negatively correlated with use of reappraisal as an emotion regulation strategy ($r = -0.251$, $p = 0.046$). Pre-pandemic internalizing symptomatology was also positively correlated with current, COVID-related internalizing symptomatology ($r = 0.590$, $p < 0.01$). COVID-related distress was positively correlated with current, COVID-related internalizing symptomatology ($r = 0.459$, $p < 0.01$). Cumulative ELS exposure was positively correlated with both pre-pubertal ELS severity ($r = 0.391$, $p < 0.01$) and pre-pandemic internalizing symptomatology ($r = 0.378$, $p < 0.01$). Additionally, the BDI and SCARED were shown to be significantly positively correlated with one another ($r = 0.58$, $p < 0.01$).

The mean number of valid volumes per participant was 747.36 (SD = 8.53), while the mean number of invalid volumes per participant was 18.64 (SD = 8.53). No participants were excluded due to an insufficient number of usable volumes post-scrubbing.

Supplement 8: Supplemental analyses

We additionally ran several supplementary serial mediation tests to examine the robustness of our findings. Specifically, we aimed to examine whether alternative operationalizations of ELS exposure would produce qualitatively different findings. The first two supplementary models shifted the cut-off age of the ELS independent variable from pre-pubertal exposure (i.e., prior to 12 years of age) to exposures experienced prior to 18 years of age. The first of these supplemental models examined the mediating effects of resting-state frontoamygdala connectivity and the use of reappraisal as an emotion regulation strategy on the relationship between ELS severity (averaged from ELS events experienced prior to the age of 18 years old) and internalizing symptomatology reported during the COVID-19 pandemic. **Table S2** displays the standardized coefficients for total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology in the serial mediation model. As found in our primary models, the direct association between ELS severity (pre-18) and COVID-related internalizing symptomatology was non-significant. All additional total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology in this primary model were also non-significant. **Table S3** shows total, individual, and serial indirect effects for pre-18 ELS severity on COVID-related internalizing symptomatology via frontoamygdala connectivity and reappraisal with bias-corrected 95% confidence intervals (CIs). There were no significant indirect effects of ELS severity on COVID-related internalizing symptomatology via frontoamygdala connectivity or via reappraisal. The second supplemental model examined the mediating effects of resting-state frontoamygdala connectivity and the use of suppression on the relationship between pre-18 ELS severity and COVID-related internalizing symptomatology. The standardized coefficients for total and direct effects on frontoamygdala

connectivity, suppression, and COVID-related internalizing symptomatology are shown in **Table S4.** All total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology in this primary model were non-significant. Additionally, there were no significant indirect effects in this model, as indexed in **Table S5**.

We additionally ran a set of supplemental models that operationalized ELS exposure in a cumulative manner as opposed to an average score of self-reported stressor severity. The third supplemental mediation model examined the mediating effects of resting-state frontoamygdala connectivity and the use of reappraisal on the relationship between a cumulative total of the number of ELS events that an individual experienced prior to age 12 and internalizing symptomatology reported during the COVID-19 pandemic. **Table S6** displays the standardized coefficients for total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology. Here, cumulative ELS had a direct and positive significant association with use of reappraisal as an emotion regulation strategy ($\beta = 0.2765$, $p = 0.037$). All additional direct and total paths in this model were non-significant. Additionally, there were no significant indirect effects in this model, as indexed in **Table S7**. The fourth supplemental mediation model examined the mediating effects of resting-state frontoamygdala connectivity and the use of suppression on the relationship between a cumulative total of the number of ELS events and internalizing symptomatology reported during the COVID-19 pandemic. **Table S8** displays the standardized coefficients for total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology. Here, all total and direct effects on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology in this primary model were non-significant. There were no significant indirect effects in this model, as indexed in **Table S9**.

In addition, a simple slope analysis was conducted to examine whether the slopes of the linear relationship between pre-pubertal ELS severity and COVID-related internalizing symptomatology for low and high levels of reappraisal usage differed significantly from 0. Results of the simple slope analysis showed that individuals who engaged in high reappraisal usage evidenced a negative relationship between ELS severity and internalizing symptomatology that was significantly different from 0 ($\beta = -0.744$, $p = 0.031$). Individuals who engaged in low reappraisal usage evidenced a positive relationship between ELS severity and internalizing symptomatology that was not significantly different from 0 ($\beta = 0.443$, $p = 0.138$).

Finally, supplemental exploratory analyses did not identify any significant interaction effects between ELS severity (pre-18) and the neurobehavioral moderators on COVID-related internalizing symptomatology, nor between cumulative ELS exposure and the neurobehavioral moderators on COVID-related internalizing symptomatology, as indexed in **Tables S10** and **S11**.

Table S1. ***Correlation analysis for all variables in primary and supplementary models.***

N = 64	ELS severity (pre-pubertal)	Frontoamygdala connectivity	Reappraisal	Suppression	COVID-related internalizing symptomatology	Age	Internalizing symptomatology (pre-pandemic)	COVID-related distress	Economic-related impact experienced during COVID-19	Elapsed time between phases	Cumulative ELS exposure	ELS severity (pre-18 years old)
ELS severity (pre-pubertal)	—											
Frontoamygdala connectivity	-0.087	—										
Reappraisal	0.142	-0.163	—									
Suppression	0.196	-0.102	0.098	—								
COVID-related internalizing symptomatology	0.045	0.104	-0.210	0.104	—							
Age	-0.250[a]	0.005	0.044	-0.363[a]	-0.236	—						
Internalizing symptomatology (pre-pandemic)	-0.015	-0.095	-0.251[a]	-0.064	0.590[a]	-0.168	—					
COVID-related distress	0.104	0.062	0.158	-0.057	0.459[a]	-0.168	0.317[a]	—				
Economic-related impact experienced during COVID-19	0.015	0.108	0.123	0.084	0.134	0.072	0.062	0.306[a]	—			
Elapsed time between phases	0.039	0.109	0.032	-0.133	0.132	0.150	0.031	0.080	0.153	—		
Cumulative ELS exposure	0.391[a]	-0.133	0.174	-0.049	0.230	-0.157	0.378[a]	0.193	0.135	-0.096	—	
ELS severity (pre-18 years old)	0.368[a]	0.125	-0.063	-0.026	0.115	-0.010	0.176	0.196	0.140	0.108	0.185	—

Legend: Table S1 provides descriptive statistics and correlations for the independent, mediating, control, and dependent variables.
M = Mean; SD = Standard deviation.
[a]significant (p<0.05)

Table S2. ***Standardized coefficients for total and direct effects of ELS experienced prior to age 18 on frontoamygdala resting-state connectivity, reappraisal, and COVID-related internalizing symptomatology***

	Frontoamygdala Connectivity	**Reappraisal**		**Internalizing Symptomatology**	
	Total/Direct Effect	**Total Effect**	**Direct Effect**	**Total Effect**	**Direct Effect**
ELS severity	0.1196	-0.0677	-0.0412	-0.0408	-0.0619
Frontoamygdala connectivity			-0.2215	0.1310	0.1018
Reappraisal					-0.1319
R^2	0.0492	0.1817		0.4858	

Legend: Table S2 displays the standardized coefficients for total and direct effects of ELS severity (for events experienced prior to age 18) on frontoamygdala resting-state connectivity, reappraisal, and COVID-related internalizing symptomatology in the serial mediation model. Also displayed is the direct effect of frontoamygdala connectivity on reappraisal, the total and direct effects of frontoamygdala connectivity on COVID-related internalizing symptomatology, and the direct effect of reappraisal on COVID-related internalizing symptomatology.

Table S3. ***Total, individual, and serial indirect effects for ELS experienced prior to age 18 on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology***

Pathway	Indirect Effect	SE	Bias-Corrected 95% CI	
			Lower	Upper
Total indirect	0.0212	0.0400	-0.0356	0.1262
ELS → Frontoamygdala connectivity → COVID-related internalizing symptomatology	0.0122	0.0199	-0.0140	0.0643
ELS → Reappraisal → COVID-related internalizing symptomatology	0.0054	0.0326	-0.0463	0.0931
ELS → Frontoamygdala connectivity → Reappraisal → COVID-related internalizing symptomatology	0.0035	0.0094	-0.0078	0.0300

Legend: Table S3 displays the total indirect, individual indirect, and serial indirect effects for ELS severity (for events experienced prior to age 18) on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology.
CI = 95% confidence interval; SE = standard error.

Table S4. ***Standardized coefficients for total and direct effects of ELS experienced prior to age 18 on frontoamygdala resting-state connectivity, suppression, and COVID-related internalizing symptomatology***

	Frontoamygdala Connectivity	Suppression		Internalizing Symptomatology	
	Total/Direct Effect	**Total Effect**	**Direct Effect**	**Total Effect**	**Direct Effect**
ELS severity	0.1196	0.0269	0.0130	-0.0408	-0.0586
Frontoamygdala connectivity			0.1162	0.1694	0.1502
Suppression					0.1654
R^2	0.0492	0.1994		0.4934	

Legend: Table S4 displays the standardized coefficients for total and direct effects of ELS severity (for events experienced prior to age 18) on frontoamygdala resting-state connectivity, suppression, and COVID-related internalizing symptomatology in the serial mediation model. Also displayed is the direct effect of frontoamygdala connectivity on suppression, the total and direct effects of frontoamygdala connectivity on COVID-related internalizing symptomatology, and the direct effect of suppression on COVID-related internalizing symptomatology.

Table S5. ***Total, individual, and serial indirect effects for ELS experienced prior to age 18 on frontoamygdala connectivity, suppression, and COVID-related internalizing symptomatology***

Pathway	Indirect Effect	SE	Bias-Corrected 95% CI	
			Lower	Upper
Total indirect	0.0179	0.0291	-0.0331	0.0858
ELS → Frontoamygdala connectivity → COVID-related internalizing symptomatology	0.0180	0.0247	-0.0182	0.0811
ELS → Suppression → COVID-related internalizing symptomatology	0.0021	0.0230	-0.0486	0.0492
ELS → Frontoamygdala connectivity → Suppression → COVID-related internalizing symptomatology	-0.0023	0.0050	-0.0119	0.0089

Legend: Table S5 displays the total indirect, individual indirect, and serial indirect effects for ELS severity (for events experienced prior to age 18) on frontoamygdala connectivity, suppression, and COVID-related internalizing symptomatology.
CI = 95% confidence interval; SE = standard error.

Table S6. ***Standardized coefficients for total and direct effects of cumulative ELS exposure experienced prior to age 12 on frontoamygdala resting-state connectivity, reappraisal, and COVID-related internalizing symptomatology***

	Frontoamygdala Connectivity	**Reappraisal**		**Internalizing Symptomatology**	
	Total/Direct Effect	**Total Effect**	**Direct Effect**	**Total Effect**	**Direct Effect**
ELS (cumulative)	-0.1233	0.3008	0.2765[a]	-0.0177	0.0360
Frontoamygdala connectivity			-0.1969	0.1242	0.0968
Reappraisal					-0.1390
R^2	0.0486	0.2419		0.4832	

Legend: Table S6 displays the standardized coefficients for total and direct effects of cumulative ELS exposure on frontoamygdala resting-state connectivity, reappraisal, and COVID-related internalizing symptomatology in the serial mediation model. Also displayed is the direct effect of frontoamygdala connectivity on reappraisal, the total and direct effects of frontoamygdala connectivity on COVID-related internalizing symptomatology, and the direct effect of reappraisal on COVID-related internalizing symptomatology.
[a]significant (p<0.05)

Table S7. ***Total, individual, and serial indirect effects for cumulative ELS exposure experienced prior to age 12 on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology***

Pathway	Indirect Effect	SE	Bias-Corrected 95% CI	
			Lower	Upper
Total indirect	-0.0539	0.0617	-0.1939	0.0484
ELS → Frontoamygdala connectivity → COVID-related internalizing symptomatology	-0.0121	0.0258	-0.0774	0.0274
ELS → Reappraisal → COVID-related internalizing symptomatology	-0.0384	0.0495	-0.1471	0.0494
ELS → Frontoamygdala connectivity → Reappraisal → COVID-related internalizing symptomatology	-0.0034	0.0106	-0.0318	0.0112

Legend: Table S7 displays the total indirect, individual indirect, and serial indirect effects for cumulative ELS exposure on frontoamygdala connectivity, reappraisal, and COVID-related internalizing symptomatology.
CI = 95% confidence interval; SE = standard error.

Table S8. ***Standardized coefficients for total and direct effects of cumulative ELS exposure experienced prior to age 12 on frontoamygdala resting-state connectivity, suppression, and COVID-related internalizing symptomatology***

	Frontoamygdala Connectivity	**Suppression**		**Internalizing Symptomatology**	
	Total/Direct Effect	**Total Effect**	**Direct Effect**	**Total Effect**	**Direct Effect**
ELS (cumulative)	-0.1233	-0.0984	-0.1140	-0.0177	0.0163
Frontoamygdala connectivity			-0.1268	0.1242	0.1453
Suppression					0.1664
R^2	0.0486	0.2098		0.4905	

Legend: Table S8 displays the standardized coefficients for total and direct effects of cumulative ELS exposure on frontoamygdala resting-state connectivity, suppression, and COVID-related internalizing symptomatology in the serial mediation model. Also displayed is the direct effect of frontoamygdala connectivity on suppression, the total and direct effects of frontoamygdala connectivity on COVID-related internalizing symptomatology, and the direct effect of suppression on COVID-related internalizing symptomatology.

Table S9. ***Total, individual, and serial indirect effects for cumulative ELS exposure experienced prior to age 12 on frontoamygdala connectivity, suppression, and COVID-related internalizing symptomatology, and bias-corrected 95% confidence intervals***

Pathway	Indirect Effect	SE	Bias-Corrected 95% CI	
			Lower	Upper
Total indirect	-0.0072	0.0122	-0.0326	0.0192
ELS → Frontoamygdala connectivity → COVID-related internalizing symptomatology	-0.0038	0.0085	-0.0229	0.0122
ELS → Suppression → COVID-related internalizing symptomatology	-0.0040	0.0071	-0.0180	0.0117
ELS → Frontoamygdala connectivity → Suppression → COVID-related internalizing symptomatology	0.0006	0.0019	-0.0023	0.0049

Legend: Table S9 displays the total indirect, individual indirect, and serial indirect effects for cumulative ELS exposure on frontoamygdala connectivity, suppression, and COVID-related internalizing symptomatology.
CI = 95% confidence interval; SE = standard error.

Table S10. ***Potential neurobehavioral moderators in the association between ELS severity (pre-18) and internalizing symptomatology reported during the COVID-19 pandemic***

Dependent Variable	Independent and Moderating Variables	*B* (95% CI)	SE	t	*p-value*
COVID-Related Internalizing Symptomatology	ELS Severity (Pre-18)	-0.276 (-0.920 to 0.367)	0.320	-0.862	0.393
	Reappraisal	-2.075 (-4.928 to 0.778)	1.421	-1.461	0.150
	ELS Severity x Reappraisal	0.270 (-0.340 to 0.880)	0.304	0.888	0.379
COVID-Related Internalizing Symptomatology	ELS Severity (Pre-18)	0.152 (-0.258 to 0.562)	0.204	0.745	0.456
	Suppression	0.761 (-0.324 to 1.845)	0.540	1.409	0.165
	ELS Severity x Suppression	-0.067 (-0.244 to 0.111)	0.089	-0.752	0.456
COVID-Related Internalizing Symptomatology	ELS Severity (Pre-18)	-0.022 (-0.152 to 0.108	0.065	-0.337	0.737
	Frontoamygdala Connectivity	2.941 (-1.709 to 7.590)	2.315	1.271	0.210
	ELS Severity x Frontoamygdala Connectivity	-0.194 (-0.948 to 0.560)	0.376	-0.517	0.608

Legend: Table S10 displays the effects of the interactions between ELS severity (experienced prior to age 18) and neurobehavioral predictors on COVID-related internalizing symptomatology.
B = unstandardized beta; CI = 95% confidence interval; SE = standard error.

Table S11. ***Potential neurobehavioral moderators in the association between cumulative ELS exposure and internalizing symptomatology reported during the COVID-19 pandemic***

Dependent Variable	Independent and Moderating Variables	*B* (95% CI)	SE	t	*p-value*
COVID-Related Internalizing Symptomatology	ELS (Cumulative)	-0.104 (-0.345 to 0.137)	0.120	-0.862	0.392
	Reappraisal	-0.795 (-1.693 to 0.102)	0.448	-1.776	0.081
	ELS (Cumulative) x Reappraisal	0.088 (-0.100 to 0.277)	0.094	0.937	0.353
COVID-Related Internalizing Symptomatology	ELS (Cumulative)	0.037 (-0.052 to 0.113)	0.041	0.747	0.459
	Suppression	0.574 (-0.058 to 1.206)	0.315	1.821	0.074
	ELS (Cumulative) x Suppression	-0.057 (-0.178 to 0.064)	0.060	-0.946	0.349
COVID-Related Internalizing Symptomatology	ELS (Cumulative)	0.009 (-0.041 to 0.058)	0.025	0.354	0.725
	Frontoamygdala Connectivity	2.399 (-0.554 to 5.352)	1.474	1.628	0.109
	ELS (Cumulative) x Frontoamygdala Connectivity	-0.177 (-0.545 to 0.190)	0.183	-0.966	0.339

Legend: Table S11 displays the effects of the interactions between cumulative ELS exposure and neurobehavioral predictors on COVID-related internalizing symptomatology.
B = unstandardized beta; CI = 95% confidence interval; SE = standard error.
[a]significant ($p<0.05$)

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