

Early life adversity and adolescent sleep problems during the COVID-19 pandemic

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Abstract

The COVID-19 pandemic resulted in a reorganization of adolescents' routines, especially their sleep schedules. Utilising 175 caregiver-adolescent dyads, the current study examined associations of biological (e.g., prenatal substance use), environmental (e.g., poverty), and relational (e.g., child maltreatment) subtypes of early life adversity (ELA) with various components of adolescents' sleep across the first year of the COVID-19 pandemic. Relational ELA explained unique variance in adolescents' sleep disturbances, but not other sleep components, following short- and longer-term exposure to the COVID-19 pandemic. However, the direction of this association switched such that relational ELA predicted decreased sleep disturbances during the initial phase of the U.S. COVID-19 pandemic in spring 2020 beyond pre-pandemic levels, but, over time, contributed to increased sleep disturbances beyond early-pandemic levels as the pandemic extended into the winter of 2020.

KEYWORDS

adolescence, COVID-19, early life adversity, longitudinal research, sleep

1 | INTRODUCTION

For many adolescents, the Coronavirus disease-2019 (COVID-19) pandemic and ensuing lockdowns resulted in a complete reorganization of daily routines amidst school closings and the cessation of extracurricular activities. Sleep patterns are an integral part of these daily routines and research to date has demonstrated both positive and negative changes in adolescents' sleep during the COVID-19 pandemic (Becker et al., 2021; Bruni et al., 2022; Santos & Louzada, 2022). Impacts of COVID-19 disruptions on adolescents' sleep duration and quality have been notable (Becker & Gregory, 2020; Ham et al., 2021), yet variable, with some youth showing increased sleep duration (Ramos Socarras et al., 2021) and others showing increased insomnia symptoms (Zhou et al., 2020). It is important to understand the short- and longer-term impacts of COVID-19 disruptions on adolescents' sleep given that sleep influences on brain development and well-being are heightened during this developmental period (Fontanellaz-Castiglione et al., 2020; Tarokh et al., 2016).

Adolescent sleep problems can negatively impact academic performance (Liu et al., 2021), mental health (Hestetun et al., 2018), and physical health (Quist et al., 2016). In recent years, researchers have examined potential contributors to adolescent sleep problems, with particular emphasis on media and technology use (Mei et al., 2018; van der Schuur et al., 2018), as well as contemporaneous life stressors (Baddam et al., 2019). However, given the organizational nature of development (Sroufe et al., 2009), we hypothesised that early childhood factors, including early life adversity (ELA), would hold unique significance for adolescents (Covington et al., 2021).

ELA encompasses negative experiences occurring early in development (e.g., poverty, child maltreatment; Lopez et al., 2021) and instantiates enduring biobehavioral disruptions that may take on increased salience during adolescence (Gunnar et al., 2019), and particularly in stressful contexts, such as during the COVID-19 pandemic (Oh et al., 2018; Petruccioli et al., 2019; Zhang et al., 2021). Indeed, longitudinal investigations of adolescent functioning across the COVID-19 pandemic have documented escalating

levels of perceived stress across the first year of the pandemic (beyond pre-pandemic levels, e.g., Molnar et al., 2023). Several dimensional models of ELA have emerged wherein events may be characterised based on the degree to which they feature threat versus deprivation (Sheridan & McLaughlin, 2014), harshness versus unpredictability (Ellis et al., 2009), and, most recently, integrative dimensions encompassing threat-based harshness, deprivation-based harshness, or unpredictability (Ellis et al., 2022). Other researchers have focused on specific types of adversity, such as relational events (e.g., abuse; Ridout et al., 2018) or environmental events (e.g., poverty; Engle & Black, 2008). Still others have argued for a cumulative approach wherein all events are summed into a single ELA composite (Evans & Whipple, 2013). Research studies using cumulative models of ELA reveal significant negative ELA impacts on sleep that extend into adolescence (April-Sanders et al., 2021).

Both dimensional and cumulative approaches to conceptualising and analysing ELA effects have their strengths. Dimensional approaches highlight the contributions of individual ELA types, whereas cumulative approaches capture the broader constellation of ELAs that children in high-risk circumstances encounter. In the current study, we characterised ELAs as biological, environmental, or relational based on the source of the adversity. In addition to supporting our evaluation of specific relations with adolescent sleep problems during COVID-19, this approach allowed us to meaningfully separate ELA subtypes from other risk factors with which they may be associated while supporting our evaluation of well-established developmental risks (e.g., parental age at birth, Bingley et al., 2000; de Kluvier et al., 2017) that are not readily captured by extant typologies. Thus, the first aim of this longitudinal investigation was to evaluate the unique, prospective, contributions of biological, environmental, and relational ELAs from birth to age 4 to adolescents' acute sleep problems during the first phase of the U.S. COVID-19 pandemic (i.e., sleep reports at age 15 during the first stay-at-home orders in spring 2020) beyond their pre-pandemic sleep reports at age 14.

Normative sleep patterns during adolescence include later bedtimes and decreased total sleep time, especially during school days (Tarokh et al., 2016). Intermittent periods of moderate sleep disturbances and daytime dysfunction (e.g., excessive daytime sleepiness) are also common during adolescence as they often coincide with pubertal development (Lagerberg et al., 2001). That said, sleep disturbances during adolescence are positively associated with both ongoing sleep problems (Dregan & Armstrong, 2010) and the emergence of psychological disorders in later development (Scott et al., 2021).

Mirroring the complexity of ELAs, sleep is a multifaceted construct (El-Sheikh & Sadeh, 2015). Extant research demonstrates differential associations between various predictors (e.g., negative family environment, parental warmth) and sleep characteristics in adolescence (Bartel et al., 2015; Khor et al., 2021). For example, Greenfield et al. (2011) found that child abuse experiences were more strongly related to adolescents' sleep disturbances than to other sleep components, such as sleep quality, latency, duration, or

efficiency. Although ELAs have been shown to influence sleep characteristics in adolescence, less is known about if and how specific ELA subtypes may influence specific components of adolescents' sleep in the context of major life stressors, such as the COVID-19 pandemic, and still less is known about whether these relations may vary across short-versus longer-term stress exposure.

Acute stress responses entail short-term physiological changes (e.g., elevated heart rate) that may or may not eventuate in later problems, but chronic stress exposure is typically associated with more enduring and more severe mental (e.g., depression) and physical (e.g., heart disease) health issues (Chu et al., 2021). With regard to adolescents' sleep patterns in the context of the COVID-19 pandemic, extant studies have focused on initial, short-term pandemic sleep responses (e.g., comparing pre- versus early-pandemic sleep patterns; Liao et al., 2021), to the detriment of understanding adolescents (Alfonsi et al., 2021). Examining *adults'* sleep patterns from March to October of 2020 in Italy, these authors found that a significant increase in sleep problems during the initial lockdown was followed by a gradual return to normative sleep patterns as lockdown restrictions relaxed. Given the potential for ELA exposure to differentially influence short-versus longer-term sleep responses to major life stressors, the second aim of this investigation was to examine if and how biological, environmental, and relational ELAs would predict adolescents' later-pandemic sleep reports in winter 2020 (i.e., 9 months into the pandemic) beyond their early-pandemic sleep reports in spring 2020.

The current study sought to fill gaps in our understanding of specific associations of biological (e.g., prenatal substance use), environmental (e.g., poverty), and relational (e.g., child maltreatment) subtypes of ELA with adolescents' sleep components (e.g., quality, sleep disturbances, daytime dysfunction) in the context of both acute and longer-term COVID-19 pandemic conditions. Following the recommendations of Evans and Whipple (2013), we improved upon a unitary cumulative adversity model to examine specific ELA subtypes, as well as the degree of ELA exposure using standardized severity scores across multiple ELAs within each subtype. Importantly, we also extended prior data suggesting ELA exerts negative effects on development beyond contemporaneous stress (Schroeder et al., 2020) by documenting the specific contributions of each ELA subtype severity to distinct components of adolescents' sleep while holding adolescents' reports of contemporaneous perceived stress constant. Finally, this study advanced beyond research documenting initial sleep patterns during the COVID-19 pandemic by examining adolescents' longer-term sleep patterns across the first 9 months of the pandemic. As suggested by El-Sheikh and Sadeh's (2015) developmental ecological systems model of sleep, we considered youth sex assigned at birth and ethnicity-race, in addition to contemporaneous measures of perceived stress *and* prior sleep patterns in all analyses.

Given that early relationships instill children with resources to navigate later relational challenges (Bowlby, 1973; Sroufe et al., 1999), we expected that children exposed to relational ELAs would be particularly vulnerable to sleep difficulties during COVID-19 for three reasons. First, COVID-19 restrictions limited these adolescents access to potentially restorative or protective relational connectives with

peers and extra-familial adults. Second, COVID-19 magnified these adolescents' exposure to potentially stressful relationships within the home. Third, although relational processes are salient for all adolescents as they (re)negotiate issues of autonomy and relatedness (Kobak et al., 2017), this (re)negotiation was disrupted by COVID-19 restrictions in ways that may have been especially challenging for youth with pre-existing relational vulnerabilities (Bülow et al., 2021).

2 | METHOD

2.1 | Participants

Participants were 175 caregiver-adolescent dyads who were drawn from an ongoing study of child development. Adolescents were diverse with regard to sex assigned at birth (49.7% female, 50.3% male) and ethnicity-race (46.9% Latine, 24.6% multiracial, 17.1% Black, and 10.9% white). Participating families were representative of the Southern California community from which they were recruited (U.S. Census Bureau, 2007). Caregivers provided data on their child's exposure to 33 distinct ELAs at the first study wave ($M_{\text{age}} = 4.07$ years, $SD = 0.23$ years) and, a decade later, adolescents provided data on their sleep patterns one year prior to the pandemic at age 14 ($N = 168$; $M_{\text{age}} = 14.23$ years, $SD = 0.50$ years), during the initial phase of the U.S. COVID-19 pandemic in spring 2020 ($N = 157$; $M_{\text{age}} = 15.22$ years, $SD = 0.57$ years), and nine months into the pandemic in winter 2020 ($N = 162$; $M_{\text{age}} = 15.84$ years, $SD = 0.56$ years).

The vast majority of our sample (95%) resided in Southern California during the first year of the COVID-19 pandemic. At the time of the first data collection wave in spring 2020, all participating adolescents had transitioned to on-line schooling and most (86.8%) remained entirely or mostly on-line at the time of the second data collection wave in the winter of 2020. Stay-at-home orders remained active in Southern California well into 2021 due to consistently high rates of COVID-19 morbidity and mortality. Of note, the predominance of ethnic-racial minority representation in the current sample (89.1%) may have resulted in particularly strong COVID-19 pandemic effects given documented disproportionalities in rates of infection, death, and serious income loss affecting ethnic-racial minorities in the United States (Tai et al., 2021).

2.2 | Procedures

Caregivers were invited to participate in a longitudinal study of children's early learning and development via flyers placed in community-based childcare centres in Southern California. Caregivers completed a brief screening by phone to ensure the target child was (1) between 3.9 and 4.6 years of age, (2) proficient in English, and (3) not diagnosed with a developmental disability. Although children had to be proficient in English due to limited interpreter resources, this was not a requirement for caregivers. At wave 1 (age 4), all families completed a three-hour assessment at our university

laboratory. A decade later, at age 14, adolescents completed a two-hour phone assessment. At ages 15 (Spring, 2020) and 15.5 (Winter, 2020), adolescents completed individual online assessments lasting 60–90 min. At each wave, caregivers and adolescents each received \$25 per hour of assessment. Informed consent was obtained from the legal guardian at all waves and informed assent was collected from adolescents. All procedures were approved by the human research review board of the participating university.

2.3 | Measures

2.3.1 | Early life adversity (ELA)

At age 4, caregivers reported on their child's lifetime exposure to 33 different adverse life events in the context of semi-structured face-to-face interviews. Two trained coders who were naïve to all information about the family coded the presence and severity of each adverse life event based on documented standards and coding manuals. Table 1 provides a summary of each ELA severity definition, reliability, and prevalence. Ten biological ELAs were evaluated during a semi-structured health interview that began with prenatal factors (e.g., prenatal care, prenatal substance exposure), extended across the child's delivery (e.g., birth complications), and covered health during infancy and early childhood. Nine environmental ELAs were coded based on caregiver reports of the family's economic status (i.e., income-to-needs; U.S. Census Bureau Housing and Household Economics Statistics Division, 2007) and housing experiences (e.g., homelessness, number of residential moves, household crowding), as well as neighbourhood data on crime (Federal Bureau of Investigation, 2007) and sociodemographic indicators (e.g., vacant homes, single parent households; U.S. Census Bureau, 2008) across the first 4 years of the child's life. Fourteen relational ELAs were assessed based on caregiver reports on the Early Trauma Inventory (ETI; Bremner et al., 2000). In addition to experiences of parental loss (e.g., death), separation (e.g., incarceration), and illness (e.g., diabetes), children's maltreatment exposure severity was coded using guidelines set forth by McGee et al. (1995).

Independent coders rated the severity of each ELA from no exposure (0), to mild (1), moderate (2), or severe (3) exposure. Although ELA chronicity is a salient influence on ELA effects (Benjet et al., 2011), we focused on ELA severity in the current study for three reasons. First, ELA severity has been linked to distinct developmental outcomes, as compared to ELA chronicity (Manly et al., 1994). Second, whereas all ELAs in the current study could be rated for severity, only a handful (e.g., health problems) could be differentiated based on actual or anticipated chronicity. Third, because we assessed ELAs during the first 4 years of life, our ability to accurately assess chronicity was limited. The Appendix provides a complete description of the ELA coding system. For these analyses, ratings were standardized and composited within subtype to yield an overall index of the child's biological ($ICC = 0.953$), environmental ($ICC = 0.883$), and relational ($ICC = 0.927$) ELA.

TABLE 1 Early life adversity (ELA) subtype definitions and descriptive statistics.

ELA domains & subtypes	Adversity definition	Severity level ICC	Percent affected ¹
Biological (N = 10)		0.953	
Maternal age at birth	Degree to which biological mom is younger or older at birth	0.957	22.1%
Paternal age at birth	Degree to which biological father's age is younger or older at birth	0.968	23.0%
Prenatal substance use	Degree of biological mother substance use during pregnancy	0.987	12.6%
Prenatal care	Degree to which biological mother received prenatal care	0.754	9.2%
Gestational age	Degree to which child was born prematurely	0.972	15.5%
Birth weight	Degree to which child was underweight or overweight	0.992	19.9%
Pregnancy complications	Degree to which biological mother and/or child experienced complications during pregnancy	0.978	33.3%
Delivery complications	Degree to which biological mother and/or child experienced complications during delivery	0.957	52.1%
Child health problems	Degree to which child has health problems	0.790	44.6%
Other biological adversity	Other biological ELA not captured by the existing subtypes	N/A	N/A
Environmental (N = 9)		0.883	
Household crowding	Degree to which the child experienced household crowding	0.558	9.9%
Residential mobility	Number of times the child moved residences	0.931	30.2%
Poverty	Degree to which the child experienced poverty	0.861	70.9%
Maternal education	Amount of education completed by the biological mother	0.963	34.3%
Homelessness	Degree of homelessness experienced by the child	0.814	2.9%
Single parenthood	Degree of single parenting with consideration of support	0.761	31.0%
Community violence	Degree of child exposure to community violence	0.972	5.2%
Neighbourhood risk	Extent to which the child resides in a risky neighbourhood based on FBI and census crime, education, and poverty indicators	N/A	N/A
Other environmental adversity	Other environmental ELA not captured by the existing subtypes	N/A	N/A
Relational (N = 14)		0.927	
Close familial death	Death of kin based on proximity of relation	0.806	11.6%
Caregiver substance use	Degree of caregiver substance use (i.e., alcohol, marijuana, street drugs)	0.896	20.0%
Caregiver health concern	Problem(s) that interferes with daily life/parenting for any length of time	0.791	31.6%
Current caregiver psychopathology	Degree to which the caregiver endorses mental health problems	0.959	23.8%
Divorce and/or separation	Degree of divorce and/or separation qualified by contact	0.623	20.8%
Caregiver incarceration	Caregiver incarceration in the life of the child qualified by contact	0.714	17.6%
Other caregiver separation	Other caregiver separation (e.g., deployment) qualified by contact	0.738	20.4%
Foster care involvement	Foster care involvement with consideration of duration and contact	0.947	8.8%
Physical abuse	Degree of harsh physical punishment and/or physical abuse of the child	0.895	12.4%
Sexual abuse	Degree of sexual maltreatment of the child	0.961	2.0%
Emotional abuse	Degree of harsh verbal punishment and/or emotional abuse of the child	0.946	6.9%
Domestic violence exposure	Degree of domestic violence exposure of the child	0.862	10.4%
Neglect	Degree of child neglect	0.874	8.8%
Other relational adversity	Other relational ELA not captured by the existing subtypes	N/A	N/A

Note: The severity of each ELA was coded from 0 (*no adversity*) to 3 (*severe adversity*). The percentage of participants with a non-zero score.

2.3.2 | Sleep

Adolescents completed the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) at ages 14 (~one year prior to the COVID-19 pandemic), 15 (early-pandemic in spring 2020), and 15.5 (later-pandemic in winter 2020). The PSQI is a well validated measure of sleep quality that is frequently used with sociodemographically diverse adolescent and adult samples (Larche et al., 2021). This study assessed relations from each ELA subtype to all seven of the components that comprise the PSQI (i.e., subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, daytime dysfunction, and use of medication to sleep). At ages 14 and 15.5, adolescents were asked to report their sleep experiences on a scale from 0 (not during the past month) to 3 (three or more times a month). At age 15, adolescents were asked to report their sleep experiences on the same scale, but we replaced “during the past month” with “during the past two weeks,” to capture sleep experiences before, during, and well into the COVID-19 pandemic. Higher scores on each sleep component indicated worse functioning (e.g., poorer sleep quality, more sleep disturbances, more daytime dysfunction). Of note, we did not analyse the global PSQI score for two reasons. First, global sleep patterns have already been documented among youth during COVID-19 (Okely et al., 2021; Varma et al., 2021). Second, researchers have discussed the importance of assessing different components of sleep to identify nuanced patterns that may be more informative for targeted prevention and intervention efforts (Bi & Chen, 2022).

2.3.3 | Perceived stress

Adolescents' reported on their perceived life stress at each COVID-19 assessment (i.e., ages 15 and 15.5) using the well-validated Perceived Stress Scale (PSS; Cohen et al., 1983). Adolescents rated the frequency with which they felt or thought a certain way during the past month (e.g., *How often have you been able to control irritations in your life?*) across 14 items on a 5-point scale from 0 (*never*) to 4 (*very often*). The PSS evidenced acceptable reliability in the current sample at both ages 15 ($\alpha = 0.809$) and 15.5 ($\alpha = 0.799$).

2.4 | Data analytic plan

Following preliminary descriptive and bivariate analyses, the primary study hypotheses were evaluated using the lavaan package in Rstudio (Rosseel, 2012). Data were missing at age 14 when 28 (16%) adolescents did not provide PSQI reports, at age 15 when 20 (11.4%) adolescents did not provide PSQI and/or PSS reports, and at age 15.5 when 15 (8.6%) adolescents did not provide PSQI and/or PSS reports. There were no significant differences between the 140 adolescents with COVID-19 sleep reports and the 35 without COVID-19 data on any of the study variables, nor with respect to sex assigned at birth and ethnicity-race.

Data for all 175 dyads who completed the initial age 4 ELA assessment and one or both COVID-19 data waves were retained in these analyses using Full Information Maximum Likelihood as supported by Little's (1988) missing completely at random test, $\chi^2(82) = 97.347$, $p = 0.12$. Path analyses evaluated each study hypothesis. In the path model, we regressed each sleep problem component at age 15 (i.e., spring 2020) and age 15.5 (i.e., winter 2020) onto each ELA subtype composite, while controlling for the corresponding prior sleep component, concurrent perceived stress, sex assigned at birth, and ethnicity-race (i.e., dichotomously coded as Latine or non-Latine). Acceptable model fit was determined using established cutoffs for several fit indices (i.e., CFI ≥ 0.90 , SRMR ≤ 0.08 ; RMSEA ≤ 0.06 ; Hu & Bentler, 1999). All ELA domains and sleep components were correlated as is the default in the lavaan package.

3 | RESULTS

3.1 | Descriptive and bivariate analyses

Descriptive statistics and bivariate correlations are shown in Table 2. A MANOVA revealed significant differences across study variables by child sex assigned at birth (Wilks' $\lambda = 0.751$, $p = 0.005$), but not by child ethnicity-race, nor their interaction. At age 14, girls reported more sleep disturbances ($M = 1.234$) than boys ($M = 0.967$). At age 15, girls reported more sleep disturbances ($M_{\text{girls}} = 1.281$, $M_{\text{boys}} = 0.917$) and perceived stress ($M_{\text{girls}} = 1.831$, $M_{\text{boys}} = 1.322$) than boys. At age 15.5, girls reported poorer sleep quality ($M_{\text{girls}} = 1.156$, $M_{\text{boys}} = 0.800$), more daytime dysfunction ($M_{\text{girls}} = 1.344$, $M_{\text{boys}} = 0.783$), and more perceived stress ($M_{\text{girls}} = 1.943$, $M_{\text{boys}} = 1.552$) than boys. Additionally, two sets of paired samples *t*-tests revealed significant differences in sleep component scores prior to the pandemic at age 14 and early in the pandemic at age 15, as well as at age 15 and later in the pandemic at age 15.5. Sleep quality and sleep latency scores at age 15 were poorer than at age 14. All mean scores at age 15.5 were significantly worse than at age 15, except for sleep latency scores, which were higher at age 15 than at age 15.5. Bivariate analyses indicated that biological and environmental ELA were positively associated with relational ELA. At age 15.5, relational ELA was associated with more sleep disturbances. Sleep problems were generally all positively related to one another within and across all data waves.

3.2 | Path analysis

Table 3 depicts parameter estimates and 95% bootstrapped confidence intervals (CIs) across 10,000 resamples for the final model with six of the seven sleep components. The model with all seven components yielded a poor fit to the data (CFI = 0.873, SRMR = 0.069, and RMSEA = 0.055). We decided to omit the sleep medication PSQI component because it was based on a single zero-inflated item (i.e., 91.7% and 83.1% of the adolescents in this sample denied any use of

TABLE 2 Descriptive statistics and bivariate correlations among study variables.

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26								
1. Biological ELA (age 4)	0.011	0.465	-																																	
2. Environmental ELA (age 4)	-0.024	0.426	0.124	-																																
3. Relational ELA (age 4)	-0.018	0.363	0.321**	0.390**	-																															
4. Sleep quality (age 14)	0.748	0.629	-0.127	-0.031	0.103	-																														
5. Sleep latency (Age 14)	0.815	0.839	0.112	-0.104	0.093	0.393**	-																													
6. Sleep duration (age 14)	0.224	0.549	-0.034	0.071	0.076	0.406**	0.330**	-																												
7. Sleep efficiency (age 14)	0.692	0.978	0.084	0.094	0.179*	0.082	0.184*	0.217*	-																											
8. Sleep disturbances (age 14)	1.102	0.533	-0.028	-0.033	0.072	0.363**	0.519**	0.419**	0.175*	-																										
9. Sleep medications (age 14)	0.204	0.661	0.135	-0.018	-0.014	0.108	0.204*	0.163	0.193*	0.193*	-																									
10. Daytime dysfunction (age 14)	0.565	0.768	-0.042	-0.084	0.066	0.325**	0.271**	0.322**	0.206*	0.444**	0.028	-																								
11. Sleep quality (Age 15)	0.987	0.827	0.016	-0.003	0.056	0.112	0.171*	0.196*	0.006	0.279**	0.098	0.213*	-																							
12. Sleep latency (age 15)	1.122	0.992	0.050	-0.117	0.094	0.193*	0.377**	0.071	-0.163	0.253**	0.185*	0.145	0.330**	-																						
13. Sleep duration (Age 15)	0.292	0.749	0.078	0.009	-0.036	0.152	0.078	0.346**	-0.047	0.226**	0.108	0.240**	0.265**	0.082	-																					
14. Sleep efficiency (age 15)	0.719	1.094	-0.051	0.055	-0.038	0.075	0.101	0.118	0.103	0.176*	0.060	0.284**	0.177*	0.127	0.344**	-																				
15. Sleep disturbances (age 15)	1.077	0.585	0.068	-0.096	-0.067	0.142	0.312**	0.233**	0.035	0.437**	0.234**	0.284**	0.282**	0.290**	0.243**	0.251**	-																			
16. Sleep medications (age 15)	0.179	0.627	0.088	0.061	0.039	0.199*	0.307**	0.202*	0.099	0.262**	0.380**	0.107	0.129	0.233**	0.177*	0.176*	0.243**	-																		
17. Daytime dysfunction (age 15)	0.673	0.796	0.022	-0.159*	-0.071	0.245**	0.144	0.142	0.044	0.162	-0.006	0.313**	0.336**	0.238**	0.288**	0.355**	0.260**	-																		
18. Perceived stress (age 15)	1.588	0.721	0.064	0.003	0.160*	0.282**	0.275**	0.272**	0.110	0.344**	0.051	0.328**	0.396**	0.281**	0.303**	0.308**	0.403**	0.154	-																	
19. Sleep quality (Age 15.5)	0.981	0.713	-0.059	0.029	0.162*	0.172*	0.171*	0.296**	0.213*	0.370**	0.216*	0.244**	0.407**	0.204*	0.172*	0.322**	0.244**	0.231**	0.235**	-																
20. Sleep latency (Age 15.5)	1.089	0.960	-0.114	0.045	0.033	0.134	0.288**	0.199*	0.033	0.333**	0.177*	0.130	0.345**	0.473**	0.105	0.268**	0.182*	0.178*	0.066	0.198*	0.356**	-														

TABLE 2 (Continued)

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	
21. Sleep duration (age 15.5)	0.277	0.630	-0.038	0.098	0.029	0.144	0.091	0.343*	0.183*	0.261**	0.107	0.304**	0.215*	0.010	0.325**	0.245**	0.263**	0.271**	0.282**	0.269**	0.487**	0.221**	-	-	-	-	-	-	-
22. Sleep efficiency (age 15.5)	0.619	1.023	-0.058	0.028	0.018	0.068	0.216*	0.330**	0.335**	0.310**	0.125	0.272**	0.152	0.012	0.130	0.340**	0.192*	0.245**	0.163	0.197*	0.306**	0.219**	0.567**	-	-	-	-	-	-
23. Sleep disturbances (age 15.5)	1.088	0.542	-0.028	0.108	0.235**	0.122	0.164	0.108	0.083	0.309**	0.183*	0.147	0.195*	0.078	0.072	0.035	0.375**	0.203*	0.074	0.280**	0.443**	0.306**	0.310**	0.164*	-	-	-	-	-
24. Sleep medications (age 15.5)	0.350	0.856	0.040	0.001	-0.055	0.112	0.044	0.125	0.113	0.057	0.237**	0.054	0.182*	0.171*	0.144	0.190*	0.270**	0.373**	0.177*	0.102	0.155	0.233**	0.222**	0.166*	0.178*	-	-	-	-
25. Daytime dysfunction (age 15.5)	1.025	0.971	-0.053	-0.137	0.061	0.235**	0.226**	0.489*	0.082	0.322**	0.116	0.321**	0.314**	0.261**	0.348**	0.233**	0.386**	0.176*	0.444**	0.460**	0.418**	0.237**	0.312**	0.255**	0.390**	0.194*	-	-	-
26. Perceived stress (age 15.5)	1.745	0.700	-0.092	0.033	0.150	0.320**	0.308**	0.295**	0.124	0.372**	-0.007	0.304**	0.401**	0.189*	0.228**	0.204*	0.358**	0.168*	0.408**	0.657**	0.386**	0.323**	0.359**	0.285**	0.402**	0.179*	0.565**	-	-

* $p < 0.05$, ** $p < 0.01$.

sleep medications at ages 15 and 15.5, respectively), which yielded a model with acceptable fit (i.e., CFI = 0.907, SRMR = 0.059, RMSEA = 0.052). The final model evaluated prospective relations of each ELA subtype at age 4 with each of the six sleep components at age 15 (i.e., early-pandemic in Spring 2020) and at age 15.5 (i.e., later-pandemic in winter 2020), while controlling for the corresponding pre-pandemic sleep component, concurrent perceived stress, sex assigned at birth (female = 1), and ethnicity-race (Latine = 1). Figure 1 displays the standardized coefficients for each pathway. Relational ELA predicted fewer sleep disturbances during the initial phase of the U.S. COVID-19 pandemic in spring 2020 ($b = -0.282$, $SE = 0.128$, $p = 0.028$, 95% CI [-0.554, -0.041]), but all other pathways were not significant. Mirroring the early-pandemic pattern, the only significant pathway predicting later-pandemic sleep problems during winter 2020 was from relational ELA to sleep disturbances. However, whereas relational ELA predicted decreased sleep disturbances early in the COVID-19 pandemic, relational ELA predicted increased sleep disturbances nine months into the pandemic in winter 2020 ($b = 0.322$, $SE = 0.138$, $p = 0.019$, 95% CI [0.095, 0.629]).¹

4 | DISCUSSION

This investigation contributes to ongoing efforts to understand how childhood experiences influence adolescent sleep components, particularly in contexts of both acute and chronic stress exposure. As hypothesised, relational ELA (e.g., child maltreatment, parental incarceration) explained unique variance in adolescents' sleep problems (i.e., sleep disturbances) following both short- and longer-term exposure to the COVID-19 pandemic, even beyond biological and environmental ELAs. Interestingly, the direction of this association switched such that relational ELA predicted fewer sleep disturbances during the initial phase of the U.S. COVID-19 pandemic in spring 2020, but, over time, predicted increased sleep disturbances as the pandemic persisted into the winter of 2020. The obtained findings were robust to several important covariates, including other ELA subtypes (i.e., biological and environmental) and contemporaneous perceived stress. This latter covariate highlights the special significance of early experience over and above current perceived stress for understanding adolescents' sleep patterns.

As anticipated, relational ELA was especially important for understanding adolescents' sleep problems during the COVID-19 pandemic. Sleep is a regulation-based phenomenon (Williams et al., 2016) and children's early relationships are particularly salient for entraining and maintaining adaptive self-regulation (Moilanen & Rambo-Hernandez, 2017), especially self-soothing, which is particularly important for sleep (Thomas et al., 2014). Although it is possible that relational ELAs emerged as more salient for later sleep problems because of their chronicity, many biological (e.g., child health problems) and environmental (e.g., poverty) ELAs were also chronic in nature. Therefore, we posit that the salience of relational ELAs may have been magnified by the unique context of COVID-19

TABLE 3 Path analysis from each early life adversity (ELA) subtype at age 4 to each sleep component at age 15 early in the pandemic (i.e., spring 2020) and at age 15.5 later into pandemic (i.e., winter 2020).

Sleep during COVID-19 Spring 2020 (Age 15)						
Variable	Sleep quality		Sleep Latency		Sleep duration	
	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)
Biological ELA	-0.014 (0.160)	0.929 (-0.312, 0.314)	-0.053 (0.202)	0.795 (-0.439, 0.349)	0.196 (0.127)	0.125 (-0.043, 0.454)
Environmental ELA	-0.012 (0.147)	0.934 (-0.280, 0.302)	-0.301 (0.180)	0.094 (-0.654, 0.054)	0.097 (0.138)	0.483 (-0.133, 0.411)
Relational ELA	0.015 (0.180)	0.935 (-0.330, 0.385)	0.273 (0.226)	0.227 (-0.185, 0.699)	-0.351 (0.173)	0.043 (-0.703, -0.0024)
Prior sleep (Age 14)	0.033 (0.115)	0.775 (-0.193, 0.263)	0.361 (0.103)	<0.001 (0.158, 0.562)	0.377 (0.196)	0.054 (-0.003, 0.767)
Sex (Female = 1)	0.253 (0.133)	0.057 (-0.008, 0.517)	0.158 (0.156)	0.313 (-0.140, 0.470)	-0.041 (0.118)	0.727 (-0.288, 0.180)
Ethnicity-race (Latine = 1)	0.127 (0.123)	0.303 (-0.115, 0.365)	0.181 (0.150)	0.226 (-0.113, 0.475)	-0.028 (0.119)	0.815 (-0.263, 0.203)
Perceived stress (Age 15)	0.393 (0.085)	<0.001 (0.232, 0.563)	0.225 (0.119)	0.058 (-0.025, 0.441)	0.276 (0.118)	0.019 (0.058, 0.517)
Daytime Dysfunction						
Variable	Sleep Efficiency		Sleep Disturbance		Daytime Dysfunction	
	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)
Biological ELA	-0.112 (0.200)	0.577 (-0.506, 0.286)	0.164 (0.115)	0.153 (-0.081, 0.371)	0.068 (0.139)	0.625 (-0.228, 0.323)
Environmental ELA	0.320 (0.247)	0.195 (-0.148, 0.824)	-0.035 (0.101)	0.729 (-0.248, 0.151)	-0.157 (0.120)	0.189 (-0.405, 0.068)
Relational ELA	-0.425 (0.263)	0.106 (-0.956, 0.087)	-0.282 (0.128)	0.028 (-0.554, -0.041)	-0.305 (0.178)	0.086 (-0.630, 0.066)
Prior sleep (Age 14)	0.090 (0.130)	0.488 (-0.157, 0.351)	0.359 (0.088)	<0.000 (0.179, 0.524)	0.149 (0.098)	0.128 (-0.051, 0.335)
Sex (Female = 1)	0.028 (0.192)	0.885 (-0.353, 0.395)	0.105 (0.087)	0.224 (-0.061, 0.280)	-0.025 (0.109)	0.818 (-0.245, 0.187)
Ethnicity-race (Latine = 1)	-0.223 (0.181)	0.219 (-0.587, 0.125)	0.007 (0.084)	0.938 (-0.161, 0.170)	0.078 (0.101)	0.436 (-0.120, 0.277)
Perceived stress (Age 15.5)	0.488 (0.127)	<0.001 (0.231, 0.725)	0.231 (0.060)	<0.001 (0.114, 0.350)	0.589 (0.083)	<0.001 (0.427, 0.753)
Sleep during COVID-19 Spring 2020 (Age 15.5)						
Variable	Sleep Quality		Sleep Latency		Sleep Duration	
	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)
Biological ELA	-0.083 (0.110)	0.448 (-0.289, 0.144)	-0.176 (0.158)	0.265 (-0.495, 0.127)	-0.020 (0.092)	0.826 (-0.195, 0.168)
Environmental ELA	-0.011 (0.137)	0.933 (-0.285, 0.249)	0.308 (0.169)	0.068 (-0.026, 0.645)	0.173 (0.113)	0.126 (-0.027, 0.418)
Relational ELA	0.243 (0.164)	0.139 (-0.089, 0.562)	-0.227 (0.186)	0.223 (-0.605, 0.137)	-0.115 (0.161)	0.473 (-0.447, 0.190)
Prior sleep (Age 14)	0.227 (0.089)	0.010 (0.054, 0.402)	0.444 (0.071)	<0.001 (0.306, 0.585)	0.222 (0.113)	0.049 (-0.002, 0.449)
Sex (Female = 1)	0.080 (0.111)	0.468 (-0.131, 0.300)	0.100 (0.138)	0.468 (-0.173, 0.365)	-0.002 (0.092)	0.985 (-0.191, 0.169)
Ethnicity-race (Latine = 1)	-0.136 (0.096)	0.159 (-0.324, 0.050)	-0.124 (0.132)	0.349 (-0.387, 0.129)	-0.174 (0.088)	0.049 (-0.355, -0.003)
Perceived stress (Age 15)	0.249 (0.076)	0.001 (0.100, 0.398)	0.308 (0.106)	0.004 (0.514, 0.308)	0.265 (0.094)	0.005 (0.089, 0.453)

TABLE 3 (Continued)

Variable	Sleep Efficiency		Sleep Disturbance		Daytime Dysfunction	
	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)	b (Bootstrapped SE)	p (95% CI; bias-corrected)
Biological ELA	-0.021 (0.170)	0.900 (-0.345, 0.323)	-0.120 (0.086)	0.162 (-0.286, 0.053)	-0.015 (0.145)	0.918 (-0.281, 0.288)
Environmental ELA	0.128 (0.212)	0.545 (-0.273, 0.564)	0.056 (0.102)	0.581 (-0.135, 0.272)	-0.285 (0.173)	0.099 (-0.615, 0.067)
Relational ELA	-0.111 (0.352)	0.752 (-0.832, 0.537)	0.322 (0.138)	0.019 (0.095, 0.629)	0.141 (0.208)	0.499 (-0.276, 0.545)
Prior sleep (Age 14)	0.203 (0.091)	0.025 (0.024, 0.379)	0.559 (0.077)	0.001 (0.112, 0.418)	0.305 (0.104)	0.003 (0.101, 0.506)
Sex (Female = 1)	0.275 (0.165)	0.096 (-0.059, 0.602)	-0.114 (0.076)	0.134 (-0.265, 0.033)	0.247 (0.127)	0.053 (0.007, 0.509)
Ethnicity-race (Latine = 1)	-0.284 (0.159)	0.074 (-0.598, 0.029)	-0.045 (0.075)	0.550 (-0.191, 0.109)	-0.196 (0.120)	0.102 (-0.432, 0.039)
Perceived stress (Age 15.5)	0.297 (0.128)	0.020 (0.044, 0.549)	0.225 (0.060)	<0.001 (0.104, 0.338)	0.595 (0.107)	<0.001 (0.372, 0.791)

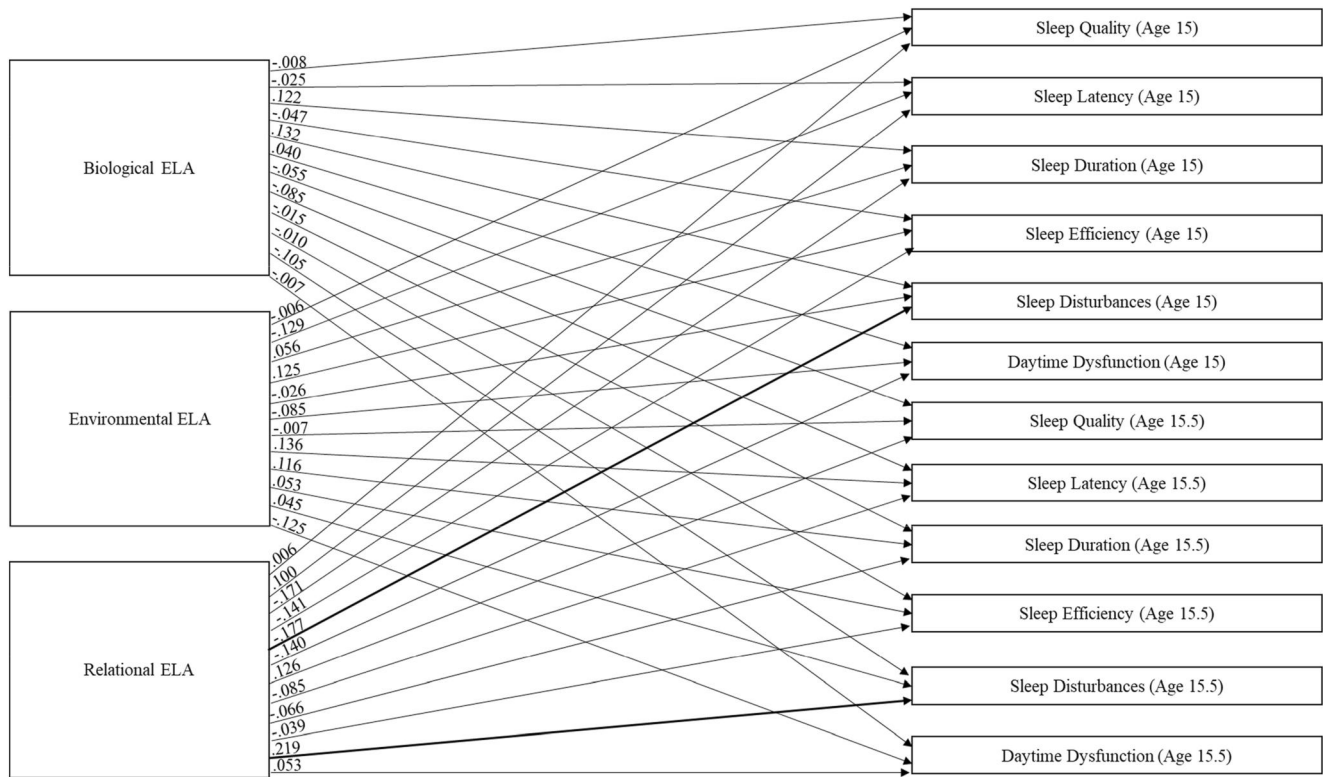


FIGURE 1 A path analysis evaluating prospective relations of each early life adversity (ELA) subtype at age 4 with each sleep component at age 15 (i.e., early in the U.S. COVID-19 pandemic during spring 2020) and at age 15.5 (i.e., later in the U.S., Covid-19 pandemic during winter 2020). Covariates (not shown for clarity) include prior sleep component levels, concurrent perceived stress, youth sex assigned at birth, and youth ethnicity-race. Pathways depict standardized coefficients with significant relations in bold.

restrictions, which led to heightened relational contact as a function of stay-at-home orders. For example, given age-appropriate relational (re)negotiations of autonomy and relatedness during adolescence (Kobak et al., 2017), as well as the distinctly disruptive impact of COVID-19 pandemic restrictions on these processes (Bülow et al., 2021), prior relational vulnerabilities may have been activated by the specific nature of the COVID-19 stressor. Indeed, most adolescents encountered developmentally-atypical increases in parental contact during the pandemic at the same time their access to peers and potentially compensatory relationships with outside adults (e.g., teachers, coaches) declined. Thus, COVID-19 restrictions prompted intensive exposure to the very things that may be most negatively affected in the context of relational ELA—family relationships. Indeed, there is mounting evidence that families already experiencing negative interactions prior to the pandemic tended to have increased difficulty adjusting to the pandemic (Qu et al., 2021; Sun et al., 2021). Additionally, that our only significant finding was from relational ELA to later sleep disturbances is in line with previous research regarding child maltreatment (e.g., Greenfield et al., 2011). In this work, child abuse predicted sleep disturbances, but not any other sleep component which may be indicative of the long-term psychobiological consequences of relational ELAs that are uniquely captured by the sleep disturbances component (e.g., bad dreams). Of note, child maltreatment was subsumed within relational ELAs.

The differential association of relational ELA to adolescents' acute versus longer-term sleep responses across the first year of the COVID-19 pandemic was somewhat surprising. Early in the pandemic, adolescents with more severe histories of relational ELA reported relatively positive sleep outcomes as indicated by significantly fewer sleep disturbances. This pattern is consistent with prior theories of steeling (Rutter, 1985) and stress inoculation (Parker et al., 2004), wherein ELA can engender subsequent resilience to the deleterious effects of stress. However, prior evidence supporting such effects have typically examined less severe adversity exposure than the current study. Further, such processes cannot explain the directional switch observed here, wherein relational ELA predicted increases in adolescents' longer-term sleep disturbances as the pandemic wore on. This predictive reversal may reflect processes of burnout, wherein prior encounters with adversity can support initial coping with a subsequent stressor, but ultimately undermine coping persistence in the face of ongoing stress. Although pandemic expressions of burnout have been seen among healthcare workers (Talaee et al., 2020), parents (Marchetti et al., 2020), and teachers (Pressley, 2021), less work has examined this phenomenon among adolescents (Moroñ et al., 2021), and no studies have considered if and how ELA may influence these patterns. As seen here, relational ELA may have simultaneously bolstered adolescents' sleep responses to early-pandemic stress yet accelerated burnout processes over the

longer-term. That said, as noted earlier, the COVID-19 pandemic represents a unique relational stressor, such that ongoing work is needed to understand how ELA subtypes influence adolescents' sleep responses to other kinds of short- and longer-term stressors. Additionally, while stress exposure duration may be one mechanism underlying the appearance of steeling versus sensitivity effects in this study, it is important to recognize the multiplicity of shifting factors across the pandemic that could have influenced these relations. For example, variations in school instruction (Hertz et al., 2022), social support (Christ & Gray, 2022), and coping styles (Wang et al., 2022) may have influenced the obtained results in ways that warrant further consideration.

4.1 | Strengths and limitations

The current study features several strengths that advance our understanding of ELA and adolescent sleep problems throughout the COVID-19 pandemic. First, we harnessed prospective data across several waves from the preschool period through adolescence and both early- and longer-term COVID-19 assessments while controlling for prior levels of sleep problems and concurrent perceived stress. These design elements support a stronger degree of directional inference than previous cross-sectional and abbreviated longitudinal research designs. Second, our use of multiple informants (i.e., caregivers and adolescents) and methods (i.e., semi-structured interviews and surveys) mitigates concerns about biased estimates due to shared variance. Third, in contrast to past ELA research, we used a comprehensive array of biological, environmental, and relational ELA composites encompassing a wider range of adverse life experiences than prior studies. Fourth, our preschool assessment of ELA captured caregiver reports of children's experiences across the first 4 years of life with a time span that reduced the risk of memory recall issues, which feature prominently in most studies using retrospective ELA reports. Notwithstanding these strengths, several limitations qualify our findings while highlighting promising directions for future research.

First, although our measure of ELA accounted for both severity and subtype, these analyses did not consider how biological, environmental, and relational ELAs may interactively affect adolescents' sleep components. Given known correlations across ELA experiences (including in the current study), evaluating interactive patterns, or considering alternate analytic models, such as person-oriented approaches, may prove fruitful in future research. Relatedly, although we considered prominent subtypes of ELA in this study, there may be other meaningful distinctions, such as experiences of threat versus deprivation (Sheridan & McLaughlin, 2014), or perhaps no meaningful distinctions at all (Smith & Pollak, 2021). Relatedly, in addition to comorbidity, the chronicity of ELA exposure has shown unique effects on development and adaptation from those of ELA severity (Manly et al., 1994). As noted earlier, the current study was particularly well-suited to evaluate ELA severity, but future work examining adversity exposure over longer time periods may meaningfully probe for unique chronicity effects on adolescent sleep patterns.

Third, despite considering multiple components of sleep problems, our study would have benefited from additional, objective measures of sleep behaviour, such as actigraphy data (Lucas-Thompson et al., 2021). Although research points to different levels of (in)accuracy across sleep measurement devices (Burkart et al., 2021), some evidence suggests that sleep data using actigraphy are comparable to self-reports among community samples of adolescents, at least with regard to sleep duration (Lucas-Thompson et al., 2021). Future investigations of ELA and adolescent sleep problems will benefit from a multi-method approach to measuring a variety of sleep features. Relatedly, we were unable to assess potential curvilinear changes in sleep disturbance as adolescents transitioned into and across the COVID-19 pandemic. Examining curvilinear changes in sleep disturbance across more than three timepoints would be a meaningful contribution to the literature.

Finally, although intriguing, the obtained findings warrant replication across other types of outcomes beyond sleep, contextual stressors beyond the COVID-19 pandemic, and periods of development beyond adolescence. In particular, given the unique relational features of the COVID-19 pandemic, it is important that future investigations determine whether the obtained findings will generalise to other types of short- and longer-term stressors.

4.2 | Implications

The current investigation represents an important contribution to the growing literature regarding adolescent sleep problems during the COVID-19 pandemic. Although some have questioned the value of ELA subtypes (Smith & Pollak, 2021), this study highlights the salience and utility of considering ELA subtypes, such as biological, environmental, and relational, for understanding youth's responses to future stressors. The current findings also illuminate the need for ongoing research efforts to understand how the duration of stress exposure may influence developmental relations between prior adversity and future adjustment. Thus far, studies of adolescents' sleep patterns during the COVID-19 pandemic have focused on patterns before and during the COVID-19 pandemic responses to future stressors. The current findings also illuminate the need for ongoing research efforts to understand how the duration of stress exposure may influence developmental relations between prior adversity and future adjustment. Thus far, studies of adolescents' sleep patterns during the COVID-19 pandemic have focused on patterns before and during the COVID-19 pandemic (Becker et al., 2021; Bruni et al., 2022; Santos & Louzada, 2022). Further research is needed to better understand how prior experiences may influence these patterns (e.g., ELA), as well as the extent to which they persist throughout the pandemic and beyond.

Moving forward, research is needed to examine both ELA subtypes and stress exposure chronicity in the context of other outcome domains, other stressors, and other periods of the life course. Beyond the levels of analysis here, future studies may also incorporate broader system effects and activities to refine our understanding of

adolescent sleep during COVID-19. For example, differential school responses to widespread stressors, such as the COVID-19 pandemic, might impact relations between ELA and adolescent sleep patterns. In the context of the COVID-19 pandemic, associations between ELA and sleep patterns may have been exacerbated early in the pandemic if schools were unable to adequately pivot to effective online or hybrid instruction. Further, as the pandemic progressed, some schools may have been better resourced to support students and restore a normal educational routine, which, in turn, may have stabilised their sleep patterns. As in the present study, such nuanced investigations may challenge the unilateral presumption that ELA-exposed youth are more vulnerable to negative outcomes in the face of subsequent challenges.

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CONFLICT OF INTEREST STATEMENT

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

DATA AVAILABILITY STATEMENT

Research data are not shared.

ETHICS STATEMENT

Research data available upon request.

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ENDNOTE

¹ We also considered two additional models. First, we replaced the individual sleep component scores with PSQI total scores across ages 14, 15, and 15.5. Fit indices were mixed (i.e., CFI = 0.938, SRMR = 0.021, RMSEA = 0.142) and, similar to our initial findings, relational ELA remained the only (negative) significant predictor ($p = 0.041$) of PSQI total scores at age 15, but not at age 15.5. Second, we replaced our ELA variables with threat (i.e., physical abuse, sexual abuse, domestic violence, and community violence) and deprivation (i.e., poverty, death of a close relative, and neglect) variables when predicting the individual sleep component scores. Although this model fit the data well (i.e., CFI = 0.972, SRMR = 0.043, RMSEA = 0.037), we received a warning that 375 bootstraps failed to run, which gives us pause regarding the validity of the final model. In this second model, there was only one significant (negative) prediction from deprivation to daytime dysfunction ($p = 0.035$) at age 15, but not age 15.5

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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