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Research paper

Does telomere length mediate the association between early life adversity and mental health in childhood?

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ABSTRACT

Background: Early-life adversity (ELA) increases the likelihood of developing depression and anxiety. Telomere length, a marker of biological aging, tends to shorten in response to oxidative stress and inflammation, which may play a role in the relationship between ELA and mental health.
Methods: Growing Up in New Zealand longitudinal study data was used to explore ELA, including parental, household, and environmental variables to determine whether child telomere length mediates associations between prenatal and postnatal adversity and offspring mental health (n = 3117, 47.7 % female). ELA measures were collected during pregnancy and when children were nine-months and four years old. Seventeen possible adverse experiences were categorized as low (0–4), moderate (5–7), and high (8+). At age four years, telomere length was measured by qPCR with DNA extracted from saliva. At age eight, children self-reported depression and anxiety symptoms.
Results: ELA was positively associated with depression and anxiety symptoms and telomere length. There was a direct effect of ELA on depression (β = 0.25, 95 % CI [0.14–0.37]), anxiety (β = 0.22, 95 % CI [0.07–0.37]), and telomere length (β = 0.03, 95 % CI [0.002–0.067]). There was no direct effect of telomere length on depression (β = 0.09, 95 % CI [−0.04–0.21]) or anxiety (β = 0.08, 95 % CI [−0.09–0.24]) and telomere length did not mediate the adversity-psychosocial relationships.
Conclusion: ELA influences children's mental well-being and telomere length. Surprisingly, telomere length was longer following ELA, which may be a compensatory mechanism to regulate the effects of accelerated telomere attrition in response to adversity. Further research is needed to understand these complex and dynamic relationships across the life-course.

1. Introduction

Anxiety and depression are two of the most prevalent mental health conditions worldwide, the onsets of which often arise in childhood and adolescence (Kessler et al., 2012; Solmi et al., 2022). In New Zealand (NZ), rates appear to be increasing, with approximately 20 % of young people experiencing mental health problems (Sutcliffe et al., 2023; Fleming et al., 2014). These internalizing disorders are associated with poorer outcomes throughout life, including lower educational attainment and self-esteem, family and peer dysfunction, poorer physical

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health, and higher levels of crime, anti-social behavior, self-harm, and suicide (McDougall, 2011; Farrell and Barrett, 2007). Early developmental periods are highly sensitive and adaptable to external exposures, potentially leading to long-lasting effects on an individual's developmental trajectory.

The Developmental Origins of Health and Disease hypothesis suggests that interactions between developmental processes and environmental exposures during sensitive periods of growth influence health and disease in adulthood (Gluckman et al., 2010; Hanson and Gluckman, 2008; Mandy and Nyirenda, 2018). Early life provides a critical window of susceptibility to negative effects of adversity due to rapid development and biological programming during this time (Räikkönen et al., 2012; Turner, 2018). Brain development is particularly sensitive, as early neuroplasticity allows adaptation to environmental exposures. However, this early life plasticity can have long-term physiological and psychological consequences (Räikkönen et al., 2012; Darling et al., 2020).

Greater exposure to early-life adversity (ELA) may exacerbate the onset and severity of mental health conditions. ELA refers to any severe and/or chronic social or environmental stressor between infancy and childhood with long-lasting psychological or physiological effects (Räikkönen et al., 2012; Darling et al., 2020; Merz and Turner, 2021; Matvienko-Sikar et al., 2021; Woo Baidal et al., 2016; Friedman et al., 2015). ELA is associated with increased morbidity and mortality and the development of non-communicable diseases (NCDs) such as cardiovascular disease, diabetes, cancer, inflammation, and psychosocial outcomes (Darling et al., 2020; Merz and Turner, 2021; Friedman et al., 2015; McLaughlin, 2016; Pechtel and Pizzagalli, 2011; Berg, 2016; Gibb et al., 2012). Frequent or chronic stress may influence physiological responses, leading to physiological and biological alterations and subsequent poorer health (Friedman et al., 2015). Severe stress resulting in elevated cortisol can cause permanent structural and functional brain changes, which could partly explain future social, emotional and physiological problems (Berg, 2016). Despite evidence between ELA and negative outcomes, the pathophysiological and mechanistic pathways remain poorly understood.

One potential pathway is the fetal programming of telomere biology hypothesis, which suggests that telomere length at birth and the rate of telomere attrition is highly plastic with sensitivity to early life experiences and may be linked to health status in later life (Entringer et al., 2018; Shalev and Belsky, 2016). Shorter telomere length is associated with the development of NCDs, abnormalities in brain structure and functioning, and earlier mortality (Bosquet Enlow et al., 2020). Notably, shorter telomere length has also been associated with psychiatric disorders, including depression and anxiety (Darrow et al., 2016). Furthermore, environmental factors leading to greater oxidative stress can negatively impact telomere length and/or attrition and the development of health and well-being outcomes, including NCDs and age-related diseases (Gavia-García et al., 2021; Barnes et al., 2019; Coluzzi et al., 2014; Rius-Ottenheim et al., 2012; Houben et al., 2008; Starkweather et al., 2014; Shalev et al., 2013; Shalev, 2012; Rizvi et al., 2014; Wang et al., 2018; Von Zglinicki, 2000). For example, stress, smoking, alcohol intake, poor diet, physical inactivity, and childhood adversities have been associated with telomere length and telomere attrition (Gorenjak et al., 2020; Balan et al., 2018; Coimbra et al., 2017; Clemente et al., 2019). Childhood adversities can include social interactions, poverty, family disruption and psychosocial factors (Gorenjak et al., 2020; Coimbra et al., 2017; Ly et al., 2019; Ridout et al., 2018a). Children and adolescents from disadvantaged neighborhoods typically have shorter telomeres than those less deprived (Coimbra et al., 2017; Coimbra et al., 2020). Exposures during intrauterine development can also influence telomere length. For example, fetal telomere attrition may be influenced during pregnancy by perceived stress and psychosocial stress, body mass index (BMI), gestational diabetes and smoking (Gorenjak et al., 2020). Thus, it is critical to assess markers of adversity across multiple developmental periods, as stress exposure may biologically accumulate over time (Ridout et al., 2018a). Stress and telomere shortening may be associated with endocrine and immune systems, mitochondrial dysfunction, and oxidative stress (Shalev, 2012), that may create a feedback loop that is especially relevant in stress-related conditions like depression and anxiety (Pousa et al., 2021). It is also important to consider that while experiencing early-life adversity may influence mental health outcomes, the biological response may differ between individuals. If telomere length reflects cumulative biological stress, then both the biological and environmental markers of stress may influence mental health trajectories in childhood. The association between early-life adversity and health later in life are likely driven by underlying biological pathways, with telomere length suggested as a potentially adaptive factor regulating biological aging that may mediate the effects of early life adversity (Shalev and Belsky, 2016; Haussmann and Marchetto, 2010).

Our study investigated whether telomere length, as a biological marker of stress exposure, mediates the association between ELA and psychosocial outcomes in childhood. Understanding this relationship is important because telomere length may be a biomarker of psychological and physical health risk among children who experience ELA. We had three hypotheses: 1) ELA is negatively associated with telomere length at four-years of age; 2) telomere length at four-years of age is negatively associated with psychosocial outcomes at eight-years of age; and 3) telomere length at four-years of age partly explains the association between ELA and psychosocial outcomes at eight-years of age.

2. Methods

2.1. Participants

Growing Up in New Zealand (GUiNZ) longitudinal study data was utilized (Morton et al., 2013). GUiNZ collected information from 6853 children born between April 2009 and March 2010 across three district health regions (Auckland, Counties Manukau, Waikato). The cohort broadly represents New Zealand's ethnic and socio-demographic birth population (Morton et al., 2015). Data from children and their families have been collected at several timepoints from before birth to eight-years of age. Families could consent to and provide biological samples from their participant child. Informed consent included the collection, use, and storage of biological samples and subsequent data. Data collection waves (DCWs) occurred during pregnancy and post-partum when cohort children were: nine-months, four-years, and eight-years of age. Data was collected through face-to-face interviews, telephone interviews, electronic questionnaires, and health records.

2.2. Measures

2.2.1. Covariates

Variables of interest included maternal BMI (pre-pregnancy height and weight), parity (first or subsequent pregnancy), maternal education (highest educational qualification), maternal age, child's biological sex (health records), child's birth weight, and child's ethnicity (parent-reported) at the four-year DCW. Adversity related variables measured at only one DCW were included as covariate variables, to ensure that adversity scores for each time point were approximately equally weighted. Antenatal covariates included maternal perceived stress (1–10; (Cohen et al., 1983)) and alcohol intake (any alcohol, stopped in pregnancy, and no alcohol consumed). Nine-month household air quality (derived from questions on condensation, dampness, and mold) and four-year parenting style (authoritative, authoritarian and permissive; (Robinson et al., 1995)) were also investigated.

2.2.2. Early-life adversity measures

Adversity measures were categorized as either maternal or household and environmental stressors (Supplementary Tables 6 and 7). Maternal variables included relationship status, perceived general

health (Ware et al., 1993), employment status, anxiety (GAD-7; (Spitzer et al., 2006)), depression (Edinburgh Postnatal Depression Scale; (Cox et al., 1987)), interparental physical conflict (Women's Abuse Screening Tool; (Brown et al., 1996)), interparental verbal conflict (from Resilience in Stepfamilies Study; (Pryor, n.d.)), maternal warmth and hostility (from the Iowa Family Interaction Rating Scale; (Melby et al., 1989)), and maternal racial discrimination (Morton et al., 2013; Thayer et al., 2023). Household and environmental adversity variables included NZ area-level deprivation (NZDep2013; (Atkinson et al., 2014)), household crowding, household tenure, household income, income-tested benefits (Statistics New Zealand, 2008), household smokers, family stress (Dunst et al., 1984), family support (Family Adaption and Cohesion Scale; (Olson et al., 1985)), and external support (Parenting Social Support Scale; (Dunst et al., 1984)).

2.2.3. Anxiety symptoms

The 8-item pediatric PROMIS anxiety scale (v2 8a) was used to measure children's self-reported anxiety symptoms at eight-years of age (Irwin et al., 2010). The scale is designed to assess anxiety symptoms in young people (aged 6–17 years) over the last seven days. Participants rated items on a five-point Likert-type scale (1 = *Almost never*, 2 = *Rarely*, 3 = *Sometimes*, 4 = *Often*, 5 = *Almost always*). Sum scores (range 8–40) were converted to T-Scores (33.5–83.3). Higher scores indicate greater anxiety symptoms and can indicate severity that is *within normal limits* (<50), *mild* (Spitzer et al., 2006; Cox et al., 1987; Brown et al., 1996; Pryor, n.d.; Melby et al., 1989; Thayer et al., 2023), *moderate* (Thayer et al., 2023; Atkinson et al., 2014; Statistics New Zealand, 2008; Dunst et al., 1984; Olson et al., 1985; Irwin et al., 2010; Andresen et al., 1994; Cawthon, 2009; Schoon et al., 2003; Shonkoff et al., 2012; Nurius et al., 2015) or *severe* (>65).

2.2.4. Depression symptoms

The 10-item self-report Center for Epidemiological Studies Depression Scale for Children (CES-DC-10) was used to measure depression symptoms at age eight (Andresen et al., 1994). Items are rated on a four-point Likert-type scale from zero to three (0 = *Not at all*, 1 = *A little*, 2 = *Some*, 3 = *A lot*). Sum scores range from zero to 30, with higher scores indicating greater depression symptoms (≥10 suggesting significant levels of disorder).

2.3. Biological sample collection

2.3.1. Saliva sample collection

For the child sample, non-invasive saliva samples (n = 4395) were collected via the passive drool method or kit sponge provided (buccal swab; n = 610) using the Oragene DNA-assisted collection 575 kits per manufacturer's protocol at the four-year DCW. Saliva samples were immediately combined with a stabilizing fluid, barcoded, and stored at room temperature until DNA extraction. DNA was extracted and purified using the PrepIT-L2P solution (DNA GenoTek). DNA samples were resuspended in TE buffer (10 mM Tris, 1 mM EDTA, pH 8.0) and stored at −80 °C.

2.3.2. Relative telomere length

The method was adapted from Cawthon (2009) and optimized, as described previously for this cohort (Ly et al., 2019). Briefly, relative telomere length was determined by quantitative PCR run in triplicate for accuracy. The relative telomere length is expressed as a T/S ratio equivalent to the average telomere length in each cell. A T/S ratio > 1 indicates that the sample has a longer average telomere length, and < 1 indicates that the sample has a shorter average telomere length than that of the standard DNA. As the DNA obtained from saliva samples contains a mixed cell population, the telomere length measured represents the mean of all cells within the sample. The primer sequences (5′ → 3′) for telomere amplification were telg (ACACTAAGGTTTGGGTTTGGGTTTGGGTTTGGGTTAGTGT) and telc (TGTTAGGTATCCCTATCCCTATCCCTATCCCTATCCCTAACA); for amplification of the single copy reference gene albumin were albu (CGGCGGCGGGCGGCGCGGGCTGGGCGGAAATGCTGCACAGAATCCT) and albd (GCCCGGCCCGCCGCGCCCGTCCCGCCGGAAAAGCATGGTCGCCTGTT). The qPCR reaction consisted of 7.5 μl of a 2× master mix (SYBR FAST qPCR Kit, Kapa BioSystems), 0.5 μM of each primer (telg, telc, albu, albd) and ~ 10 ng of extracted DNA to yield a final reaction volume of 15 μl. Each plate contained a 6-point standard curve from 0.05 ng to 10 ng of genomic DNA. qPCR was performed in a Roche LightCycler 480 II, and the thermal cycling conditions were 1 cycle of 95 °C for 20 s and 40 cycles of 95 °C for 1 s, 56 °C for 20 s, 72 °C for 1 s with signal acquisition, and 88 °C for 1 s with signal acquisition. Threshold cycle (C_t) values were calculated from the raw qPCR data using the R package qpcR. Samples with a coefficient of variation greater than 2.5 % were repeated. The coefficient of variation was 0.98 % for the telomere assay and 0.78 % for the reference assay. The average amplification efficiency was 99.87 % for the telomere assay and 99.40 % for the reference assay. The T/S ratio was calculated using the -ΔΔC_t formula. E_{tel} and E_{alb} are the amplification efficiency of the telomere and albumin sequences, respectively. $C_t$tel-standard is the C_t value for the telomere sequence of the standard DNA, $C_t$tel-sample is the C_t value for the telomere sequence of the sample DNA, $C_t$alb-standard is the C_t for the albumin sequence of the standard DNA, and $C_t$alb-sample is the C_t for the albumin sequence of the sample DNA.

$$T/S = \frac{E_{tel}(C_t tel - standard - C_t tel - sample)}{E_{alb}(C_t alb - standard - C_t alb - sample)}$$

2.4. Statistical analysis

2.4.1. Data screening

The biological cohort (n = 4340) telomere (T/S) ratio distribution was assessed to identify outliers and determine normality (Supplementary Fig. 1A–D). Overall, 55 outliers (+/− 3SD) were removed. Variables were assessed for normality, distributions and proportions. Transformed variables included telomere length ($\log^2$), anxiety and depression symptoms (Box-cox). Categorical variables were tested for homogeneity of variance, and continuous variables were tested for homoscedasticity. Missing data, longitudinal attrition and bias were investigated (Table 2 and Supplementary Tables 1–3). The biological cohort did not demonstrate attrition bias for child sex, maternal age, or maternal education. Bias was observed for ethnicity and areal-level deprivation.

2.4.2. Early-life adversity

Complete cases were utilized in the adversity scores and subsequent modelling. ELA was calculated using variables that met the inclusion criteria outlined in Table 1 (Supplementary Fig. 2 and Table 4). Adversity variables included maternal relationship status, area-level deprivation, household crowding, household tenure, receiving an income-tested benefit and household smoking. Adversity variables were then dichotomized (0 = none/low adversity; 1 = moderate/high adversity) according to distributions and association with telomere length and summed into an adversity score (antenatal, nine-months and four-years). Variables with multi-level responses were dichotomized.

Table 1
Early-life adversity variable inclusion and exclusion criteria used for adversity scores.

Inclusion criteria	Exclusion criteria
<10 % missing data	Present at only one DCW - classed as covariates
Collected at multiple DCWs	Repeated measures that only met inclusion criteria at one DCW became a covariate
Significantly associated with telomere length ($p < .05$) at one timepoint and a minimum of a trend association at another time point ($p < .1$)	

The distribution and association with telomere length at four-years are presented for all variables (Supplementary Table 8). Adversity scores from each DCW were combined to create a raw quantitative ELA score. The R^2, AIC, BIC and *p*-value of the raw quantitative model and a grouped quantitative model (ELA was categorized into low (0–4), moderate (McDougall, 2011; Farrell and Barrett, 2007; Gluckman et al., 2010) and high (8+) groups) were evaluated and compared with psychosocial outcomes and telomere length.

2.4.3. Modelling

The association between adversity variables and telomere length were assessed using univariate ANOVA or Kruskal-Wallis tests. Significant categorical variables with three or more levels underwent Tukey's Honestly Significant Difference (HSD) testing. Where appropriate, continuous variables significantly associated with telomere length were converted to categorical variables. Groups were determined by literature recommendations or by using quantiles (to maintain sub-group statistical power). Linear regression tested the association between telomere length and psychosocial outcomes. The grouped adversity model was numeric in statistical analyses to assess the additive effect of adversity on telomere length and psychosocial outcomes. Interaction analyses were performed between telomere length and covariates on both psychosocial outcomes. Covariates were tested for correlation and multicollinearity using a variance inflation factor (VIF; < 5) and eigenvalues prior to modelling. Four mediation analyses (with and without the inclusion of covariates) were performed to test the association between ELA and psychosocial outcomes with telomere length as the mediator. Mediation analyses were conducted in R using Lavaan and LavaanPlot packages.

3. Results

3.1. Sample

The demographics of the main ($n = 6853$) and biological cohort with questionnaire data across all time points ($n = 3812$) were compared using z-tests (Table 2). Families could skip DCWs, and 282 children opted out of the study. The final sample for modelling was 3117 after the removal of incomplete cases for all adversity variables, covariates, biological data, and psychosocial outcomes.

3.2. Sample characteristics and telomere length

Summary statistics and univariate association analyses with telomere length are reported in Supplementary Tables 5–7. Child factors significantly associated with telomere length included sex assigned at birth ($\eta^2 = 0.0047$, $p < .001$) and ethnicity ($\eta^2 = 0.0258$, $p < .001$). Antenatal maternal covariates associated with telomere length included age ($\eta^2 = 0.0014$, $p = .01$), alcohol consumption ($\eta^2 = 0.0030$, $p = .002$), perceived stress ($\eta^2 = 0.0015$, $p = .02$), depression ($\eta^2 = 0.0010$, $p = .04$), and relationship status ($\eta^2 = 0.0013$, $p = .02$). Nine-month covariates included household air quality ($\eta^2 = 0.0029$, $p < .001$), employment status ($\eta^2 = 0.0013$, $p = .04$), and relationship status ($\eta^2 = 0.0011$, $p = .03$).

3.3. Adversity

Adversity variables significantly associated with telomere length included area-level deprivation at antenatal ($\eta^2 = 0.006$, $p < .001$), nine-months ($\eta^2 = 0.004$, $p = .001$), four-years ($\eta^2 = 0.005$, $p < .001$), household crowding at antenatal ($\eta^2 = 0.0047$, $p < .001$), nine-months ($\eta^2 = 0.0072$, $p < .001$), four-years ($\eta^2 = 0.0023$, $p = .005$), household tenure at antenatal ($\eta^2 = 0.0086$, $p < .001$), nine-months ($\eta^2 = 0.0109$, $p < .001$), four-years ($\eta^2 = 0.0067$, $p < .001$), income-tested benefits at nine-months ($\eta^2 = 0.0010$, $p = .04$), four-years ($\eta^2 = 0.0012$, $p = .02$) and household smoking at four-years ($\eta^2 = 0.0021$, $p = .03$) (Supplementary Table 7). Variables with more than two groups (area-level deprivation, household crowding, household tenure, household smoking) remained significantly associated with telomere length following dichotomization (Supplementary Figs. 3–5 and Table 8). The dichotomized variables were combined at each timepoint to create an adversity score for each DCW (Supplementary Fig. 3). Individual adversity scores were significantly associated with telomere length at

Table 2
Cohort demographics of the main cohort ($n = 6853$) and biological cohort ($n = 3812$), including distributions of child sex, child prioritized ethnicity, total ethnicity, maternal age, maternal education, and NZ area-level deprivation collected at the antenatal DCW. The *p*-value indicates the statistical significance of the difference between the two cohorts. Significant values are bold.

Variable	Main cohort n	%	Biological cohort n	%	p-value
Child sex assigned at birth					
Female	3321	48.5	1834	48.1	0.73
Male	3532	51.5	1978	51.9	
Missing	0	0	0	0	–
Child prioritized ethnicity					
European	3645	53.2	2415	63.4	**<0.001**
Māori	950	13.9	451	11.8	**0.003**
Pacific	997	14.5	367	9.6	**<0.001**
Asian	1002	14.6	453	11.9	**<0.001**
MELAA	144	2.1	53	1.4	**0.009**
NZ	95	1.4	66	1.7	0.16
Missing	20	<1.0	<10	<1.0	–
Total ethnicity[a]					
European	4248	62.0	2740	71.9	**<0.001**
Māori	1266	19.2	623	16.3	**0.006**
Pacific	1156	16.9	449	11.8	**<0.001**
Asian	1091	15.9	493	12.9	**<0.001**
MELAA	168	2.5	68	1.8	**0.02**
NZ	146	2.1	90	2.4	0.44
Missing	<10	<1.0	0	0	–
Maternal age					
<20	327	4.8	124	3.3	**<0.001**
20–24	995	14.5	460	12.1	**<0.001**
25–29	1672	24.4	912	23.9	0.58
30–34	2127	31.0	1272	33.4	**0.01**
35–39	1439	21.0	873	22.9	**0.02**
40+	292	4.3	171	4.5	0.59
Missing	<10	<1.0	0	0	–
Maternal education					
No secondary school qualification	489	7.1	202	5.3	**<0.001**
Secondary school/ NCEA 1–4	1627	23.7	820	21.5	**0.009**
Diploma/Trade cert/ NCEA 5–6	2095	30.6	1127	29.6	0.28
Bachelor's degree	1552	22.6	974	25.6	**0.001**
Higher degree	1070	15.6	681	17.9	**0.003**
Missing	20	<1.0	<10	<1.0	–
NZ area-level deprivation					
1	522	7.6	339	8.9	**0.02**
2	586	8.6	371	9.7	**0.04**
3	600	8.8	380	10.0	**0.04**
4	645	9.4	417	10.9	**0.01**
5	529	7.7	312	8.2	0.39
6	647	9.4	410	10.8	**0.03**
7	684	10.0	368	9.7	0.59
8	753	11.0	386	10.1	0.17
9	895	13.1	438	11.5	**0.02**
10	989	14.4	390	10.2	**<0.001**
Missing	<10	<1.0	<10	<1.0	–

[a] The sum of total ethnicity does not equal 100 % as mothers were able to select multiple ethnicities.

four-years (Supplementary Fig. 4) and anxiety and depression (Supplementary Fig. 5). Each DCW adversity score was combined to create a raw quantitative ELA from zero to 15 (possible range 0–17). The raw quantitative model was the best predictor for anxiety (AIC = 12,878, BIC = 12,896, $R^2 = 0.023$, $p < .001$) and depression (AIC = 11,353, BIC = 11,371, $R^2 = 0.029$, $p < .001$). The grouped quantitative model was the best predictor for telomere length (quantitative: AIC = 3382.7, BIC = 3400.8, $R^2 = 0.0028$, $p = .003$; Grouped: AIC = 3373.5, BIC = 3391.7, $R^2 = 0.0058$, $p < .001$) and was nearly as good a predictor for both psychosocial outcomes (Anxiety: AIC = 12,897, BIC = 12,915, $R^2 = 0.017$, $p < .001$; Depression: AIC = 11,371, BIC = 11,390, $R^2 = 0.023$, $p < .001$). Thus, grouped quantitative modelling is presented.

3.4. Grouped adversity, telomere length and psychological outcomes

Grouped quantitative adversity showed significant positive associations with telomere length (β(SE) = 0.061(0.014), $p < .001$, $R^2 = 0.005$; Fig. 1), anxiety (β(SE) = 0.486(0.066), $p < .001$; $R^2 = 0.017$; Fig. 2C) and depression (β(SE) = 0.441(0.051), $p < .001$, $R^2 = 0.023$; Fig. 2D) at eight years (Table 4).

3.5. Telomere length and psychosocial outcomes

Telomere length was positively associated with anxiety symptoms as a continuous score (β(SE) = 0.018(0.083), $t = 2.18$, $p = .03$, df (13115), $R^2 = 0.0015$). When anxiety was grouped into *normative* ($n = 1561$, 50.1 %), *mild* ($n = 597$, 19.2 %), *moderate* ($n = 746$, 23.9 %), and *severe* ($n = 213$, 6.8 %) symptoms, each group had a mean (SD) telomere length of T/S = 0.406 (0.413), T/S = 0.419 (0.417), T/S = 0.428 (0.429), and T/S = 0.425 (0.400), respectively. Grouped anxiety was not significantly associated with telomere length at four-years.

Telomere length showed a positive trend association with depression symptoms as a continuous score (β(SE) = 0.125(0.065), $t = 1.91$, $p = .06$, df (13115), $R^2 = 0.001$). When grouped into no depression (68.6 %) and depression (31.4 %), the mean telomere length was T/S = 0.405 and T/S = 0.438, respectively. Grouped depression was significantly associated with telomere length at four-years (β(SE) = 0.041(0.020), $p = .043$, $R^2 = 0.0013$).

3.6. Multivariate modelling

VIF and eigenvalue analyses showed no multicollinearity between any of the covariates. When adjusting for covariates (Table 3 and Supplementary Tables 5 and 6), the multivariate model with telomere length as an outcome was significant for grouped adversity (β(SE) = 0.043 (0.017), $t = 2.58$, $p = .01$, df (13100), $R^2 = 0.031$). Modelling with anxiety as the outcome was also significant for grouped adversity (β(SE) = 0.206(0.077), $t = 2.67$, $p = .008$, df (13100), $R^2 = 0.037$) as was depression (β(SE) = 0.250(0.061), $t = 4.12$, $p < .001$, df (13100), $R^2 = 0.039$).

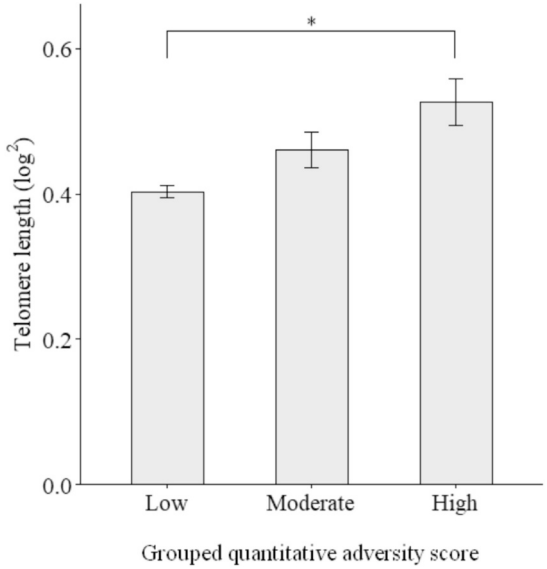

Fig. 1. Grouped quantitative adversity score and telomere length.

3.7. Covariates and interaction analyses

There was a significant positive relationship between telomere length and: child sex; child ethnicity; maternal perceived stress; maternal depression; antenatal alcohol intake; household air quality (Table 3). Due to insufficient statistical power, subset analyses were not conducted.

3.8. Mediation analysis

The direct effect of adversity on telomere length, anxiety and depression was positive and statistically significant (Table 4). The direct effect of telomere length on both psychosocial outcomes was not significant, nor was the resulting mediation (Table 4 and Fig. 3). The pattern of results for mediation analysis remained the same when adjusting for covariates (Table 5 and Fig. 4).

4. Discussion

Our study investigated the relationship between early-life adversity, broadly defined, and mental health outcomes in NZ children at eight-years of age. In addition, we assessed whether telomere length at four-years of age mediated the association between ELA and eight-year psychosocial outcomes. Adversity was positively associated with depression and anxiety symptoms. Telomere length was also positively associated with depression and anxiety symptoms. However, the association between ELA and psychosocial outcomes were not mediated by telomere length (a potential biomarker of stress). Surprisingly, ELA was positively associated with telomere length.

Our results support previous findings that cumulative or prolonged adversity is significantly associated with negative psychosocial outcomes (Schoon et al., 2003; Shonkoff et al., 2012; Nurius et al., 2015; Reiss, 2013). Children from socioeconomically disadvantaged families have a higher risk of developing negative psychosocial outcomes (Schoon et al., 2003; Reiss, 2013; Macintyre et al., 2018). Factors such as low parental education, unemployment, low income, debt, marital status, and area-level factors (poverty, poor housing) have the greatest effects (Reiss, 2013; Macintyre et al., 2018; Silva et al., 2016; Friedli and World Health Organisation (WHO), 2009) and were reflected by adversity measures used in the current study (maternal relationship status, household smokers, area-level deprivation, household crowding, household tenure, and income-tested benefits).

Our positive associations between telomere length and mental health is largely inconsistent with other (typically cross-sectional) studies (Darrow et al., 2016; Rius-Ottenheim et al., 2012; Huzen et al., 2010; Simon et al., 2006; Wade et al., 2020; Vincent et al., 2017; Zhang et al., 2010; Simon et al., 2015; Malan et al., 2011; Schaakxs et al., 2015). For example, greater depression and anxiety symptoms have been associated with shorter leukocyte telomere length in a sample ($n = 995$) of 11–17-year-olds. However, there is heterogeneity in the literature exploring the relationship between telomere length, adversity, and mental health. In a study of adolescents with or without anxiety disorders, it was reported that telomere length did not change for those with persistent anxiety disorders at a 5-year follow-up ($n = 76$). Critically, those in the "variable" anxiety disorder group (anxiety at only one time point) had longer telomeres at baseline compared to those in the no anxiety group, and there was a trend towards accelerated telomere

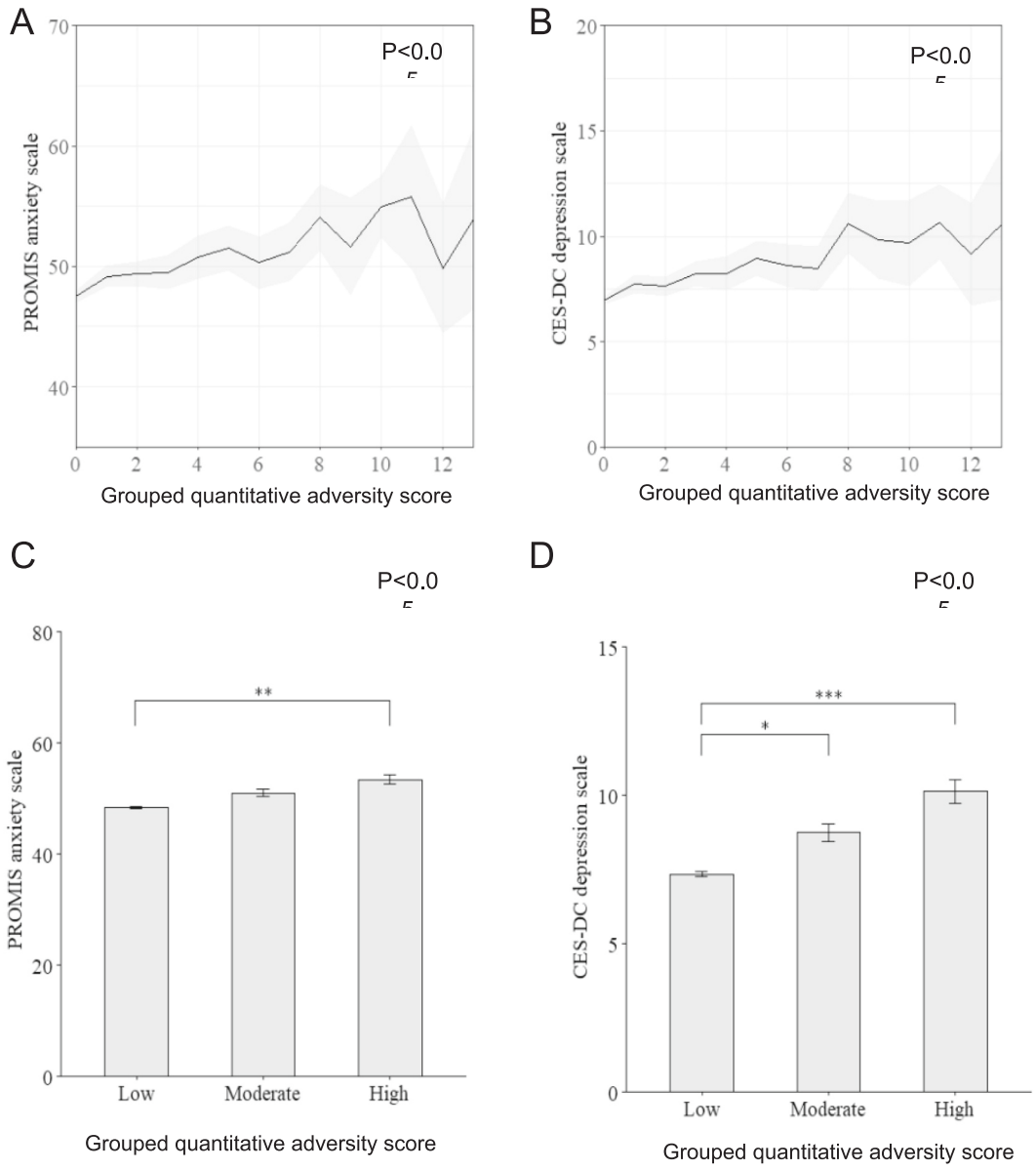

Fig. 2. Multivariate analysis of the raw and grouped quantitative adversity score and eight-year anxiety and depression scores. The relationship between (A) raw quantitative adversity score and eight-year anxiety, (B) raw quantitative adversity score and eight-year depression, (C) grouped quantitative adversity score and eight-year anxiety, (D) grouped quantitative adversity score and eight-year depression.

erosion over time (Cerveira de Baumont et al., 2021). Telomere length has also been associated with brain function and connectivity of regions involved in emotional processing (Powell et al., 2019). Notably, longer buccal telomere length was associated with greater amygdala activation, which may indicate a greater risk of internalizing conditions, such as depression and anxiety. Furthermore, in a study of high-risk young adults who had previously been in youth residential care institutions, it was reported that more severe adversity (childhood adversity, potentially traumatic exposures, or criterion A trauma) was associated with longer leukocyte telomeres. Having a diagnosis of Post-Traumatic Stress Disorder (PTSD) was also associated with longer telomeres. Other studies have reported similar results, with adversity and PTSD being associated with longer telomere length from buccal samples (Küffer et al., 2016) and saliva samples (Womersley et al., 2022). Furthermore, longitudinal research of soldiers found that PTSD was associated with telomere lengthening over a six-month period (Boks et al., 2015). More generally, reviews and meta-analyses assessing the association between adversity or mental health outcomes and telomere length have highlighted small effect sizes as well as heterogeneity and evidence of publication bias (Ridout et al., 2018a; Pepper et al., 2018; Bürgin et al., 2019; Li et al., 2017; Ismail et al., 2025; Malouff and Schutte, 2017). Pepper et al. (2018) suggested that the observed publication bias may not be due to the absence of null results, but rather that contrary results are suppressed. These studies, in conjunction with our findings, highlight that further research is required to understand the relationship between adversity, mental health and telomere biology.

Our positive relationship between adversity and telomere length is inconsistent with the literature (Coimbra et al., 2017; Ridout et al., 2018a; Malouff and Schutte, 2017; Tyrka et al., 2016; Mitchell et al., 2018; Puterman et al., 2016; Kananen et al., 2010; Wojcicki et al., 2016; Wojcicki et al., 2015; Willis et al., 2019; Willis et al., 2018; Lynch et al., 2017; Carroll et al., 2013; Shiels et al., 2011). Adversity exposure is believed to accelerate telomere attrition, likely due to greater oxidative stress (Barnes et al., 2019; Ridout et al., 2018a; Shiels et al., 2011; Ridout et al., 2018b; Marasco et al., 2022). Many studies report a negative association between adversity and telomere length, regardless

Table 3
Summary of interaction analyses of all covariates in the association between telomere length and each psychosocial outcome (anxiety and depression). Significant values are bold.

Covariate	Anxiety		Depression	
	Coefficient	p-value	Coefficient	p-value
Child sex assigned at birth				
Female	0.202	**0.045**	−0.012	0.88
Male	0.158	0.12	0.268	**<0.001**
Child prioritized ethnicity				
Asian	0.332	0.08	−0.075	0.61
European	−0.113	0.25	−0.064	0.41
Māori	0.667	**<0.001**	0.528	**<0.001**
MELAA	0.621	0.25	0.013	0.98
NZ	−0.163	0.38	0.087	0.55
Pacific	1.146	**<0.001**	0.787	**<0.001**
Maternal age				
Maternal age	0.003	0.21	0.002	0.26
Maternal perceived stress				
Low	0.084	0.35	0.054	0.45
High	0.451	**<0.001**	0.324	**0.001**
Maternal depression				
No depression	0.111	0.20	0.072	0.29
Depression	0.626	**<0.001**	0.460	**<0.001**
Antenatal alcohol intake				
Stopped drinking	0.060	0.56	0.003	0.97
Any drinking	0.082	0.51	0.177	0.07
Non-drinkers	0.527	**<0.001**	0.302	**0.004**
Household air quality				
0	0.038	0.74	−0.018	0.85
1	0.045	0.72	0.022	0.83
2	0.255	**0.04**	0.214	**0.03**
3	0.911	**<0.001**	0.701	**<0.001**

Table 4
Mediation (without covariates) analysis summary. Significant values are bold.

Outcome	Model	Estimate	SE	p-value	95 % CI
Telomere length	Direct effect of adversity on telomere length	**0.06**	0.014	**<0.001**	[0.033, 0.089]
Anxiety	Direct effect of telomere length on anxiety	0.14	0.082	0.10	[−0.027, 0.297]
	Direct effect of adversity on anxiety	**0.49**	0.066	**<0.001**	[0.349, 0.606]
	Mediation effect of adversity on anxiety	0.008	0.005	0.13	[−0.002, 0.019]
Depression	Direct effect of telomere length on depression	0.08	0.065	0.20	[−0.044, 0.209]
	Direct effect of adversity on depression	**0.44**	0.051	**<0.001**	[0.336, 0.537]
	Mediation effect of adversity on depression	0.01	0.004	0.22	[−0.003, 0.013]

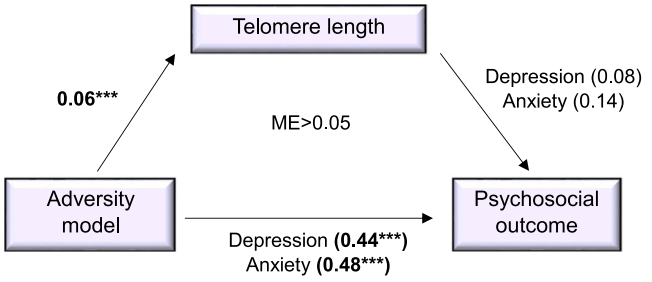

Fig. 3. Mediation analysis using SEM without covariates. Direct effects of the associations between early life adversity, four-year telomere length, and eight-year anxiety and depression. Indirect effect of telomere length on the association between adversity and both psychosocial outcomes. ME = Mediation effect.

Table 5
Mediation (with covariates) analysis summary. Significant values are bold.

Outcome	Model	Estimate	SE	p-value	95 % CI
Telomere length	Direct effect of adversity on telomere length	**0.04**	0.017	**0.01**	[0.002, 0.067]
Anxiety	Direct effect of telomere length on anxiety	0.08	0.083	0.36	[−0.086, 0.239]
	Direct effect of adversity on anxiety	**0.21**	0.077	**0.01**	[0.067, 0.369]
	Mediation effect of adversity on anxiety	0.003	0.003	0.40	[−0.003, 0.009]
Depression	Direct effect of telomere length on depression	0.09	0.065	0.19	[−0.042, 0.213]
	Direct effect of adversity on depression	**0.25**	0.060	**<0.001**	[0.135, 0.372]
	Mediation effect of adversity on depression	0.003	0.003	0.27	[−0.002, 0.008]

Note. Covariates included: Child sex assigned at birth; child externally prioritized ethnicity; maternal antenatal alcohol consumption, maternal age, maternal perceived stress, maternal depression, and air quality (Table 3).

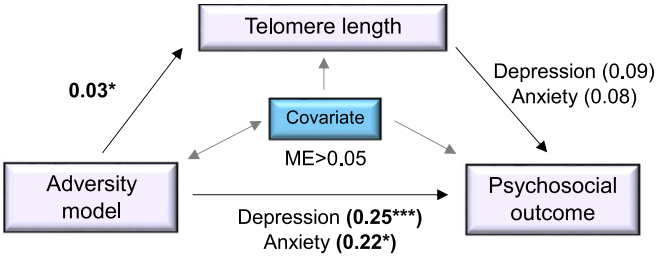

Fig. 4. Mediation analysis using SEM with the inclusion of covariates. Direct effects of the associations between early life adversity, four-year telomere length, and eight-year anxiety and depression. Indirect effect of telomere length on the association between adversity and both psychosocial outcomes. ME = Mediation effect.

of age at adversity (Willis et al., 2019; Willis et al., 2018; Oliveira et al., 2016; Drury et al., 2012). However, telomere dynamics and the response to adversity may change throughout life. The positive association observed in our results may indicate initial lengthening during early life.

Longitudinal child research using saliva telomere length from early life (8 months, 2 years, and 3 years) suggests that telomere attrition is not linear, with evidence of both telomere lengthening and telomere shortening for different individuals (Bosquet Enlow et al., 2020). Telomere lengthening was more likely in females than males, and for children whose families had fewer financial reserves. Speculatively, if telomere length is partly determined during the antenatal period, individuals exposed to negative stressors in-utero may have longer telomeres as a protective mechanism to compensate for accelerated telomere attrition, which may occur due to traumatic or continued adversity experienced throughout life. Some studies report increased telomere length over time, which may be a compensatory mechanism to overcome potential telomere shortening (Bosquet Enlow et al., 2020; Shalev, 2012; Boks et al., 2015), but replication is needed due to methodological differences (Steenstrup et al., 2013). Interestingly, individuals with longer telomeres may have an increased telomere attrition rate (Verhulst et al., 2013; Révész et al., 2016). Thus, there may be a trilateral relationship between adversity, telomere length and telomere attrition, which warrants further investigation. Although the timing is unlikely to contribute to result heterogeneity, there is a bias in study reporting, with a distinct lack of data exploring telomere length in children and longitudinal data (Pepper et al., 2018).

Telomere length captures biological responses to stress and highlights that biological stress may differ between individuals experiencing similar stressors or adverse exposures. Moreover, biological aging, which telomere length is believed to reflect, is associated with inflammation, oxidative stress, and mitochondrial dysfunction, all of which are linked to increased risk of depression and anxiety. Thus, telomeres may serve as a biologically meaningful indicator of stress-related health vulnerability. Our mediation analysis was exploratory, aiming to assess whether telomere length is statistically associated with part of the pathway between early life adversity and later mental health. Telomere length did not mediate the positive association between ELA and anxiety or depression symptoms. Our null result may be due to the age of participants (8 years old), since the onset of depression and anxiety typically occur at older ages (Solmi et al., 2022). Approximately 38 % of anxiety and fear-related disorders and 2.5 % of mood disorders including depression emerging by age 14. It has been suggested that shorter telomeres may be associated with a greater risk of depressive disorders in children by inhibiting the proliferation and differentiation of neural stem cells, subsequently affecting neural morphology, organization, and connectivity associated with various mood disorders (Ferrón et al., 2009; Liu et al., 2018; Zhou et al., 2016). Girls (aged 10–14 years) who had depressed mothers and a higher familial risk of depression had shorter telomeres compared to girls with a lower depression risk (Gotlib et al., 2015). Furthermore, greater cortisol reactivity to stress was associated with shorter telomere length. As such, hypothalamic-pituitary-adrenal (HPA) axis dysregulation and telomere length may be biological markers for major depressive disorder prior to onset. Conversely, a longitudinal study ($n = 195$) using buccal swabs to measure telomere length found that telomere length was statistically negatively associated with internalizing factors at ages 12–14 but only showed a negative trend association at ages 8–10 years old. Internalizing factors at ages 8–10 predicted shorter telomere length at age 12–14, suggesting that telomere erosion may be related to experiencing earlier internalizing factors (Wade et al., 2020). However, longitudinal research on the association between telomere length and psychosocial outcomes is limited. If telomere length is a biological marker of mental health conditions prior to onset, it may occur later in life and may not be apparent at eight years old.

Our null result for the mediation analysis may also be due to confounding interactions within covariates. For example, we found a positive association between depression and telomere length for males but not females. The opposite pattern was found for anxiety, with a positive association for females but not males. Research on the prevalence of childhood depression and anxiety between sexes is limited. Rates may be equal between pre-pubertal males and females (Altemus et al., 2014; Son and Kirchner, 2000; Avenevoli et al., n.d.; Cummings et al., 2014; Kendall et al., 2010; Kendall et al., 2008), though some suggest depression is slightly more common in male children under the age of 12 (Avenevoli et al., n.d.; Douglas and Scott, 2014), and anxiety is more common in female children (Schneier and Goldmark, 2015; Chaplin and Aldao, 2013; Aune and Stiles, 2009; Leikanger et al., 2012). The theory of opposing interactions may also apply to certain covariates in our study, including maternal perceived stress, maternal depression, maternal alcohol intake, household air quality, and child ethnicity (Table 3). Notably, the association between ethnicity and psychosocial outcomes is likely shaped by social inequities; thus, adjusting for SES may lead to decreased statistical associations. Though we were underpowered to investigate subgroup analyses, telomere length regulation is complex and likely involves many interactions between environmental, social, genetic and health-related influences (Coimbra et al., 2020).

The current study has many strengths, including assessing telomere length early in life and using a broad definition of adversity. We also have a multi-ethnic and socioeconomically diverse cohort, large sample size ($n > 3000$), proximal collection of biological and survey data, and longitudinal methodology. Comparatively, many studies examining telomere in early life are cross-sectional and have small sample sizes ($n < 100$) (Lindrose et al., 2021), which may explain the variability in results from telomere length studies (Pepper et al., 2018). Most research has focused on adult populations and analyzed telomere length concurrently with psychosocial data. Our results provide a more predictive approach using four-year telomere length and later anxiety or depression. Utilizing longitudinal child cohort data provides insight into sensitive periods, with limited recall bias, and allows investigation into the development of psychosocial outcomes over the life-course.

Our study had limitations and methodological considerations that may contextualize findings. For example, our crowding variable does not account for room sharing (e.g., couples or children) and may reflect positive social, cultural, and economic support in NZ (Poland et al., 2007; Torshizian and Grimes, 2021). Furthermore, some adversity measures were not collected at all the DCWs, and some measures identified in the literature were unavailable. For instance, severe adversities that inflict trauma, such as family violence, institutionalization, loss of a family member, exposure to violence, abuse or neglect, can lead to adverse psychosocial outcomes (Coimbra et al., 2017; Ridout et al., 2018a; Beal et al., 2019), were not measured or not included due to limited cell sizes.

Research heterogeneity may help contextualize findings in terms of populations (e.g., age, sex, or ethnicity), tissue and cell types, and the age of adversity studied. For instance, females and African individuals have longer telomeres than males and Europeans (Aviv and Shay, 2018; Brown et al., 2017; Hansen et al., 2016), though mechanisms are undetermined. Sex-differences in telomere length may be due to differences in estrogen, which can regulate telomerase (Aviv and Shay, 2018). Additionally, differences between ethnic groups may be influenced by polygenic adaptation, as there may have been selection for shorter telomeres in European individuals to counteract the increased risk of melanoma (Aviv and Shay, 2018; Hansen et al., 2016). Importantly, our study cohort was broadly generalizable to the NZ population based on census data from the time of data collection, even after removing children with missing data. Compared to the NZ census total response ethnicity, the biological cohort was broadly representative for participants who identified as European/NZ (74 % vs 74.3 %), Māori (14.9 % vs 16.3 %), Asian (11.8 % vs 12.9 %), and Middle Eastern/ Latin American/ African (MELAA; 1.2 % vs 1.8 %), respectively (Statistics New Zealand, 2024). Notably, Pacific people were overrepresented in the biological cohort (11.8 %) compared to the census (7.4 %). Representation of self-reported ethnic identification provides valuable insights into telomere biology in often underrepresented groups that may be marginalized and experience stressors due to colonization, racism, discrimination, and other social determinates of health (Coimbra et al.,

2020; Thayer et al., 2023).

Another consideration in telomere biology is parental age. In line with the literature and as previously discussed for this cohort (Ly et al., 2019), maternal age was positively associated with child telomere length. Paternal age has also been shown to be positively associated with child telomere length and may be an important covariate to consider (De Meyer et al., 2007). However, for the current cohort maternal and paternal ages were shown to be highly correlated ($r = 0.714$) (Ly et al., 2019) with available data from fathers being limited, hence, maternal age was used to maximize sample size. Though future research may consider utilizing both maternal and paternal data.

Tissue type may moderate the association between adversity and telomere length (Ridout et al., 2018a). Saliva and buccal swab samples are non-invasive methods for pediatric telomere research, though saliva samples may have lower cellular content and more variability in cell types, which can affect the precision of telomere measurement (Rej et al., 2021). Furthermore, saliva and buccal swab samples differ in cellular composition (Shalev et al., 2013; Oliveira et al., 2016). Specifically, saliva samples primarily consist of leukocytes, whereas buccal samples mainly consist of epithelial cells (Aviv and Shay, 2018; Theda et al., 2018). Buccal epithelial cells have greater exposure to exogenous stressors compared to leukocytes. They are highly prolific, which may influence telomere length by accelerating telomere attrition (Küffer et al., 2016). In contrast, leukocyte DNA from peripheral blood is considered the gold standard for telomere source tissue. Cross-tissue analyses in older aged samples than the current cohort suggest differences in telomere length (Goldman et al., 2018). A recent meta-analysis also indicated that tissues source, age, assay methodology, and assessment tools may moderate the association between telomere length and depression (Ismail et al., 2025). However, the impact of source tissue (leukocytes vs others) was attenuated in multivariable meta-regression and no longer significant. Conversely, some meta-analyses found no or minimal impact of sample/tissue type on findings (Pepper et al., 2018). While our use of saliva samples aligns with common practice and ethical considerations in child research, the limited longitudinal data across tissue types prevents ruling out these effects. Another consideration is that longer telomeres observed early in life are associated with accelerated telomere attrition throughout life (Révész et al., 2016; Weischer et al., 2014; Aviv et al., 2009; Nordfjäll et al., 2009; Ehrlenbach et al., 2009), which we could not assess with the available data. As such, these results should be interpreted accordingly when considering tissue-specific telomere dynamics and methodological variability. However, given these concerns we conducted sensitivity analysis using only participants with saliva samples, which showed the same pattern of results. Critically, replication is required, considering tissue type, telomere dynamics, age, severity of mental health outcomes and experiences of adversity.

Overall, we found a positive association between ELA and four-year telomere length as well as eight-year anxiety and depression symptoms. Our findings emphasize the cumulative effects of ELA on biological and neurological pathways, which are particularly important for improving health and well-being. The positive association between adversity and telomere length was unexpected and warrants further investigation. There may be a complex telomere regulation and maintenance system that responds to external stimuli and acts to protect telomere length from subsequent accelerated attrition. Longitudinal measurement of adversity, telomere length, and psychosocial outcomes is critical to understanding these dynamic, cumulative, and time-sensitive relationships across the life-course.

CRediT authorship contribution statement

Benjamin D. Fletcher: Writing – review & editing, Writing – original draft, Funding acquisition, Conceptualization. **Gemma Hughes-Waldon:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Emma J. Marks:** Writing – review & editing, Writing – original draft, Supervision, Methodology. **Karen E. Waldie:** Writing – review & editing, Funding acquisition, Conceptualization. **Russell G. Snell:** Writing – review & editing, Supervision, Resources, Methodology. **Zaneta M. Thayer:** Writing – review & editing, Methodology, Conceptualization. **Susan M.B. Morton:** Project administration, Funding acquisition, Conceptualization. **Sarah Knowles:** Methodology, Funding acquisition, Conceptualization. **Kien N. Ly:** Methodology, Funding acquisition, Conceptualization. **Caroline G. Walker:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation.

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Declaration of competing interest

All authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2025.120600.

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