Prospective association of adverse childhood experiences and social support with all-cause mortality among young adults in the USA

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ABSTRACT

Background This study examined the independent effects of adverse childhood experiences (ACEs) and social support on all-cause mortality among US young adults and assessed whether social support modified the ACEs–mortality association.

Methods We used data from the National Longitudinal Study of Adolescent to Adult Health, a population-based prospective US cohort. Our study sample included 12 421 children and adolescents aged 11–17 years at baseline (1994–1995) and followed up until 2016–2018. ACEs were assessed from youth and parent responses at baseline. Social support was assessed using a subjective measure of nine items and categorised into high, moderate and low. Weighted multivariable Cox proportional hazards models and Aalen's additive hazards models estimated HRs and hazard differences (HDs) per 10 000 individuals per year.

Results At baseline, participants' mean age was 15.4 years (SD=1.5), 49% were females and 46% had ≥1 ACEs. 327 deaths (2.6%) occurred during 301 416 person-years of follow-up. High social support was associated with decreased risk of all-cause mortality independent of ACEs after controlling for sociodemographic factors ($HR_{adjusted}$: 0.63 (0.42 to 0.93); $HD_{adjusted}$: −5.00 (−9.33 to −0.67)). Compared with those without ACEs and high social support, those with ≥1 ACEs and low social support had over two times greater hazards of premature mortality ($HR_{adjusted}$: 2.03 (1.45 to 3.49))—equivalent to approximately 9.4 additional deaths per 10 000 people per year ($HD_{adjusted}$: 9.41 (2.08 to 16.74)), after adjusting for baseline sociodemographic factors. Stratified analyses showed that ACEs were associated with increased risk of mortality in the low social support group only.

Conclusions Complementing ACEs prevention efforts with social support interventions could be effective strategies to reduce premature deaths among US young adults.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Adverse childhood experiences (ACEs) are associated with poor health outcomes, including adulthood mortality.
⇒ Social support may buffer against the mortality risk associated with ACEs. Prospective studies examining long-term effects through young adulthood are limited.

WHAT THIS STUDY ADDS

⇒ In this nationally representative sample of US adolescents, greater social support was associated with lower all-cause mortality through young adulthood, independent of ACEs.
⇒ ACEs had heterogeneous associations across social support levels, with significantly greater mortality risk only among those with a low level of social support.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Population-level interventions that promote social support among those experiencing adverse childhood experiences may help reduce premature deaths among young adults in the USA.

INTRODUCTION

Adverse childhood experiences (ACEs) are potentially traumatic experiences occurring during childhood/adolescence such as abuse or neglect, caregiver mental illness or substance use problems, and domestic and community violence. Exposure to adverse family and community environments during this critical developmental period alters physiological, behavioural and psychological mechanisms affecting lifelong health and development.[1 2] A recent review of meta-analysis estimated that ACEs accounted for 15% of total mortality in the USA from leading causes of deaths, including heart diseases, cancer and suicide.[3] Annual economic costs attributable to ACEs approximate to about $750 billion.[4] Findings of increased mortality risk during early and mid-adulthood associated with ACEs have been reported using data from the British Birth Cohort,[5] Danish Nationwide Registers[6] and Swedish cohort,[7] which included large samples and prospective assessment of ACEs, highlighting the need to study mortality risks among US youths and identify intervention targets.

A 2009 study among adult members of the Kaiser Permanente health insurance group in the USA found no clear association between ACEs and mortality. However, two-thirds of the study sample were aged ≥50 years at baseline, the follow-up duration was only 10 years, and the sample was not representative of the US population.[8] Similar studies using the Midlife Development in the United States and the Health and Retirement Survey found a greater

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risk of mortality during adulthood associated with retrospective reports of ACEs.[9 10] Findings from a large non-representative US pregnancy cohort that followed participants over 38 years from childhood to middle adulthood reported 27%–45% greater risk of premature mortality through middle adulthood associated with ≥2 ACEs.[11] While much scientific focus has been given to understanding the health effects of ACEs, evidence is limited on the buffering effect of modifiable factors in the social environment in which ACEs occur. This is important to identify potential areas for public health and policy interventions. Positive experiences during childhood, such as positive psychosocial resources including social support have been shown to lower the risk of mortality through adulthood.[12–14] Further, social support has been found to buffer the effect of ACEs on mental health outcomes.[15 16] However, prospective evidence is lacking on the moderating role of social support on the impact of ACEs on risk for premature mortality among young adults.

To address these current limitations, we used data from a prospective cohort of a nationally representative sample of US adolescents followed through young adulthood. We examined independent associations of ACEs and perceived social support with the risk of premature all-cause mortality and tested for potential effect modification by social support on the ACEs-mortality association. Our specific hypotheses were: (a) ACEs and perceived social support are independently associated with greater and lower all-cause mortality through young adulthood, respectively and (b) Social support modifies the risk of mortality associated with ACEs.

METHODS

Study population and setting

We used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health).[17] In 1994, Add Health used a stratified school-based design to select a nationally representative sample of high schools and a paired feeder school from a list of all high schools in the USA. An in-school questionnaire was administered to all students who attended the selected schools during the 1994–1995 school year. Then, from the school rosters, a core sample was selected using systematic random sampling for in-depth interviews at home with adolescents and one parent. Additionally, specific populations such as ethnic minorities, genetically related siblings, adopted youths and those with disabilities were oversampled. A total of 20 745 adolescents, representative of the US adolescents enrolled in grades 7–12 (age 11–21 years) in 1995 (wave I), constituted the baseline sample/wave for this study. There were five waves of follow-up. Wave V (age 33–43 years) during 2016–2019 included mortality surveillance that identified and tracked deceased participants. Those with missing sampling weights (n=1821), missing age (n=14), those 18 years of age or older at baseline (n=3216), those with missing ACEs or their combinations (n=2129), those with missing social support information (n=313) or those with missing covariates or their combinations (n=831) were excluded (figure 1). A total of 12 421 participants who had complete information on exposure, outcome and covariates were included in our primary analysis. We used restricted-use data which is available from the Carolina Population Center on contractual agreement.

Exposure measures

Adverse childhood experiences

We included five ACEs available in the Add Health baseline survey. They included the death of a biological parent (father or mother), parental divorce, substance use in the household (alcoholism, illegal drug availability), suicide attempt by a family member and exposure to community violence. These ACEs, adapted from the original ACEs questionnaire and the expanded ACEs, have been shown to adversely affect health and well-being throughout adulthood.[18 19] Data on parental divorce and household substance use were obtained from a parent survey, while data on other ACEs were obtained from in-home interviews with the adolescent participant. We dichotomised the responses for each ACE item to indicate presence or absence of the ACEs exposure (1=presence, 0=absence) (online supplemental eTable 1). We then summed the dichotomous responses to each item to create a cumulative ACEs score which ranged from 0 to 5, with higher values indicating exposure to a higher number of ACEs. To test for a potential dose–response relationship between ACEs and mortality, we categorised ACEs into three categories: 0, 1 and ≥2 ACEs. For effect modification analyses, to gain statistical power, we used a binary ACEs variable (0 and ≥1 ACEs).

Social support

We defined social support using a summary indicator involving nine items that asked respondents the perceived level of support from three sources—parents, family and school at baseline (online supplemental eTable 2). The 5-point Likert scale responses to each of the nine items were assigned an ordered score, with those strongly disagreeing with the positive statements about perceived social support coded as 1, and those strongly agreeing with the positive statements coded as 5. We then summed each recoded Likert scale variable to create a social support summary index which ranged from 9 to 45. We used quartiles to divide respondents into four groups with respect to the level of social support—the lowest quarter was defined as low social support, the intermediate two quarters as moderate social support, and the highest quarter as high social support.

Outcome measure

All-cause mortality

Mortality was measured, as dead or alive, by the end of 2019 using the date of death for those who died. Because only the month and year of death were available, we assigned the 15th day of the month as the universal day of death for all deceased. Information about the ascertainment of decedents in the Add Health cohort can be found elsewhere.[20] We assigned the start date of follow-up as the date of baseline interview. Survival was censored at the end of the study period. For all deceased (study endpoint), we calculated survival time in years, using the difference between the baseline interview date in 1994–1995 and the date of death. For those alive, we calculated the follow-up time as the difference between the baseline interview date and the National Death Index matching date, that is, 31 December 2019.

Covariates

A set of a priori defined potential confounders of the association between ACEs and mortality and between social support and mortality were included as covariates in all multivariable analyses (online supplemental eFigure 1). This included baseline sociodemographic and bio-behavioural variables. The sociodemographic variables included participants' age at baseline (continuous), sex (male/female), race/ethnicity (Hispanic/White/Black/Other), parental educational attainment (high school, GED or less/some college or 2-year college/4-year college or higher) and parents reporting obesity or diabetes (yes/no). The bio-behavioural variables included participant's body mass index

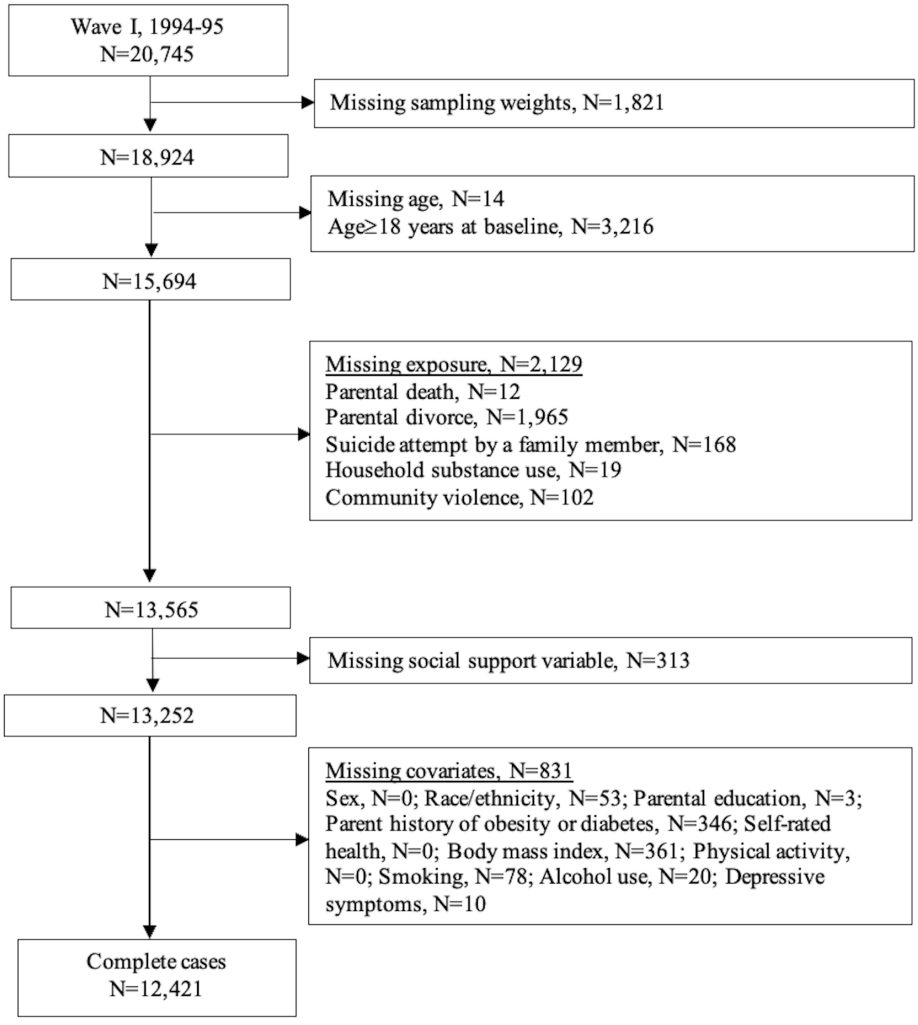

Figure 1 Study sample.

(BMI) (underweight/normal weight/overweight/obesity), physical activity (continuous), smoking (yes/no), alcohol use (yes/no) and depressive symptoms (continuous) (online supplemental eMethods).

Statistical analysis

We compared baseline characteristics of the respondents across ACEs categories (0, 1 and ≥2 ACEs) using descriptive statistics. We fitted Cox proportional hazards models and Aalen's additive hazards models to respectively estimate relative (hazard ratio, i.e., HR) and absolute (hazard difference, i.e., HD) measures of mortality associated with ACEs and social support.[21 22] The additive hazards models do not assume proportionality of hazards and provide a direct measure of the burden of mortality which is more appropriate in an epidemiological context for public health planning and resource allocation. For independent effect analyses, we adjusted estimates for the confounding effect of each other and other variables. To test for effect modification by social support, we included interaction terms in multivariable Cox and Aalen models. We also presented stratum-specific estimates by fitting stratified models where we evaluated the effect of ≥1 ACEs within each social support category in both Cox and Aalen models. Sociodemographic variables and bio-behavioural risk factors were included as potential confounders. We presented the mortality hazard estimates without and with adjustment for bio-behavioural factors. Adjustment for bio-behavioural factors may underestimate the true association because these factors may act as the mechanisms through which ACEs affect mortality. Time, in years, from the baseline date was used as a timescale in all models. Survey weights were used in all models to account for unequal probability of selection, clustering and stratification. We used two-sided $p<0.05$ to infer statistical significance.

We did several sensitivity analyses. First, we ran models to test the effect of parental, family and school support independent of each other and ACEs, and their potential effect modification effects on ACEs-mortality association using separate stratified models. Second, we repeated the analysis using BMI percentile as a continuous covariate. Third, we compared estimates, limiting our sample to include only those who died at least after 3 years of baseline assessment to reduce potential confounding bias from pre-existing disease. Fourth, to evaluate the potential effect of missing data, we compared the findings after imputing missing data for covariates using multiple imputation by chained equations.[23] Finally, we repeated the analyses without baseline age restriction to evaluate the potential effect of restricting the age to below 18 at exposure. Data analyses were carried out using 'survival' and 'timereg' packages in R V.4.4.2.

Table 1 Participant characteristics, overall and by ACEs exposure categories

		Number of ACEs			
	N* (%)	0 ACEs	1 ACE	≥2 ACEs	P value†
Overall	12 421 (100%)	54.2%	30.4%	15.4%	
Age, mean (SD)	15.4 (1.5)	15.3 (1.5)	15.4 (1.5)	15.5 (1.4)	0.281
Age group					0.177
11–15	4163 (42.0%)	55.9%	29.3%	14.8%	
16–18	8258 (58.0%)	52.9%	31.2%	15.9%	
Sex					0.002
Female	6267 (48.8%)	56.4%	29.3%	14.3%	
Male	6154 (51.2%)	52.1%	31.4%	16.5%	
Race and ethnicity					<0.001
Hispanic	1938 (10.9%)	48.2%	33.1%	18.7%	
Non-Hispanic white	7148 (71.1%)	57.8%	28.6%	13.6%	
Non-Hispanic black	2569 (14.1%)	38.3%	38.3%	23.4%	
Non-Hispanic other	766 (3.9%)	62.3%	26.8%	10.9%	
Parental education					<0.001
High school/GED or less	5204 (43.7%)	46.9%	33.4%	19.7%	
Some college/2-year college	2683 (22.0%)	52.7%	31.7%	15.7%	
4-year college or greater	4534 (34.3%)	64.4%	25.8%	9.8%	
Parental obesity or diabetes					0.004
Yes	3220 (26.3%)	51.0%	31.1%	17.8%	
No	9201 (73.7%)	55.3%	30.1%	14.6%	
Body mass index classification					0.028
Underweight	379 (3.2%)	57.9%	31.8%	10.3%	
Normal weight	9105 (72.9%)	54.8%	30.3%	14.9%	
Overweight	1678 (13.9%)	52.3%	31.0%	16.8%	
Obesity	1259 (10.0%)	51.2%	29.7%	19.1%	
Physical activity, mean score (SD)	3.9 (2.2)	4.0 (2.2)	3.9 (2.2)	3.9 (2.1)	0.447
Smoking					<0.001
Yes	3022 (25.5%)	42.6%	34.4%	23.0%	
No	9399 (74.5%)	58.1%	29.0%	12.8%	
Alcohol use					<0.001
Yes	3426 (27.4%)	42.4%	34.5%	23.1%	
No	8995 (72.6%)	58.6%	28.8%	12.5%	
Depressive symptoms, mean CES-D-5 score (SD)	2.3 (2.4)	2.0 (2.1)	2.6 (2.5)	3.0 (2.8)	<0.001
Social support					<0.001
Low	3328 (25.4%)	42.5%	34.0%	23.5%	
Moderate	6126 (49.3%)	54.6%	30.6%	14.8%	
High	2967 (25.2%)	65.1%	26.3%	8.5%	

*Unweighted; percentages and mean values in the table are weighted estimates.
†P values obtained from χ^2 test (for categorical variables) and Wald test (for continuous variables), adjusted for complex survey and multiple testing.
ACEs, adverse childhood experiences; CES-D-5, The Center for Epidemiologic Studies - Depression Scale-5 Item Version.

RESULTS

About 46% of the respondents had at least one ACE, and 15% had at least 2 ACEs at baseline. ACEs were higher among males, non-Hispanic Black individuals, those whose parents had lower educational attainment, those whose parents had a history of obesity or diabetes, those with higher BMI, those who smoked, used alcohol, those with higher CES-D scores and those with low levels of social support (table 1).

After a median follow-up time of 24.5 years, a total of 327 deaths (2.63% of the study sample) were observed. The underlying causes of deaths included motor vehicle accidents (19%), cardiometabolic diseases and cancer (19%), accidental poisoning (16%), suicide (13%), homicide (8%) and others (17%). The overall mortality rate was 10.84 per 10 000 individuals per year. The mortality rate per 10 000 individuals per year was incrementally higher among those exposed to a greater number of ACEs (0 ACEs: 8.95 (95% CI 7.60 to 10.55); 1 ACE: 11.80 (95% CI 9.82 to 14.19); ≥2 ACEs: 15.30 (95% CI 12.15 to 19.26)) and lower among those with greater perceived social support ((high: 9.29 (95% CI 7.32 to 11.80); moderate: 10.96 (95% CI 9.41 to 12.78); low: 12.02 (95% CI 9.86 to 14.67)).

In independent effects models, exposure to ≥2 ACEs was associated with increased but non-significant hazards (p=0.10) for all-cause mortality through young adulthood compared with those not exposed to ACEs after controlling for baseline social support and sociodemographic variables. Independent of ACEs, those with high levels of social support had 37% lower hazards of premature mortality ($HR_{adjusted}$: 0.63 (95% CI 0.42 to 0.93)) compared with those with low social support level—the lower hazards corresponding to about five fewer deaths per 10 000 individuals per year ($HD_{adjusted}$: −5.00 (95% CI −9.33 to

Table 2 Independent effect estimates for hazards of all-cause mortality associated with ACEs and social support

	Model 1*		Model 2†	
ACEs	HR (95% CI); p value	HD (95% CI); p value	HR (95% CI); p value	HD (95% CI); p value
ACEs				
0 ACEs	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
1 ACE	0.99 (0.71 to 1.39); p=0.951	−0.26 (−3.98 to 3.47); p=0.892	0.94 (0.66 to 1.33); p=0.723	−1.04 (−4.86 to 2.78); p=0.592
≥2 ACEs	1.36 (0.94 to 1.97); p=0.101	4.42 (−1.14 to 9.98); p=0.120	1.20 (0.81 to 1.82); p=0.359	2.78 (−2.92 to 8.48); p=0.339
Social support				
Low	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
Moderate	0.87 (0.65 to 1.15); p=0.323	−1.81 (−5.61 to 1.99); p=0.352	1.00 (0.74 to 1.35); p=0.997	0.03 (−3.85 to 3.91); p=0.989
High	0.63 (0.42 to 0.93); p=0.022	−5.00 (−9.33 to −0.67); p=0.023	0.80 (0.52 to 1.24); p=0.316	−2.13 (−6.78 to 2.52); p=0.369

HDs from Aalen's model presented as per 10 000 individuals per year.
HRs from Cox model.
*Adjusted for age, sex, race/ethnicity, parental education, and parents reporting obesity or diabetes, and social support or ACEs.
†Adjusted for age, sex, race/ethnicity, parental education, parents reporting obesity or diabetes, body mass index class, physical activity, smoking, alcohol use, depressive symptoms and social support or ACEs.
ACEs, adverse childhood experiences; HD, hazard difference; HR, hazard ratio.

–0.67)). Further adjustment for bio-behavioural factors attenuated the effect of ACEs and social support (table 2).

Interaction analyses revealed that compared with those without ACEs and high levels of perceived social support, those reporting ≥1 ACEs and low levels of social support had over two times greater hazards of premature mortality, corresponding to about 9.4 additional deaths per 10 000 individuals per year, after adjusting for baseline sociodemographic factors ($HR_{adjusted}$: 2.03 (95% CI 1.45 to 3.49); $HD_{adjusted}$: 9.41 (95% CI 2.08 to 16.74)). Further adjustment for bio-behavioural factors resulted in a higher but non-significant effect (figure 2).

Stratified models revealed the heterogeneous mortality effects of ACEs across social support levels. Among those with moderate and high levels of social support, ACEs were not associated with mortality. Among those with low levels of social support, those reporting ≥1 ACEs had over two times greater hazards of premature mortality ($HR_{adjusted}$: 2.66 (95% CI 1.49 to 4.75)), corresponding to about 12 additional deaths per 10 000 individuals per year ($HD_{adjusted}$: 11.90 (95% CI 5.00 to 18.80)), after adjusting for baseline sociodemographic variables. Further adjustment for bio-behavioural factors did not attenuate the association of ACEs with mortality in this group (figure 3).

When sources of social support were analysed separately, we found that school support was directly associated with reduced mortality independent of ACEs and other social support sources, and ACEs were significantly associated with greater mortality risk among those who had poor family support (online supplemental eTables 3 and 4). Findings remained robust when using BMI as a continuous covariate (online supplemental eTable 5), or after excluding participants who died within 3 years of baseline assessment (online supplemental eTable 6), or after imputing data for missing covariates (online supplemental eTable 7), or after including participants aged ≥18 years at baseline (online supplemental eTable 8).

DISCUSSION

To our knowledge, this is the first study that examined the prospective associations of ACEs and social support with mortality risk through young adulthood in a representative sample of US adolescents. We found that perceived social support reduced the risk of all-cause mortality prior to age 43 independent of ACEs. Further, perceived social support significantly modified the association between ACEs and mortality, with the ACEs-mortality association only evident in the low social group. These findings lend support to the stress-buffering hypothesis[24] suggesting that social support buffered the harmful effects of ACEs.

Social support may buffer the mortality effects of ACEs through several interrelated biological, psychological and behavioural mechanisms. Exposure to ACEs during sensitive developmental periods, without positive, secure and nurturing relationships, can activate the body's immune, neuroendocrine and metabolic

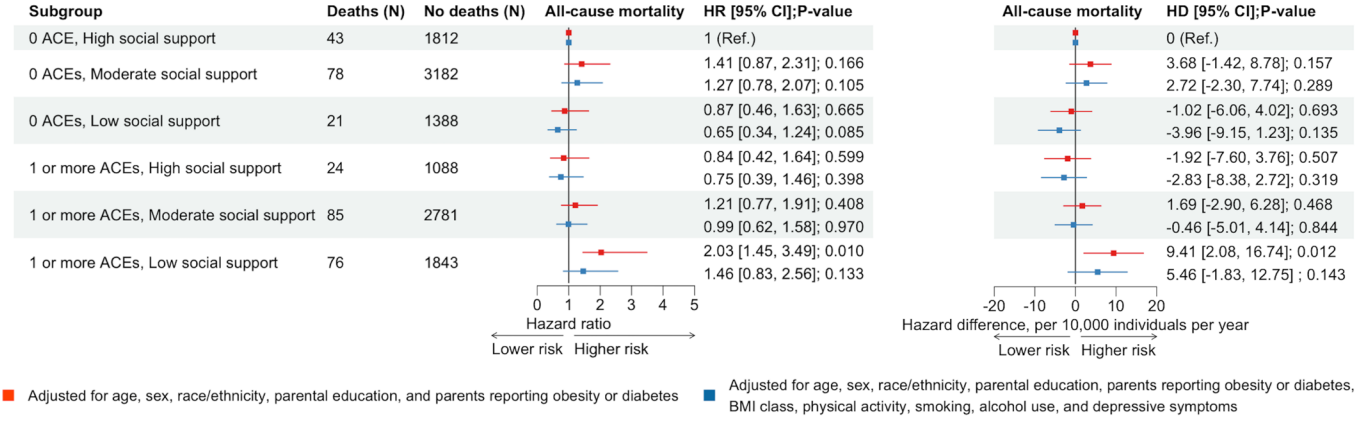

Figure 2 All-cause mortality hazards associated with adverse childhood experiences (ACEs) and social support.

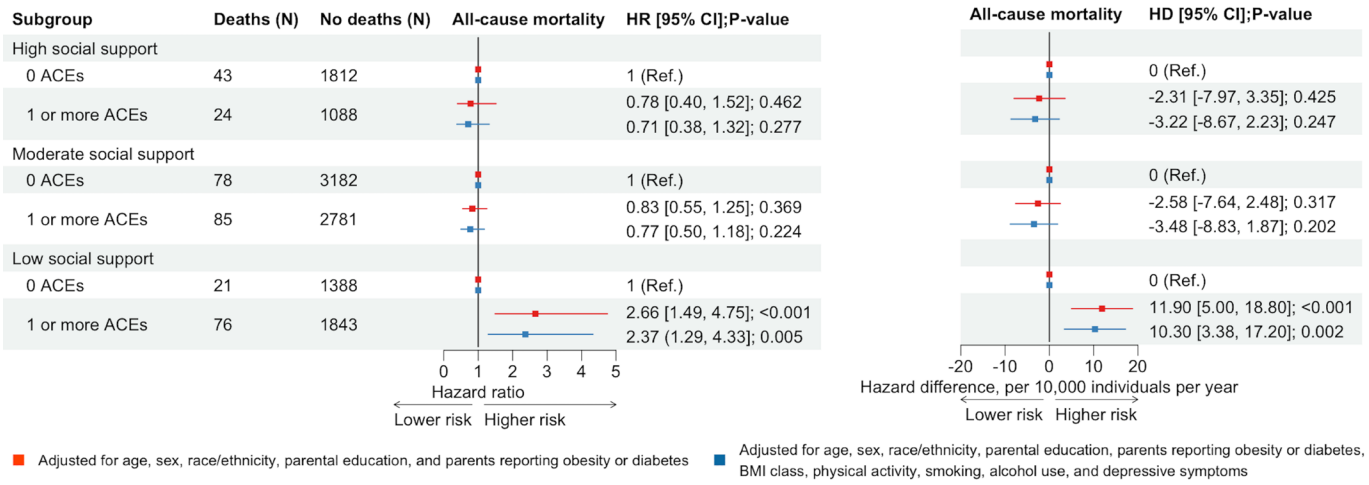

Figure 3 All-cause mortality hazards associated with ≥1 adverse childhood experiences (ACEs), stratified by social support.

pathways, acting as causal precursors to unhealthful behaviours, increased morbidity and early mortality.[25 26] While these biological effects typically accumulate with age,[2 27] our findings indicate that social support may mitigate physiological stress responses even in early life, preventing premature deaths from stress-related diseases. Further, ACEs can create biological vulnerability even in the absence of unhealthy behaviours among those with poor social support. Similarly, social support may improve self-esteem, promote emotion regulation and discourage engagement in health risk and maladaptive coping behaviours, thus protecting trauma-exposed individuals against several mental health and substance use disorders which are important pathways linking ACEs to early mortality.[3 28]

Our findings emphasise that critical attention is needed to complement ACEs prevention efforts with social support interventions among US adolescents. Moreover, enhancing perceived social support among those experiencing or with a history of ACEs may have an added benefit in reducing the burden of mortality among young adults. Among those with moderate or high social support, those with ACEs showed lower but non-significant mortality risk compared with those without ACEs. While this may be a chance finding, it is also possible that more vulnerable adolescents had greater benefits from social support, further underscoring the importance of ensuring that social support is effectively targeted toward those who need it most. Our analyses of social support sources suggest that family support is an important buffer of early mortality associated with ACEs. Consistent with existing literature that recognises school connectedness as a crucial protective factor against the development of youth risk behaviours,[29] we found that perceived school support was linked to lower mortality irrespective of ACEs exposure. This emphasises the need for social support interventions to go beyond promoting a positive parental/family environment to improving the school environment.[30] School interventions may focus on creating a safe and supportive climate, promoting student engagement and a sense of belonging through various activities and peer support groups, and maintaining positive interaction including mentoring.[31 32]

At policy level, few researches have demonstrated the positive impact of safety net policies that support families in reducing ACEs or related outcomes.[33 34] The Centers for Disease Control and Prevention's framework focuses on ensuring children have 'safe, stable, and nurturing relationships and environments' during childhood through strengthening economic supports for families, promoting social norms that protect against violence and adversity, teaching skills and connecting youths to caring adults.[32 35] Population-level interventions that target families and educational settings to improve the availability, accessibility and quality of perceived social support among vulnerable school-aged youths are critical to reduce premature deaths. Primary care interventions may target training health professionals to recognise ACEs, and their deleterious health consequences.[36]

This study has several strengths. Most prior studies retrospectively assessed ACEs exposure among participants who were well into their adulthood and among which morbidity and mortality rates are relatively high. Similarly, the use of proxy responses from a parent/caregiver or teacher may not provide complete or accurate information about the child's experiences. Further, the study sample in most of the prior studies was non-representative, limiting generalisability to the target population. This study addressed the concern of temporality and used adolescents-reported data from a representative US sample. Further, we provide both relative and absolute risks of mortality, enhancing the interpretability and practical relevance of our findings.

Our study has some limitations. Our study did not capture some of the most severe forms of ACEs such as sexual abuse and domestic violence. However, due to the clustering nature of ACEs, the prevalence of cumulative ACEs in our study is comparable to prior studies.[37] Second, the cause of death was not analysed due to the small number of deaths, and as such, the magnitude and the mechanisms of effect of ACEs and/or social support may differ for natural versus unnatural causes among the young population. Residual confounding from unmeasured contextual factors such as household income or neighbourhood socioeconomic disadvantage is a possibility.

CONCLUSIONS

ACEs were not associated with all-cause mortality independent of social support. High social support was protective against mortality independent of ACEs. ACEs conferred all-cause mortality risk only in the presence of low social support. Interventions that target increasing perceived social support among those with ACEs may have the greatest public health benefit in reducing premature deaths among young adults in the USA.

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Contributors KT and JRT conceptualised the study. KT, YS and JC were responsible for the methodology. KT conducted the formal analysis and curated the data. KT wrote the original draft, with review and editing contributions from YS, JC, EAV and JRT. YS, JC, EAV and JRT supervised the study. KT is the guarantor of this work.

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Competing interests None declared.

Patient consent for publication Not applicable.

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REFERENCES

1 Hertzman C. The biological embedding of early experience and its effects on health in adulthood. *Ann N Y Acad Sci* 1999;896:85–95.
2 Berens AE, Jensen SKG, Nelson CA. Biological embedding of childhood adversity: from physiological mechanisms to clinical implications. *BMC Med* 2017;15:135.
3 Grummitt LR, Kreski NT, Kim SG, *et al*. Association of Childhood Adversity With Morbidity and Mortality in US Adults: A Systematic Review. *JAMA Pediatr* 2021;175:1269–78.
4 Bellis MA, Hughes K, Ford K, *et al*. Life course health consequences and associated annual costs of adverse childhood experiences across Europe and North America: a systematic review and meta-analysis. *Lancet Public Health* 2019;4:e517–28.
5 Kelly-Irving M, Lepage B, Dedieu D, *et al*. Adverse childhood experiences and premature all-cause mortality. *Eur J Epidemiol* 2013;28:721–34.
6 Rod NH, Bengtsson J, Budtz-Jørgensen E, *et al*. Trajectories of childhood adversity and mortality in early adulthood: a population-based cohort study. *Lancet* 2020;396:489–97.
7 Jackisch J, Brännström L, Almquist YB. Troubled childhoods cast long shadows: Childhood adversity and premature all-cause mortality in a Swedish cohort. *SSM Popul Health* 2019;9:100506.
8 Brown DW, Anda RF, Tiemeier H, *et al*. Adverse childhood experiences and the risk of premature mortality. *Am J Prev Med* 2009;37:389–96.
9 D'Arcy-Bewick S, Turiano N, Sutin AR, *et al*. Adverse childhood experiences and all-cause mortality risk in adulthood. *Child Abuse Negl* 2023;144:106386.
10 Puterman E, Weiss J, Hives BA, *et al*. Predicting mortality from 57 economic, behavioral, social, and psychological factors. *Proc Natl Acad Sci U S A* 2020;117:16273–82.
11 Yu J, Patel RA, Haynie DL, *et al*. Adverse childhood experiences and premature mortality through mid-adulthood: A five-decade prospective study. *Lancet Reg Health Am* 2022;15:100349.
12 Vila J. Social Support and Longevity: Meta-Analysis-Based Evidence and Psychobiological Mechanisms. *Front Psychol* 2021;12:717164.
13 Shor E, Roelfs DJ, Yogev T. The strength of family ties: a meta-analysis and meta-regression of self-reported social support and mortality. *Soc Networks* 2013;35:626–38.
14 Pierce JB, Kershaw KN, Kiefe CI, *et al*. Association of Childhood Psychosocial Environment With 30-Year Cardiovascular Disease Incidence and Mortality in Middle Age. *J Am Heart Assoc* 2020;9:e015326.
15 Su Y, D'Arcy C, Meng X. Social Support and Positive Coping Skills as Mediators Buffering the Impact of Childhood Maltreatment on Psychological Distress and Positive Mental Health in Adulthood: Analysis of a National Population-Based Sample. *Am J Epidemiol* 2020;189:394–402.
16 Wan Y, Chen R, Ma S, *et al*. Associations of adverse childhood experiences and social support with self-injurious behaviour and suicidality in adolescents. *Br J Psychiatry* 2019;214:146–52.
17 Harris KM, Halpern CT, Whitsel EA, *et al*. Cohort Profile: The National Longitudinal Study of Adolescent to Adult Health (Add Health). *Int J Epidemiol* 2019;48:1415–1415k.
18 Felitti VJ, Anda RF, Nordenberg D, *et al*. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am J Prev Med* 1998;14:245–58.
19 Cronholm PF, Forke CM, Wade R, *et al*. Adverse Childhood Experiences: Expanding the Concept of Adversity. *Am J Prev Med* 2015;49:354–61.
20 Trani EA, Hill MJ, Loehr LR, *et al*. *Add health wave V documentation: mortality outcomes surveillance, part I: ascertaining decedents*. Carolina Population Center, 2022.
21 Cox DR. Regression models and life-tables. *J R Stat Soc Series B* 1972;34:187–202.
22 Aalen O. A model for nonparametric regression analysis of counting processes. Mathematical Statistics and Probability Theory: Proceedings, Sixth International Conference, 1978; 1978:1–25.
23 Buuren S, mice G-O. mice: Multivariate imputation by chained equations in R. *J Stat Softw* 2011;45:1–67.
24 Cohen S, Wills TA. Stress, social support, and the buffering hypothesis. *Psychol Bull* 1985;98:310–57.
25 Shonkoff JP. Leveraging the biology of adversity to address the roots of disparities in health and development. *Proc Natl Acad Sci U S A* 2012;109 Suppl 2:17302–7.
26 Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *JAMA* 2009;301:2252–9.
27 Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav* 2012;106:29–39.
28 Power C, Hertzman C. Social and biological pathways linking early life and adult disease. *Br Med Bull* 1997;53:210–21.
29 Bond L, Butler H, Thomas L, *et al*. Social and school connectedness in early secondary school as predictors of late teenage substance use, mental health, and academic outcomes. *J Adolesc Health* 2007;40:357.
30 Soleimanpour S, Geierstanger S, Brindis CD. Adverse Childhood Experiences and Resilience: Addressing the Unique Needs of Adolescents. *Acad Pediatr* 2017;17:S108–14.
31 Garner A, Yogman M, Committee On Psychosocial Aspects Of C. Preventing Childhood Toxic Stress: Partnering With Families and Communities to Promote Relational Health. *Pediatrics* 2021;148:e2021052582.
32 Centers for Disease Control and Prevention. *Preventing adverse childhood experiences: leveraging the best available evidence*. Atlanta, GA: National Center for Injury Prevention and Control, Centers for Disease Control and Prevention, 2021.
33 Austin AE, Shanahan ME, Frank M, *et al*. Association of State Expansion of Supplemental Nutrition Assistance Program Eligibility With Rates of Child Protective Services-Investigated Reports. *JAMA Pediatr* 2023;177:294–302.
34 Farmer K. A review of economic policies to reduce and prevent child maltreatment and other adverse childhood experiences. 2022.
35 Centers for Disease Control and Prevention. Preventing adverse childhood experiences: data to action (PACE:D2A). 2022.
36 McBain RK, Levin JS, Matthews S, *et al*. The effect of adverse childhood experience training, screening, and response in primary care: a systematic review. *EClinicalMedicine* 2023;65:102282.
37 Crouch E, Probst JC, Radcliff E, *et al*. Prevalence of adverse childhood experiences (ACEs) among US children. *Child Abuse Negl* 2019;92:209–18.