ADVERSE CHILDHOOD EXPERIENCES: PREVALENCE AND ASSOCIATIONS WITH CARDIOMETABOLIC RISK AND PREMATURE MORTALITY AMONG YOUNG ADULTS IN THE UNITED STATES

by

KIRAN THAPA

(Under the Direction of Janani Rajbhandari-Thapa)

ABSTRACT

Epidemiological evidence has consistently demonstrated elevated risks of adverse health outcomes including premature mortality associated with cumulative exposure to adverse childhood experiences (ACEs). This dissertation research seeks to explore the current prevalence of ACEs and positive childhood experiences such as social support, and their interrelationship with cardiometabolic health and premature mortality in the United States (US). Two nationally representative population-based data sources, namely the National Survey of Children's Health (NSCH) and the National Longitudinal Study of Adolescent to Adult Health (Add Health) were used. First, using Add Health, a longitudinal cohort of school-aged children followed through young adulthood, we examined the association of ACEs with objectively measured cardiometabolic risk (CMR), and whether psychological health and social support modified the association between ACEs and CMR. Estimates are reported as beta coefficient or odds ratios (ORs) obtained from linear and logistic regressions. Second, using the same cohort, we examined the independent and joint associations of ACEs and social support with all-cause mortality through young adulthood. Analyses included Cox regressions to estimate mortality hazard ratios (HRs). Third, using NSCH data from 2016 to 2020, we examined state-level prevalence of ACEs and positive protective factors (PPFs) among US school-aged children. We

find that cumulative exposure to ACEs significantly predicted overall cardiometabolic risk during young adulthood (b: 0.053 [95% CI: 0.002, 0.105]; p=0.041) after adjustment for potential confounders including social support. Exposure to ≥2 ACEs was associated with 51% greater hazards for premature all-cause mortality in the overall cohort (aHR: 1.51; 95% CI: 1.02, 2.23), and particularly among low social support group (aHR: 1.78; 95% CI: 1.15, 2.77). About 1 in 10 US children aged 6-17 experienced >2 ACEs (of 9 total) and ≤5 PPFs (of 7 total), with significant variations across states. To conclude, ACEs are highly prevalent among US children, and ≥2 ACEs are associated with elevated cardiometabolic risk and premature mortality through young adulthood. Social support modified the association of ACEs with obesity and premature mortality. Interventions that target to increase perceived social support in addition to ACEs prevention may reduce cardiometabolic risk and early deaths among young adults in the US.

INDEX WORDS: Adverse childhood experiences, National Survey of Children's Health, National Longitudinal Study of Adolescent to Adult Health, Cardiometabolic risk, Premature mortality

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DEDICATION

To my parents, Maya Thapa and Hum Bahadur Thapa, in loving memory

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CHAPTER I: INTRODUCTION

Adverse childhood experiences (ACEs)

It has been long known that health is a function of physical, mental, social, and emotional experiences. And health is dynamic, meaning the physical, mental, emotional, and social factors that produce health interact in numerous ways to impact health across the lifespan. Moreover, the role of non-medical factors such as social, economic, and environmental determinants in shaping a person's health has been increasingly recognized, some estimates suggesting that they may account for over 50% of the population health outcomes. [1-3] The scope of social epidemiology as such is ever growing, and the application of life-course approach to study the relationship of social structures, institutions, and relationships with health outcomes is increasingly viewed as critical to advance understanding of the field and generate evidence to translate into public health impact. In contrast to a focus on a relatively specific exposure or disease (e.g., cardiovascular, or nutritional epidemiology), social epidemiologists focus on the complex features of the social, economic, and physical environment, for example, racial and ethnic disparities, structural racism, poverty, intergenerational trauma, and the social connections and networks.[4-7]

Stressful experiences in early life, especially during critical stages of childhood and adolescence, have been studied for many decades in relation to health and developmental outcomes[8-11]. What constitutes a stressful experience is specific to an individual and multifaceted depending on factors ranging from the individual's neuro-biological or physiologic functioning to broad environmental and contextual determinants. However, certain stressors as experienced by a population, especially at a young age, have shown to consistently predict poor health outcomes across the life course.[11-13] Different terminologies, such as 'adverse childhood experiences (ACEs)', 'childhood maltreatment and household dysfunction', 'early life adversity'

or ‘early life stressors’ have been used to identify a range of experiences and events that one experiences during early years of development and are demonstrated to have negative impact on physical and mental health.[14-16] The World Health Organization (WHO) defines adverse childhood experiences (ACEs) as “some of the most intensive and frequently occurring sources of stress that children may suffer early in life”.[17] Similarly, the Centers for Disease Control and Prevention (CDC) defines adverse childhood experiences (ACEs) as “potentially traumatic events that occur in childhood (0-17 years)”.[18] These definitions are quite broad because the sources of trauma or toxic stress can be manyfold. While scientific research is constantly evolving to address definitional and conceptual gaps, a bulk of childhood adversity research thus far have primarily focused on the child/ adolescent’s familial and social environment. The commonly assessed ACEs include child abuse and neglect, household challenges such as parental incarceration, parental psychopathology, household substance drug use, and exposure to different forms of violence at home, school, or neighborhood.

The seminal 1994 ACEs study conducted in the United States (US) included seven categories of ACEs, as reported retrospectively by the adults: history of psychological abuse, physical abuse, sexual abuse, household substance abuse, household mental illness, violent treatment of mother, and criminal behavior in the household.[19] The study found a significant dose-response relationship between number of ACEs and the odds for all the leading causes of deaths including smoking, drug abuse, depression, suicide, obesity, heart disease, cancer, and multiple health outcomes. Since then, multiple studies have investigated a variety of other stressors in the child’s multi-dimensional environment such as poverty, homelessness, exposure to neighborhood violence, racism, and bullying as potentially traumatic experiences, and have linked these to poor health outcomes.[20] ACEs such as child abuse and neglect are considered complex trauma and are independently associated with many adverse physical and psychological outcomes including long-term disability. Some of these stressors such as racism and historical trauma are deeply rooted in social constructs, systemic inequalities, and public

policies. Despite increased understanding of the link between ACEs and health outcomes, this has also led to a considerable between-studies variation in assessment of ACEs and their health impact.[21,22] Scientists have developed different frameworks to understand how different adversities may heighten the risk for toxic stress response, by focusing on different aspects such as the effect of specific adversity, cumulative exposure to adversities, chronicity and recency of the exposure, or the occurrence of exposure in specific developmental period.[23,24] And despite predominant focus of the scientific literature on ACEs and negative stress, less is known about the positive aspects of the social and physical environment in which children live and grow. The policy statement of the American Academy of Pediatrics (AAP) on childhood toxic stress emphasizes on the eco-bio-developmental model to understand both adverse as well as positive childhood experiences, as they both may have similar biological embedding to affect child development.[25] In recent years, ACEs have been increasingly viewed from a life-course perspective to better understand stressors occurring as early as gestation and their health impact during childhood, adolescence, adulthood, and later life. Research has also explored how adversity persists across generations to predict consistently poor health outcomes and contribute to systemic inequality.[26,27]

Adverse childhood experiences are common among both young and adult populations in the US. The prevalence of at least one ACE and more than two ACEs retrospectively self-reported by adults aged >=18 years in the Behavioral Risk Factor Surveillance System (BRFSS) were 57.8% and 21.5% respectively.[28] Similarly, in a population cohort of older adults in the Health and Retirement Study, the prevalence of at least one ACE was 35%, but only four ACEs were assessed.[29] Certain population groups experience greater number of adversities compared to others. Those in younger age groups, Blacks and multiracial individuals, females, low-income, lower educational attainment, and sexual minorities consistently report greater exposure to adversities during childhood.[28,30-32] Among young population, child maltreatment is more common among children younger than 3 years, and among girls.[33,34] A systematic review of the

meta-analyses found that childhood adversity is a leading contributor to morbidity and mortality in the US, accounting for approximately 439,072 deaths annually, through associations with leading causes of death including heart disease, cancer, and suicide.[35] ACEs have cascading, cumulative and long-term consequences; exposure to ACEs, especially during sensitive periods of development leads to adverse health and socio-economic outcomes that extend over the lifespan and across generations (43,44).[14,36-39]

ACEs and cardiometabolic health

The genetic predispositions and early life environment jointly determine the neural responses to stress (amygdala reactivity), psychological and social resources, which in turn affect our autonomic, neuroendocrine (cortisol reactivity), and immune systems through interacting dysregulations leading to physical and mental health problems. Repeated activation of the biological stress regulatory systems during early childhood due to exposure to harsh early environments compromise the functioning of these systems ultimately conferring health risks. In animal experimental studies involving rats, social isolation and hypervigilance was associated with increase in incidence of mammary tumors and compromised immune response to stress.[40,41] In humans, social isolation, loneliness, and low social support – which are alternative forms of social adversity – have been studied in relation to biomarkers of inflammation, cardiometabolic health, and cumulative physiological dysregulation as measured by allostatic load.[42-44] A study demonstrated that social isolation increased the risk of inflammation, hypertension, and overall physiological dysregulation in both early and later life, in a magnitude similar to physical inactivity or chronic risk factors.[45] Exposure to stressors during early life including relationships deficits, dysfunctional household, and violence can activate the immune, neuroendocrine, and metabolic pathways leading to cardiovascular and other chronic diseases. This relationship, in majority of the published literature, has been examined among older adults among which morbidity and mortality rates are relatively high. However, emerging evidence

shows that early life experiences including maltreatment and poverty may activate biological stress response systems at the time and increase risks for inflammation and cardiovascular diseases throughout adulthood.[45-47] Similarly, specific adversity such as those who reported parental incarceration in childhood had higher allostatic load scores compared to those without a history of parental incarceration.[48] A meta-analysis examining the associations between childhood adversity and adult cardiometabolic diseases found that cumulative childhood adversity was modestly associated with adult cardiometabolic disease (HR = 1.42, 95% CI [1.20, 1.67]; OR = 1.36 [1.27, 1.46]), with effects somewhat stronger for CVD clinical outcomes (hypertension, coronary heart disease, ischemic heart disease, myocardial infarction, stroke, cerebrovascular disease).[49] More studies are needed to assess the cardiometabolic health impact of the different types of and timing of childhood adversities during early adulthood.

ACEs and psychological health

Childhood adversity has been shown to be longitudinally associated with a range of psychological and mental health outcomes including cognitive functioning, depression, anxiety, suicide, substance use disorder, and psychotic disorder.[15,16,50-53] A cohort study of 548,721 adolescents and young adults in Sweden found a dose-response risks for suicide associated with cumulative adversity exposure (suicide risks, as incidence rate ratios, for specific adversity ranged from 1.6 (95% CI: 1.1, 2.4) for residential instability to 2.9 (95% CI: 1.4, 5.9) for suicide in the family).[51] Studies in both animals and humans have suggested that the hypothalamic-pituitary-adrenal (HPA)-axis regulation is one mechanism through which childhood adversity negatively affects health outcomes.[52] Several research have suggested that accumulation of hair cortisol may be a valid and reliable biomarker of chronic psychosocial stress as it captures cortisol exposure over extended periods of time that may occur through HPA-axis activity.[54] Studies have observed correlations between hair cortisol concentrations and experiences of serious life events and perceived stress, concluding that hair cortisol concentration could serve

as a biomarker of major life stressors .[54,55] A meta-analysis reporting the strength of associations between adversity – which included wide-ranging experiences including maltreatment, domestic violence, and exposure to war, and natural disasters – and hair cortisol concentration reported a small but significant positive association.[55] Some studies have also reported association between elevated levels of hair cortisol and cardiometabolic risk factors such as body mass index, waist-hip ratio, systolic blood pressure, as well as cardiovascular diseases.[56]

Psychological resilience and cardiometabolic health

There is substantial evidence that identify the negative effect of psychological distress on health.[57-59] Psychological resilience, in simple terms, is 'thriving amid adversity' i.e., maintaining positive psychological functioning amidst exposure to early adversity expected to bring about negative sequelae. From a developmental perspective, psychological resilience can be defined as 'the capacity of individuals exposed to early adversity to maintain or achieve positive psychological health in adulthood'.[60] Identifying resilience promoting factors may inform prevention and intervention targets to reduce the effects of early life adversity including chronic disease burden among adults. Only a handful of studies have empirically tested the role of psychological resilience in the association between early adversity and adult cardiometabolic outcomes – the results from most studies suggesting that psychological resilience may confer protection against development of cardiometabolic diseases into adulthood.[61] However, longitudinal population-based studies are scarce, and resilience has not been studied in the context of early life adversity. Therefore, it is not clear whether cardiometabolic conditions are associated with psychological resilience in the context of early life adversity. In the US, one study using data from the Midlife in the United States (MIDUS) study found that psychologically non-resilient individuals had 43% higher odds [95% CI: 1.10, 1.85] of incident cardiometabolic outcomes and elevated cardiometabolic risk [RR: 1.13, 95% CI: 1.01 to 1.27] in midlife

compared to resilient individuals.[62] Prospective evidence examining psychological resilience to childhood adversity and cardiometabolic health is limited among young adults.

Social support and cardiometabolic health

Two aspects of social support – structural (e.g., frequency or diversity of social contact) and functional (e.g., perceived support) have been primarily studied in public health.[63] Perceived social support has been shown to be the most reliable and strong predictor of well-being.[64] A broader view of the health-promoting effects of social support suggests that perceived support largely operates via a stress-buffering model, with the greatest and most reliable benefits of support found under conditions of psychological stress, when the support is most needed.[65] Extant literature suggests that strong social connections and supportive relationships play a crucial role in promoting healthier lifestyle behaviors, including regular physical activity, balanced nutrition, and adherence to medical recommendations, which collectively contribute to lower cardiometabolic risk.[66-68] Moreover, social support serves as a buffer against stress, a significant contributor to cardiometabolic disorders.[69,70] Recognizing the pivotal role of social support in cardio-metabolic health underscores the importance of fostering strong interpersonal relationships and supportive environments in preventive healthcare strategies.

ACEs and mortality

Few studies have reported increased risk of mortality associated with ACEs in European and US population cohorts, especially among middle and old age adults.[71-79] Using the National Longitudinal Study of Adolescent to Adult Health (Add health) cohort in the US, two studies have investigated correlates of suicide deaths among youths, finding greater suicide deaths among those who reported family member suicide, expulsion from school, running away from home, using weapon, smoking, and exhibited risk factors for externalizing behaviors such as violence, bullying, aggression.[80,81] However, due to small number of suicide deaths, statistical

power was limited to detect significant difference, and perform multivariate analyses. Another study using same cohort found that drunk driving is the major risk factor for premature mortality (OR = 2.48; 95% CI: 1.12–5.50).[82]

Social support and mortality

Studies have consistently demonstrated that individuals with greater perceived social support and strong support networks tend to live longer than those who are socially isolated or lacking in support.[83,84] Social support can influence mortality through various pathways, including its impact on mental health, health behaviors, and physiological processes.[85] Current evidence highlights the importance of promoting safe, supportive, and nurturing relationships to reduce mortality, such as, caregiver-child connection, school connectedness, policy support, etc.

Innovation

Much of the past literature have examined ACEs in retrospective among adult respondents, increasing the likelihood for recall bias and misclassification bias.[86] However, evidence of the impact of ACEs is not well-understood during emerging adulthood when young adults are transitioning to bearing adult roles and responsibilities including personal identity, social relationships, family obligations, and work responsibilities. ACEs predispose children at risk for poor socioeconomic opportunities during young adulthood, further perpetuating the negative consequences across this important developmental transition. Evidence is limited on the potential effects of specific adversities during childhood and adolescence, and their health effects during young adulthood. Despite a strong retrospective association between ACEs and cardiometabolic risk factors, little is known about the prospective impact of ACEs on early behavioral markers of cardiometabolic risk including obesity and diabetes in young adulthood. This study examined a rich set of adversities experienced during childhood and adolescence and assess their link to cardiometabolic health and all-cause mortality outcomes. Because most

children who experience ACEs do not develop adverse health outcomes later in life, understanding of heterogeneity in the effects of different types of stressors could shed light on the potential mechanisms through which childhood adversities affect risk for poor adult physical and psychological health outcomes. It is important to identify factors that protect or buffer children from developing adverse health outcomes related to ACEs exposure. How positive experiences may interact in explaining the relationship between ACEs and health outcomes in unclear. Examining the potential interaction effects of ACEs and social support may help to better illuminate disease risk factors and better identify target interventions to improve long-term health outcomes. Using a nationally representative prospective cohort of early adolescents who are followed through young adulthood, we prospectively examined the independent and joint associations of ACEs and perceived social support with cardiometabolic disease risk and all-cause premature mortality through young adulthood. Additionally, we utilized another nationally representative survey to assess the current national and across-state prevalence of ACEs and positive protective factors (PPFs) among children and adolescents in the US.

Conceptual framework

This dissertation research is guided by a conceptual model adapted from the American Heart Association.[87] ACEs could affect health and mortality outcomes through three potential interacting mechanisms – biological, psychological, and behavioral (Figure 1.1). First, biological embodiment of ACEs can occur through disruptions in biological processes and functioning of children including immune, metabolic, neuroendocrine, and autonomic nervous systems, which in turn may lead to cardiometabolic risk factors and disease. Second, ACEs could increase or exacerbate the mental health problems among children such as anxiety, depression, and post-traumatic stress disorder (PTSD), which may then predispose children to early manifestations of cardiometabolic diseases (e.g., increased blood pressure, adiposity, elevated glucose level) including mortality outcomes. Third, children exposed ACEs are at greater risk of engaging in

poor health behaviors and maladaptive coping (e.g., smoking, inactivity, alcohol use, poor sleep, unhealthy eating) which may in turn affect life-course physical and mental health. The conceptual model also recognizes the moderating role of modifiable positive protective factors (e.g., parental warmth, social support) that could buffer the effect of ACEs on morbidity and mortality. Other non-modifiable factors including sex, race/ethnicity, and genetic predisposition may exacerbate or protect children from the adverse health effects of childhood adversity.

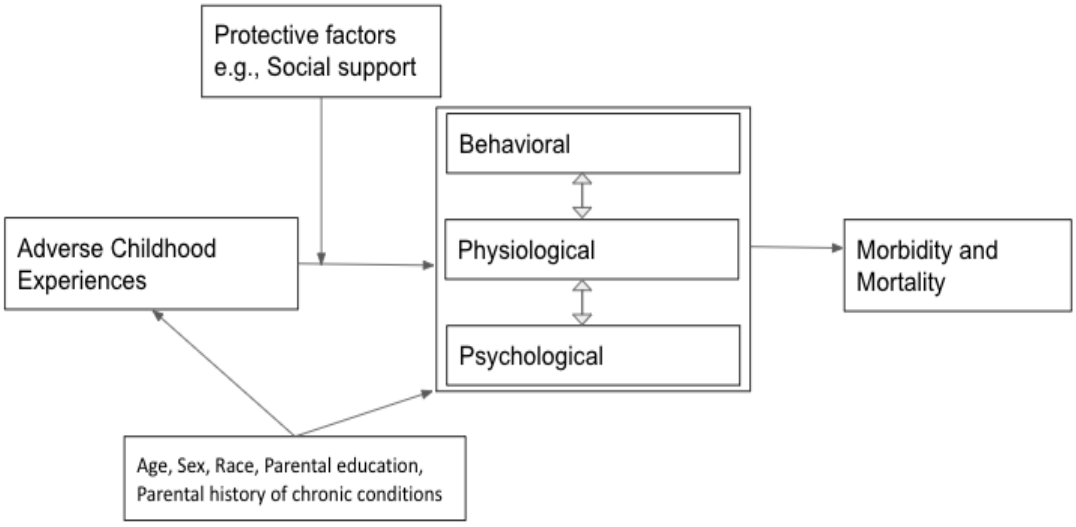

Figure 1.1 Conceptual Framework

Specific Aims

The overarching goal of this dissertation research was to conduct a systematic epidemiological investigation to provide a better understanding of young adulthood cardiometabolic health risk and all-cause mortality effect associated with ACEs and social support, and further assess the current scenario of ACEs and protective factors among children in the US. The specific aims and hypotheses of the study were:

<u>Aim 1 (Analytical):</u>

Assess the effect of ACEs on cardiometabolic risk during young adulthood and examine the role of psychological resilience and social support. We hypothesize that ACEs is significantly

associated with increased cardiometabolic risk during young adulthood. We also hypothesize that psychological health and social support modify the associations between ACEs and cardiometabolic risk. Furthermore, we hypothesize that resilience to adversity (i.e., positive psychological health following the experience of adversity) significantly predicts young adulthood cardiometabolic risk.

Aim 2 (Analytical):

Evaluate the associations of ACEs with all-cause premature mortality. We hypothesize that cumulative exposure to ACEs is significantly associated with higher all-cause mortality through young adulthood. Further, we hypothesized that childhood social support buffers the mortality hazards associated with ACEs.

Aim 3 (Descriptive):

Examine the national and across-state prevalence of ACEs and positive protective factors in the US.

Datasets description

Aim 1 and 2: For Aim 1, data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) was utilized (68). Add Health used school-based design to sample 20,745 adolescents enrolled in 7th through 12th grade (age 12-18 y) in the 1994-1995 academic year. The adolescents were prospectively followed through adulthood. Add Health used a multistage, stratified cluster design to sample public and private high schools and feeder schools (middle and junior high) across the United States in 1994-95 – 79% (*n* = 152 schools, 7^{th}-12^{th} graders) of the recruited schools participated, and all students attending these schools were invited to complete in-home surveys (participants survey & parent survey). To date, five waves of data have been completed: Wave I (N=20,745, age 12-18 y) began in 1994-95. Among those eligible

for follow-up, 14,738 (89%) completed Wave II in 1996, 15,197 (age 18-26 y) (77%) completed Wave III in 2001-02, 15,701 (age 24-32 y) (80%) completed Wave IV in 2008-09, and 12,300 (age 33-43 y) (72%) completed Wave V in 2016-18. Add Health ensures representativeness of the US adolescent population with regard to urbanity, region, school size, school type (public or private), and ethnicity. Add Health collects extensive longitudinal data on respondents' social, economic, psychological, and physical well-being along with contextual data on family, neighborhood, school, friendships, and community. It thus provides unique opportunities to study how environmental exposures and behaviors in early adolescence are linked to health and well-being in adulthood. From wave IV, Add Health started collecting data on biomarkers of physiological functioning. In Wave V, mortality surveillance was carried out to identify and track deceased participants in the Add Health study. This provided a unique opportunity to study exposure-health/mortality associations in a nationally representative sample. Further information on the Add Health study design and sampling procedures can be found at in the website (https://addhealth.cpc.unc.edu/about/).

For Aim 1, we utilized data from wave I, III (2001-2002), IV (2008-2009), and V to examine the associations of ACEs, psychological resilience, and social support with objectively measured cardiometabolic risk as implicated in biomarkers in young adulthood (wave V). While some measures of ACEs were assessed prospectively at baseline (wave I) when participants were 18 years or less, or retrospectively at wave III and IV when participants were early adults. For Aim 2, we examined associations of prospectively collected ACEs and perceived social support at baseline (wave I) when participants were younger than 18 years of age with all-cause mortality through young adulthood when participants were in their mid to early 40s. To produce nationally representative estimates, survey weights were used in all analyses. The weights in Add Health account for unequal probability of sample selection, clustering of individuals within schools as well as differential attrition across waves.

Aim 3:

For Aim 3, five years of combined data (2016-2020) from the National Survey of Children's Health (NSCH) was utilized to conduct a population-based cross-sectional study. The NSCH is representative of the civilian, noninstitutionalized children and adolescents' population aged 0-17 years in the US. The survey includes parent-reported measures of child health and well-being indicators including family, neighborhood, and social factors. The NSCH has been conducted annually since 2016. The data is publicly available through the Data Resource Center for Child and Adolescent Health (DRC) website (www.childhealthdata.org). The study was limited to children and adolescents between 3 and 17 years of age. In NSCH data, the missing values are imputed for several demographic variables such as child sex, race, Hispanic origin, parent education, household size, and total family income using hot-deck imputation and sequential regression imputation methods. All analyses used weighting, stratification, and clustering to account for complex survey design and obtain nationally representative estimates.

CHAPTER II: ADVERSE CHILDHOOD EXPERIENCES, PSYCHOLOGICAL HEALTH, SOCIAL SUPPORT AND YOUNG ADULTHOOD CARDIOMETABOLIC RISK

Thapa, K., Rajbhandari-Thapa, J., Shen, Y., Vall, E.A., & Cordero, J. To be Submitted to the Journal of American Heart Association.

Abstract

No studies have evaluated the comprehensive nature of ACEs, psychological health, and social support in relation to CMR among young adults, who may be at an underlying risk for development of adverse cardiometabolic outcomes. The objectives of this study were: (i) To examine the associations between ACEs and young adulthood CMR, (ii) To evaluate whether psychological health and social support modify the relationship between ACEs and CMR. We used longitudinal data from the National Longitudinal Study of Adolescent to Adult Health. Cumulative ACEs exposure was defined using ten ACEs items. Psychological health was measured using depressive symptoms and self-esteem measures. Social support assessed the perceived level of support from parents, family, and school. CMR was measured using six objectively collected biomarkers from in-home health examination. Survey weighted linear and logistic regression models were run to estimate the main and interaction effects of ACEs with psychological health and social support on CMR. Over two decades of follow-up, ACEs was significantly associated with overall cardiometabolic risk (b: 0.053 [95% CI: 0.002, 0.105]; p=0.041), general obesity (adjusted odds ratio (aOR): 1.26; 95% CI: 1.13, 1.41), and central obesity (aOR: 1.22; 95% CI: 1.07, 1.38) during young adulthood, after adjustment for baseline socio-demographic, bio-behavioral and psychological variables. Social support and psychological health did not modify young adulthood cardiometabolic risk. Sex and social support modified the association between ACEs and general obesity. ACEs may become biologically embedded as children enter young adulthood. Intervening on upstream ACEs exposures through population-based programs may prevent development of downstream cardiometabolic diseases.

Introduction

Cardiometabolic disorders present a significant public health problem in the United States. Among 35-44-year-olds, heart diseases are the top four leading causes of death and have

significant economic costs.[88,89] The American Heart Association (AHA) has emphasized seven risk factors – smoking, poor diet, physical activity, body weight, blood pressure, cholesterol, and blood glucose – as 'Life's Simple 7' to promote lifestyle modification and help achieve ideal cardiovascular health.[90] While these are proximal factors that lead to poor cardiometabolic outcomes, early life experiences including socioeconomic disadvantage early in life can lead to proximal risk factors of cardiovascular diseases.[91,92] Adverse childhood experiences (ACEs) are potentially traumatic experiences such as abuse, neglect, and parental/household challenges that have been shown to affect health and development across the lifespan, including cardiovascular diseases.[14,16,38,49,93,94] A growing body of scientific research has demonstrated the negative effect of adverse childhood experiences (ACEs) on cardiometabolic risk factors later in life, including smoking[95], obesity[96], physical inactivity[19,97], high blood pressure[98], and diabetes[99]. A recent review reported that adults with four or more ACEs have over twice greater risk of developing cardiovascular disease compared to those with no ACEs.[100]

One plausible pathway from exposure to negative stressors such as ACEs to poor adulthood cardiometabolic health occurs through the detrimental effect of ACEs on mental health and coping skills.[98,101,102] Poor mental health including depression and stress have been linked to behavioral risk factors for cardiometabolic diseases.[103-105] ACEs also amplify the associations between psychological distress and adult health outcomes.[106] In addition, the adverse cardiometabolic health effects of ACEs may be further explained by the intergenerational effect of ACEs on socio-economic and educational attainment and cumulative disadvantage across the life-course.[107-109] Some studies have longitudinally examined the positive dimensions of psychosocial environment such as social support, parent-child relationship, resiliency skills and socio-emotional skills in relation to cardiovascular and metabolic outcomes.[110-114] Social support has been shown to be independently associated with reduced cardiometabolic risk and alleviate the effect of ACEs and distress on cardiometabolic risk behaviors.[43,44,85,115] Social support in early life may facilitate the development of effective emotion regulation and coping strategies to

mitigate the impact of stress and reduce CVD risk factors associated with ACEs. However, we found no studies that have examined the psychological resilience in the context of prospectively ACEs among children in the US. Most prior research has relied on retrospective reports of ACEs among adult or elderly population. Although most young adults may not have yet developed cardiometabolic outcomes, they may be at an underlying risk which could be captured using allostatic load. Allostatic load, as derived from biomarkers of several physiological systems (multi-system physiological dysregulation) consistently predict mortality.[116] In addition, most studies have assessed a narrow range of adverse experiences. This study aims to evaluate the prospective associations of childhood adversity, perceived social support, and psychological resilience with young adulthood cardiometabolic outcomes among young adults in the US. Our hypotheses were: (i) exposure to ACEs is associated with greater risk of cardiometabolic risk factors and a composite cardiometabolic risk, (ii) Social support modifies the relationship between ACEs and cardiometabolic risk, (iii) Psychological health modifies the relationship between ACEs and cardiometabolic risk, and (iv) Those who are psychologically resilient to ACEs have lower cardiometabolic risk compared to those who are non-resilient.

Methods

Study population and setting

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health used school-based design to sample 20,745 adolescents enrolled in 7th through 12th grade (age 12-18 y) in the 1994 - 1995 academic year. The adolescents were prospectively followed through adulthood. Add Health collects extensive longitudinal data on respondents' social, economic, psychological, and physical well-being along with contextual data on family, neighborhood, school, friendships, and community. To date, five waves of data have been collected, with latest wave (Wave V) collected during 2016-18 (age 33-43 y). Of the 20,745 Wave I respondents, 12,300 participated in the Wave V survey data collection, of whom

5,381 completed an in-home health examination. Our analytic sample included respondents who had available biomarkers from the home health examination, had a valid sampling weight, had complete exposure and covariate information, and did not have a self-reported history of chronic health conditions (heart conditions, asthma, diabetes) in childhood (Figure 1.2). More information on the Add Health study design and sampling procedures can be found elsewhere (https://addhealth.cpc.unc.edu/about/).

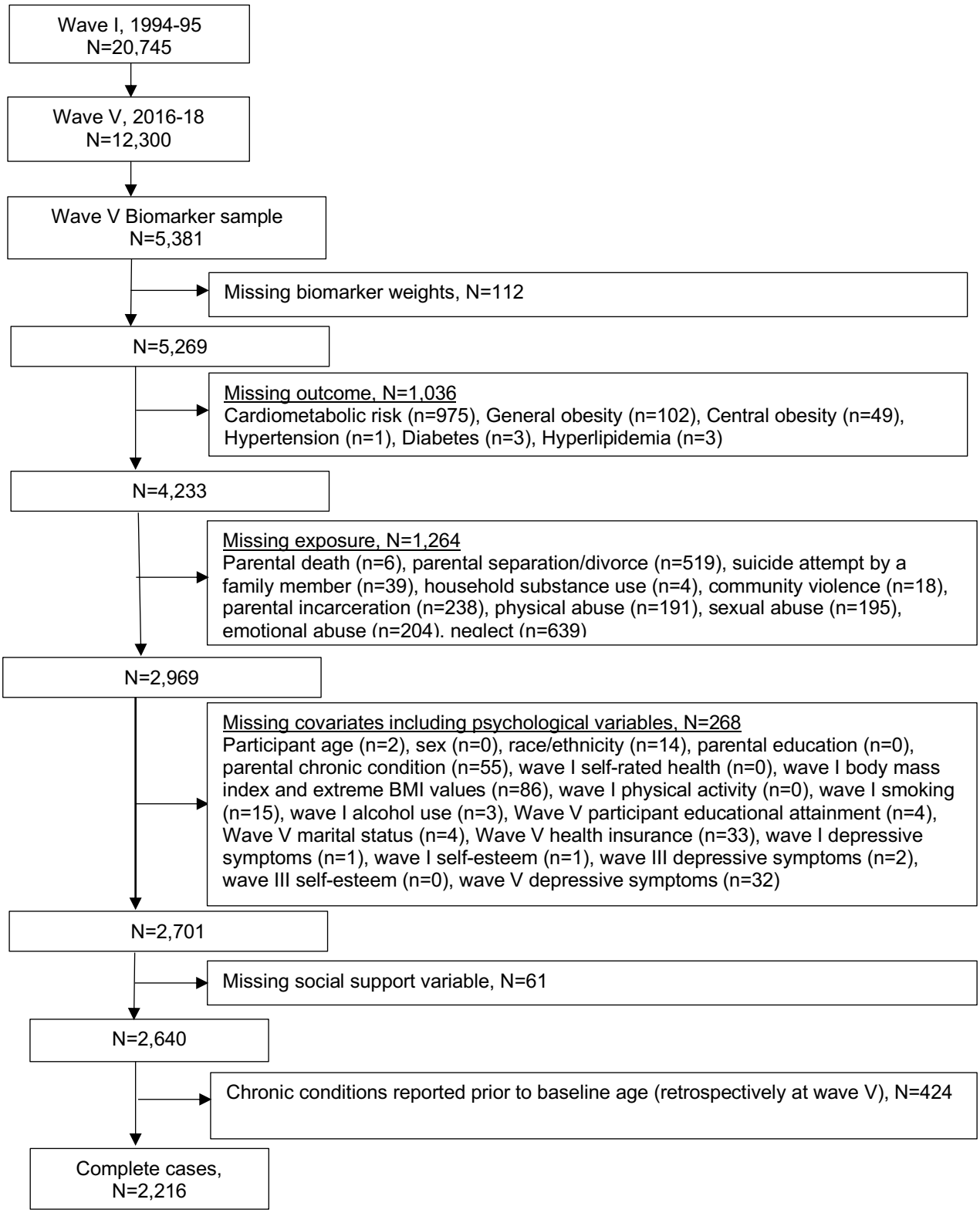

Figure 2.1 Study sample -- Aim 1

Exposures

Adverse childhood experiences (ACEs)

A total of 10 ACEs were included: parental death, parental divorce or separation, household substance use, suicide attempt by a family member, community violence, parental incarceration, physical abuse, sexual abuse, emotional abuse, and neglect (Table 2.1). A cumulative measure of ACEs score was created by summing responses to ten items (range 0-10). The ACEs score was tested as a continuous and a categorical variable.

Psychological resilience

Psychological resilience was characterized using ACEs exposure and early adult psychological health, encompassing distress and positive wellbeing.[62,117] Individuals were considered exposed to ACEs if they reported experiencing at least one ACEs. Consistent with prior research indicating greater risk of negative health outcomes among those exposed to any adversity (relative to none), we classified individuals into exposed (≥1 ACEs) and unexposed (0 ACEs) using cumulative adversity score.

The three items from the Center for Epidemiologic Studies Depression Scale (CESD)[118] and three items from the Rosenberg's self-esteem scale[119] included in the Add Health were utilized to assess psychological health at wave I and wave III. The three negative affect CES-D items assessed how often, in the last 7 days, participants felt: (i) depressed, (ii) sad, and (iii) could not shake off the blues, even with the help from your family and friends. Responses for CES-D items include ‘never or rarely’, ‘sometimes’, ‘a lot of the time’, and ‘most of the time or all of the time’. A cumulative score with possible values ranging from 0 to 9 was constructed. On self-esteem, respondents were asked whether they have good qualities, have a lot to be proud of, and like themselves as they are. Responses were captured using Likert scales which included ‘strongly agree’, ‘agree’, ‘neither agree nor disagree’, ‘disagree’, and ‘strongly disagree’. Items

were reverse coded to ensure higher scores reflect higher self-esteem. The cumulative self-esteem score was created with score ranging from 0-12.

Overall psychological health variable at two time points, wave I and III, was created by summing the inverse of standardized CES-D scores and standardized self-esteem scores, with higher scores reflecting better psychological health. We used binary ACEs measure and binary psychological health variable constructed at wave I and III to define a categorical psychological resilience variable by cross-classification: resilient (ACEs exposed, psychological health above median in wave I and III), non-resilient (ACEs exposed, psychological health below median in wave I and III), vulnerable (no ACEs, psychological health below median in wave I and III), and psychologically healthy (no ACEs, psychological health above median in wave I and III)). As an alternative parameterization, we also examined joint associations of a continuous childhood adversity exposure and a continuous standardized psychological health measure.

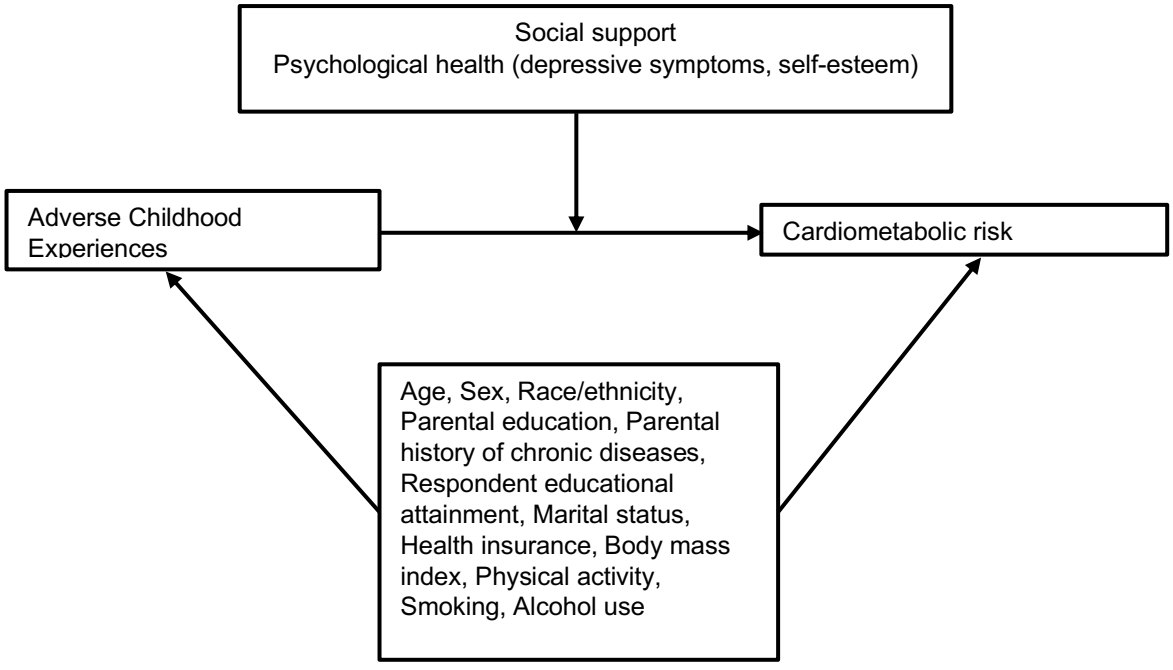

Figure 2.2 Directed Acyclic Graph (DAG) – Aim 1

Table 2.1 ACEs measures -- Aim 1

Variables	Question	Response options[#]	Response recategorization	Wave/s
Exposure: ACEs				
Parental death or bereavement	Is he [biological father] still living? Is she [biological mother] still living?	No Yes	0 = *No* 1 = *Yes*	I
Parental divorce	Parent Survey: "What is your current marital status?"	1 = single, never married 2 = married 3 = widowed 4 = divorced 5 = separated	0 = not divorced or separated 1 = divorced or separated	Parent survey (I)
Parental alcoholism	Parent Survey: "Does respondent's biological father currently have the following health problem: Alcoholism" Parent Survey: "Does respondent's biological mother currently have the following health problem: Alcoholism"	No Yes	0 = *No* 1 = *Yes*	Parent survey (I)
Household substance use	Are illegal drugs easily available to you in your home?	No Yes	0 = *No* 1 = *Yes*	I
Household mental illness (suicide attempts by a family member)	Have any of your family members tried to kill themselves during the past 12 months?	No Yes	0 = *No* 1 = *Yes*	I
Parental imprisonment/incarceration	Has your biological father ever served time in jail or prison? (Has/did) your biological mother ever (spent/spend) time in jail or prison? (Has/did) your [father figure] ever (spent/spend) time in jail or prison? (Has/did) your [mother figure] ever (spent/spend) time in jail or prison?	No Yes	0 = *No* 1 = *Yes*	IV
Physical abuse	How often had your parents or other adult caregivers slapped, hit, or kicked you?	One time Two times Three to five times Six to ten times	0 = *less than 3 times* 1 = *3 or more times*	IV

		More than ten times This has never happened		
Sexual abuse	How often had one of your parents or other adult caregivers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?	One time Two times Three to five times Six to ten times More than ten times This has never happened	0 = *1 or more times* 1 = *never*	IV
Emotional abuse	Before your 18th birthday, how often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved?	One time Two times Three to five times Six to ten times More than ten times This has never happened	0 = M*ore than 10 times* 1 = *never or less than 10 times*	IV
Physical neglect	How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing?	One time Two times Three to five times Six to ten times More than ten times This has never happened	0 = never of once 1 = *two or more times*	III
Community violence	During the past 12 months, did the following happen? If so, how often? You saw someone shoot or stab another person. Someone pulled a knife or gun on you. Someone shot, cut, or stabbed you	Never Once More than once	0 = Never 1 = Once or more	I

[#]Excludes refused, legitimate skip, and don't know

Outcomes

Cardiometabolic risk

The first outcome was a composite cardiometabolic risk score from wave V. The risk score was derived from a set of cardiometabolic biomarkers (Table 2.2), which have been extensively studied in previous studies of cardiometabolic risk and disease, including using Add health data (54,69-71).[49,87,120-122] The biomarkers were collected by trained health staff during in-home clinical visits and cover four domains of physiological systems: cardiovascular functioning (resting heart rate, systolic blood pressure, diastolic blood pressure), glucose metabolism (blood glucose), lipid metabolism (low-density lipoprotein (LDL) cholesterol), and inflammation (c-reactive protein). We standardized the values of each biomarker using z-scores, calculated the mean score, and then again standardized the resulting score to obtain final cardiometabolic risk measure.

Table 2.2 Biomarkers used to create a composite cardiometabolic risk score.

Cardiometabolic biomarkers	**Physiological system**
Resting heart rate (bpm)	Cardiovascular functioning
Blood glucose (mg/dL)	Glucose metabolism
Systolic blood pressure (mmHg)	Cardiovascular functioning
Diastolic blood pressure (mmHg)	Cardiovascular functioning
LDL-cholesterol (mg/dL)	Lipid metabolism
C-reactive protein	Inflammation

Cardiometabolic disease risk factors

Our secondary outcomes included five cardiometabolic disease risk factors: General obesity, Central obesity, Hypertension, Diabetes, and Hyperlipidemia (Table 2.3).

Table 2.3 Cardiometabolic risk factors definitions used in the study.

Cardiometabolic risk factors	Definitions used
General obesity	BMI>30 kg/m2
Central obesity	Waist circumference>102 cm for males and >88 cm for females
Hypertension	EITHER hypertension stage 1 or 2 i.e., systolic BP>= 140 or diastolic BP >=90 OR took an antihypertensive medication in the past 4 weeks OR has ever been diagnosed with high blood pressure or hypertension
Diabetes	EITHER fasting glucose >= 126 mg/dl OR non-fasting glucose >= 200 mg/dl OR HbA1c >= 6.5% OR a self-reported history of diabetes except during pregnancy OR has taken an anti-diabetic medication in the past 4 weeks
Hyperlipidemia	EITHER a self-reported history of high cholesterol or triglycerides OR taken an antihyperlipidemic medication in the past 4 weeks OR has a fasting TG concentration >= 500 mg/dl OR an LDL-C concentration >= 190 mg/dl

Covariates

A set of a-priori defined potential confounders were included as covariates in all the multivariate analyses. These included baseline sociodemographic variables and bio-behavioral variables, and young adulthood socioeconomic circumstances. The socio-demographic variables included participant's age at baseline interview, sex, race/ethnicity, parental educational attainment, and parental history of chronic condition. The age of the participant was measured in years (continuous), as the difference between baseline interview date and the date of birth. Because only the month and year of birth were available, we assigned 15 as the universal day of birth for those who reported their month and year of birth. We also divided age into 11-15 years and 15-18 years. Sex of the participant was coded as male or female. Race/ethnicity was coded as Hispanic, non-Hispanic White, non-Hispanic Black, or other. Parental education, defined as highest level of education attained by either father or mother, was coded as less than high

school, high school or GED, some college or 2-y college, or 4-y college or greater. Missing parental education in in-home survey with participants were replaced with parent survey responses. Parental history of chronic condition was coded as yes for those who reported having obesity or diabetes in the parent survey, and no for those who did not.

We also included the following bio- behavioral variables collected at baseline: self-rated health, body mass index (BMI), physical activity, smoking, alcohol use, and depressive symptoms. Self-rated health was binary coded as good or poor. BMI was calculated from the 2000 age and sex-standardized CDC growth charts and newer metrics that more accurately characterize BMIs above the CDC 97th percentile using self-reported height and weight. Extreme BMI values (z-score< -4 or z-score >8) were removed. BMI was classified into underweight (<5th percentile), normal weight (5th–85th percentile), overweight (85th–95th percentile), and obesity (≥95th percentile). Physical activity was measured using an index created by summing responses across three items that asked participants the frequency of engaging in different activities in the past week (‘Not at all’, ‘1 or 2 times’, ‘3 or 4 times’, and ‘5 or more times’) (range 0-9). The responses were standardized between-subjects using z-scores, with higher number indicating higher physical activity level. Smoking status was coded as ‘yes’ for those who reported having smoked cigarette at least one day in the past month, and as ‘no’ otherwise. Alcohol use was coded as ‘yes’ if the participant reported drinking at least 3-12 times in the past 12 months, and as ‘no’ otherwise. Depressive symptoms were assessed using a shortened version of Center for Epidemiological Studies Depression (CES-D) scale involving five items with responses as ‘never or rarely’, ‘sometimes’, ‘a lot of the time’, and ‘most of the time or all the time’. Responses were summed across items (range 0-15) and standardized using z-scores, with higher score indicating greater depressive symptoms. Further, we also examined young adulthood socioeconomic circumstances which could potentially mediate ACEs-cardiometabolic risk. These included educational attainment (less than high school/High school or GED/Some college

or 2-year college/4-year college or greater), marital status (Never married/Married/Divorced, Widowed or Separated), and health insurance status (Yes/No) collected at wave V.

Statistical analyses

We presented participant characteristics by ACEs exposure categories (0,1, ≥2 ACEs). We then estimated a series of linear and logistic regression models to examine the associations of ACEs, social support, and psychological resilience on overall cardiometabolic risk and factors during young adulthood. First, the main and interaction effects of ACEs and social support on cardiometabolic risk and risk factors were examined. Second, the associations between psychological resilience and cardiometabolic risk was examined. Third, we examined interaction between ACEs and psychological health on cardiometabolic risk. Lastly, we also tested for effect modification by sex and race/ethnicity on the associations between ACEs and cardiometabolic risk and risk factors.

A mix of the following covariates were included in several models in a stepwise fashion: first, baseline sociodemographic factors (age, sex, race/ethnicity, parental education attainment, and parental history of chronic diseases as potential confounders) were added, then baseline bio-behavioral factors (Self-rated health, BMI, physical activity, smoking, alcohol use) were added, then psychological factors were added, and finally, the adult circumstances at wave V including educational attainment, marital status, and health insurance coverage were added. All analyses used survey weights to account for the unequal probability of selection and differential attrition from baseline to wave V.

Results

About 53.5% of the respondents were exposed to at least one ACEs. Non-Hispanic Blacks, those with lower parental education, those with poor self-rated health, those with obesity, those reported smoking, and those who reported using alcohol at baseline were more likely to experience 2 or more ACEs (Table 2.4).

Table 2.4 Participant characteristics by ACEs at baseline.

Characteristic	0 ACE (N = 1,030)	1 ACE (N = 634)	2 or more ACEs (N = 552)	p-value
Age, in years	15.84 (1.68)	15.92 (1.60)	15.93 (1.62)	0.5
Age group				0.2
<=15y	348 (49%)	189 (27%)	172 (24%)	
>15y	682 (45%)	445 (30%)	380 (25%)	
Sex				0.2
Female	618 (47%)	362 (27%)	341 (26%)	
Male	412 (46%)	272 (30%)	211 (24%)	
Race/ethnicity				<0.001
Hispanic	109 (40%)	85 (31%)	80 (29%)	
Non-Hispanic Black	117 (32%)	124 (34%)	121 (33%)	
Non-Hispanic Others	49 (48%)	28 (27%)	26 (25%)	
Non-Hispanic White	755 (51%)	397 (27%)	325 (22%)	
Parental education				<0.001
4-y college or higher	536 (58%)	235 (25%)	159 (17%)	
High school/GED or less	286 (36%)	247 (31%)	256 (32%)	
Some college/2-y college	208 (42%)	152 (31%)	137 (28%)	
Parental history of obesity or diabetes	283 (45%)	186 (30%)	154 (25%)	0.7
Self-rated health (wave I)				<0.001
Good	1,001 (47%)	606 (29%)	510 (24%)	
Poor	29 (29%)	28 (28%)	42 (42%)	
BMI class (wave I)				0.005
Normal weight	807 (48%)	477 (28%)	407 (24%)	
Obesity	60 (33%)	57 (31%)	64 (35%)	
Overweight	135 (46%)	84 (29%)	72 (25%)	
Underweight	28 (53%)	16 (30%)	9 (17%)	
Physical activity, z-score (wave I)	0.06 (1.03)	0.02 (1.02)	0.00 (0.99)	0.7
Smoking (wave I)	177 (35%)	164 (33%)	162 (32%)	<0.001
Alcohol use (wave I)	232 (38%)	186 (31%)	191 (31%)	<0.001
Participant educational attainment (wave V)				<0.001
4-y college or greater	644 (57%)	287 (25%)	206 (18%)	
GED or high school	81 (33%)	73 (30%)	89 (37%)	
Less than high school	15 (28%)	19 (36%)	19 (36%)	
Some college/2-y college	290 (37%)	255 (33%)	238 (30%)	
Participant marital status (wave V)				<0.001
Divorced or separated or widowed	121 (42%)	75 (26%)	92 (32%)	
Married	729 (50%)	398 (27%)	324 (22%)	
Never married	180 (38%)	161 (34%)	136 (29%)	

Health insurance (wave V)	979 (47%)	595 (29%)	503 (24%)	0.009
Social support (wave I)				<0.001
High	328 (63%)	125 (24%)	65 (13%)	
Moderate	496 (45%)	334 (31%)	263 (24%)	
Low	207 (34%)	176 (29%)	224 (37%)	
Psychological health at wave I, quartiles				0.14
First	302 (49%)	166 (27%)	154 (25%)	
Fourth	228 (41%)	167 (30%)	155 (28%)	
Second	311 (49%)	178 (28%)	148 (23%)	
Third	189 (46%)	123 (30%)	95 (23%)	
Psychological health at wave III, quartiles				0.065
First	264 (47%)	159 (28%)	140 (25%)	
Fourth	215 (43%)	132 (26%)	152 (30%)	
Second	259 (47%)	164 (30%)	124 (23%)	
Third	292 (48%)	179 (29%)	136 (22%)	
Positive psychological functioning at wave I and wave III	219 (42%)	153 (30%)	144 (28%)	0.080
General obesity	396 (40%)	292 (30%)	293 (30%)	<0.001
Central obesity	456 (41%)	326 (30%)	321 (29%)	<0.001
Hypertension	255 (41%)	206 (33%)	160 (26%)	0.003
Diabetes	62 (36%)	52 (30%)	60 (34%)	0.003
Hyperlipidemia	140 (44%)	98 (31%)	83 (26%)	0.5
Cardiometabolic risk, z-score	-0.17 (0.96)	0.00 (1.00)	0.03 (1.03)	<0.001

[1] *Mean (SD); n (%)*
[2] *Kruskal-Wallis rank sum test; Pearson's Chi-squared test*
Numbers and percentages in the table are unweighted.

Associations with overall cardiometabolic risk

ACEs, social support, and cardiometabolic risk

Cumulative exposure to ACEs was associated with increased cardiometabolic risk during young adulthood independent of social support, after adjustment for baseline socio-demographic and bio-behavioral and psychological risk factors (b=0.053 [95% CI: 0.002, 0.105]; p=0.041). Further adjustment for young adulthood socioeconomic circumstances including health insurance status, the associations between ACEs and cardiometabolic risk became lower, and statistically non-significant, suggesting that adult socioeconomic circumstances potentially mediate the risk

of ACEs on cardiometabolic health (Table 2.5). Social support did not significantly modify the relationship between ACEs and cardiometabolic risk (Table 2.6).

Table 2.5 Main effects of ACEs and social support on cardiometabolic risk

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Characteristic	**β (95% CI); p**	**β (95% CI); p**	**β (95% CI); p**	**β (95% CI); p**	**β (95% CI); p**
Number of ACEs	0.097 (0.035, 0.158); p=0.002	0.063 (0.009, 0.116); p=0.023	0.053 (0.001, 0.105); p=0.046	0.053 (0.002, 0.105); p=0.041	0.032 (-0.017, 0.080); p=0.201
ACEs categories					
0 ACE	Ref.	Ref.	Ref.	Ref.	Ref.
1 ACE	0.152 (0.016, 0.288); p=0.028	0.082 (-0.046, 0.209); p=0.208	0.061 (-0.065, 0.187); p=0.343	0.057 (-0.070, 0.185); p=0.377	0.016 (-0.116, 0.148); p=0.810
2 ACEs	0.095 (-0.109, 0.300); p=0.358	0.015 (-0.179, 0.209); p=0.879	0.007 (-0.182, 0.197); p=0.939	0.009 (-0.181, 0.199); p=0.925	-0.036 (-0.223, 0.151); p=0.703
3 ACEs	0.130 (-0.116, 0.375); p=0.298	0.017 (-0.231, 0.265); p=0.893	-0.014 (-0.265, 0.237); p=0.910	-0.015 (-0.266, 0.235); p=0.903	-0.053 (-0.304, 0.197); p=0.674
4 or more ACEs	0.578 (0.220, 0.936); p=0.002	0.442 (0.137, 0.748); p=0.005	0.409 (0.108, 0.710); p=0.008	0.409 (0.113, 0.705); p=0.007	0.284 (0.001, 0.567); p=0.049
Social support categories					
High	Ref.	Ref.	Ref.	Ref.	Ref.
Moderate	0.002 (-0.131, 0.134); p=0.979	-0.017 (-0.143, 0.109); p=0.784	-0.034 (-0.161, 0.094); p=0.599	-0.036 (-0.164, 0.091); p=0.572	-0.032 (-0.161, 0.096); p=0.617
Low	0.039 (-0.151, 0.229); p=0.684	0.039 (-0.130, 0.208); p=0.652	-0.007 (-0.179, 0.165); p=0.939	-0.005 (-0.176, 0.166); p=0.957	-0.035 (-0.209, 0.138); p=0.688
Social support					
high	—	—	—	—	
low	-0.007 (-0.141, 0.128); p=0.920	-0.005 (-0.131, 0.122); p=0.942	-0.022 (-0.153, 0.108); p=0.736	-0.025 (-0.156, 0.105); p=0.702	-0.042 (-0.171, 0.087); p=0.518

Model 1: Unadjusted (ACEs and social support as individual predictors)
Model 2: adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.
Model 3: further adjusted for baseline self-rated health, BMI class, physical activity, smoking, alcohol use.
Model 4: further adjusted for psychological health at baseline and wave III.
Model 5: further adjusted for participant educational attainment, marital status, and health insurance status.

Table 2.6 Joint effects of ACEs and social support on cardiometabolic risk

Characteristic	Beta	95% CI	p-value
MODEL 1			
Number of ACEs	0.078	-0.037, 0.194	0.182
Social support			
high	—	—	
low	0.009	-0.139, 0.158	0.903
Number of ACEs * Social support			
Number of ACEs * low	-0.019	-0.156, 0.117	0.779
MODEL 2			
Standardized ACEs score	0.114	0.035, 3.277	0.001
Standardized social support score	0.004	-0.062, 0.069	0.891
Standardized ACEs score * Standardized social support score	-0.004	-0.066, 0.058	0.896

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

When ACEs were analyzed as a categorical variable, we did not observe consistent associations with cardiometabolic risk, although having 4 or more ACEs seemed to predict poor cardiometabolic health in models 1-4 (Table 2.7).

Table 2.7 ACEs categories and cardiometabolic risk

	Model 1			Model 2		
Characteristic	**Beta**	**95% CI**	**p-value**	**Beta**	**95% CI**	**p-value**
Number of ACEs						
0	—	—		—	—	
1	0.159	0.025, 0.292	0.020	0.085	-0.042, 0.212	0.186
2	0.103	-0.105, 0.312	0.329	0.018	-0.178, 0.214	0.854
3	0.141	-0.107, 0.388	0.263	0.027	-0.225, 0.279	0.831
4	0.557	0.076, 1.04	0.024	0.414	0.015, 0.812	0.042
5	0.455	-0.280, 1.19	0.223	0.332	-0.455, 1.12	0.405
6	0.986	0.115, 1.86	0.027	0.832	0.161, 1.50	0.016
Social support						
high	—	—		—	—	
low	-0.010	-0.144, 0.124	0.879	-0.006	-0.132, 0.120	0.930
	Model 3			**Model 4**		
Characteristic	**Beta**	**95% CI**	**p-value**	**Beta**	**95% CI**	**p-value**

Number of ACEs						
0	—	—		—	—	
1	0.062	-0.064, 0.188	0.332	0.059	-0.068, 0.186	0.359
2	0.006	-0.185, 0.198	0.947	0.009	-0.181, 0.200	0.924
3	-0.010	-0.264, 0.244	0.936	-0.010	-0.264, 0.244	0.936
4	0.395	0.003, 0.787	0.048	0.388	-0.004, 0.780	0.052
5	0.206	-0.570, 0.981	0.600	0.231	-0.535, 0.998	0.551
6	0.806	0.128, 1.48	0.020	0.813	0.150, 1.48	0.017
Social support						
high	—	—		—	—	
low	-0.022	-0.152, 0.108	0.741	-0.024	-0.154, 0.106	0.710

	Model 5			Model 1: Unadjusted Model 2: adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes. Model 3: further adjusted for baseline self-rated health, BMI class, physical activity, smoking, alcohol use. Model 4: further adjusted for psychological health at baseline and wave III. Model 5: further adjusted for participant educational attainment, marital status, and health insurance status.
Characteristic	**Beta**	**95% CI**	**p-value**	
Number of ACEs				
0	—	—		
1	0.018	-0.114, 0.150	0.787	
2	-0.037	-0.224, 0.150	0.695	
3	-0.049	-0.302, 0.204	0.701	
4	0.234	-0.163, 0.631	0.245	
5	0.147	-0.560, 0.853	0.681	
6	0.759	0.050, 1.47	0.036	
Social support				
high	—	—		
low	-0.040	-0.169, 0.088	0.537	

Psychological resilience and cardiometabolic risk

We did not observe significant associations between psychological resilience and cardiometabolic risk among our sample of young adults (Table 2.8).

Table 2.8 Associations between psychological resilience to ACEs and cardiometabolic risk

Characteristic	**N**	**Beta**	**95% CI**	**p-value**
ACEs exposure, High psychological functioning	297	—	—	
ACEs exposure, Poor psychological functioning	890	-0.071	-0.272, 0.130	0.486
No ACEs exposure, High psychological functioning	220	-0.143	-0.403, 0.117	0.279
No ACEs exposure, Poor psychological functioning	811	-0.146	-0.353, 0.060	0.163

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

ACEs, psychological health, and cardiometabolic risk

In alternative models, we did not observe significant interaction effects between ACEs exposure and psychological health in predicting cardiometabolic risk among our sample of young adults (Table 2.9).

Table 2.9 Joint effects of ACEs and psychological health on cardiometabolic risk

Characteristic	Beta	95% CI	p-value
Number of ACEs	0.044	-0.019, 0.108	0.168
Positive psychological functioning	-0.031	-0.194, 0.132	0.708
Number of ACEs * Positive psychological functioning	0.061	-0.078, 0.201	0.385

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

Furthermore, we did not find effect modification of ACEs and cardiometabolic risk by sex or race/ethnicity (results not shown).

The results showing the associations between ACEs and cardiometabolic risk factors (i.e., separately for general obesity, central obesity, hypertension, diabetes, and hyperlipidemia) including the potential effect modification by social support, sex and race/ethnicity is shown in the appendix A. In summary, we found that ACEs is associated with increased risk of general and central obesity independent of social support. We also observed some potential effect modification by sex the ACEs-general obesity relationship and ACEs-diabetes relationship and by race/ethnicity on central obesity, hypertension, and hyperlipidemia.

Discussion

The findings of this study are consistent with findings from prior studies that have examined the cumulative impact of ACEs on cardiometabolic outcomes.[49,70,87,98,122,123] ACEs are thought to biologically embed into our skin through altered physiological processes. Our results indicate that ACEs may become biologically embedded sooner than later, as soon as children enter young adulthood.[12,38,124] Previous studies using Add health cohort have used cardiometabolic

risk, and long-term CVD prediction models as outcomes based on risk factors collected at wave IV or V.[87,120,125,126] Our results further delineate potential heterogeneity in cardiometabolic health effects of ACEs by examining potential interactions with social support, sex, and race/ethnicity. Our person-focused hypothesis of psychologically resilient individuals having lower risk of cardiometabolic risk was not empirically supported. However, the role of psychological factors including emotional-cognitive resources and social environment to affect cardiometabolic health needs further exploration using life-course and multilevel epidemiological data.

Evidence is clear that exposure to adversity and adverse life events during sensitive period of childhood has lifelong negative consequences on overall health, learning, and behavioral outcomes.[31,93] Several studies have attempted to understand the factors that contribute to the risk-resilience continuum and pathways through which adversity and resilience during childhood impact health and developmental outcomes over time.[127,128] Negative experiences such as ACEs deprive children of the needed stimulation critical for their development and leads to poor health outcomes over time through physiological and psychological mechanisms. In contrast, having safe, supportive, and nurturing relationships promote child wellbeing and protect children from negative effects of ACEs. Achieving ideal markers of cardiometabolic health can be protective against developing cardiovascular disease and deaths. The current discourse in ACEs research should extend beyond prevention of ACEs to prevention of early risk of cardiometabolic diseases by promoting positive psychosocial environment and focusing on preventable sources of childhood stress.[21,129] Addressing upstream ACEs through equitable population- and policy-based interventions is crucial to promote better cardiometabolic health across all groups.

In the US, screening for ACEs in primary care settings is increasingly viewed as an important element of trauma-informed care to identify patients who may be experiencing toxic stress due to ACEs. There is no routine clinical screening of childhood trauma or ACEs yet. A 2024 review by the United States Preventative Services Task Force (USPSTF) concluded that the evidence

is insufficient to assess the effect of primary care interventions to prevent child maltreatment.[130] In 2020, California became the first US state to begin screening for ACEs in all outpatient clinics, albeit some critics questioning if the potential benefits outweigh the harms.[131,132] A systematic review of four ACEs screening studies reported that screening for ACEs increases identification of adversity and may increase referral to services, but the effect on health outcomes was not determined due to limited data.[133] As proposed in numerous childhood adversity literature, development of a validated and objective risk assessment tool is needed to identify children more accurately who are at risk of maltreatment, trauma, or poor health outcomes.

There are some limitations to this study. When examining childhood adversities, we used cumulative risk approach (i.e., examining number of adversities as an exposure) which is useful to test dose-response association.[134] Examining ACEs as individual exposures (specificity approach) or a cluster of exposures (dimensional approach) may provide additional insights into heterogeneity of effects across individuals with differing trajectories and potential mechanisms.[135,136] Future research should attempt to utilize longitudinal datasets with time-varying exposures and individual and household-level covariates to estimate the causal effect of ACEs and psychosocial environment on cardiometabolic outcomes.

Conclusions

The study highlights the significant association between ACEs and adverse cardiometabolic health outcomes including obesity and diabetes among young adults in the US. Understanding the impact of ACEs on cardiometabolic health has significant implications for intervention and prevention strategies. It underscores the critical need for a holistic approach to addressing the impact of childhood trauma on cardiometabolic health, with implications for both research and public health practice. Early identification and intervention for children exposed to ACEs, along with targeted interventions to address the psychological and social consequences of trauma,

may help mitigate the long-term health consequences. Policies and programs to strengthen social support networks, improve access to mental health services, and implement trauma-informed care approaches across healthcare and social service systems may help alleviate long-term adverse health outcomes.

CHAPTER III: ADVERSE CHILDHOOD EXPERIENCES AND PREMATURE MORTALITY: EFFECT MODIFICATION BY SOCIAL SUPPORT

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Abstract
We examined the direct and joint associations of exposure to adverse childhood experiences (ACEs) and perceived social support on the risk of all-cause mortality through young adulthood. The sample consisted of 12,421 children from the National Longitudinal Study of Adolescent to Adult Health (Add Health) who were 11 to 17 years at baseline (1994-95) and followed up until 2016-18. Multivariable Cox proportional hazards models and Aalen's additive hazards models were used to estimate hazard ratios (HRs) and hazard differences (HDs) per 10,000 individuals per year. Over two decades of follow-up, ≥2 ACEs and low social support were directly associated with increased risk of mortality when controlling for ACEs and other potential confounders. We found significant sub-additive and sub-multiplicative interaction between ACEs and social support. Compared to those without ACEs exposure and high level of perceived social support, those reporting ≥1 ACEs and low level of social support had over twice greater hazards of premature mortality, after adjusting for baseline socio-demographic variables. This represented about 9.4 additional deaths per 10,000 individuals per year. Promoting social support interventions in addition to efforts to preventing ACEs could be effective strategies to reduce premature deaths among young adults in the United States.

Introduction
Childhood is a sensitive period in human development characterized by rapid biological, neurocognitive, and emotional development, along with changes in social relationships. Exposure to adverse family and community environments during this critical developmental period gets 'biologically embedded' through altered physiological, behavioral, and psychological mechanisms affecting lifelong health and development. [12,124] Adverse childhood experiences (ACEs) are potentially traumatic experiences occurring during childhood such as abuse or neglect, caregiver mental illness or substance use problems, and domestic and community violence. A recent review of meta-analysis estimated that ACEs accounted for 15% of total

mortality in the United States (US), and it is associated with leading causes of deaths including heart diseases, cancer, and suicide. [35] Annual economic costs attributable to ACEs approximate to about $750 billion. [137] Findings of increased mortality risk during early and mid-adulthood associated with ACEs have been reported using data from the British Birth Cohort [72,75], Danish Nationwide Registers [73], and Swedish cohort [77], which included large samples and prospective assessment of ACEs, highlighting the need to study possible mechanisms and identify intervention targets in the US.

The current literature on ACEs and mortality is limited and mostly based on non-representative small datasets which lack generalizability. Others are based on retrospective recall of ACEs or face other methodological challenges such as short follow-up periods. A 2009 study among adult members of the Kaiser Permanente health insurance group in the US found no clear association between ACEs and mortality. However, two-thirds of the study sample were aged ≥50 years at baseline, the follow-up was short (10 years), and the sample was not representative of the US population. [79] The studies utilizing the Midlife Development in the United States (MIDUS) and the Health and Retirement Survey (HRS) found a greater risk of mortality during adulthood associated with retrospective reports of ACEs (mean age at baseline 47 years), particularly, with physical, and emotional abuse. [78,138] Few studies have prospectively assessed ACEs during childhood and examined the associated risk of mortality during adulthood. 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. [71] More prospective evidence is needed on the long-term consequences of ACEs among young adults.

While much scientific focus has been given to understanding the health effects of ACEs, there is limited data on the modifiable factors in the social environment in which ACEs occur, which is important to identify potential areas for public health and policy interventions. Some positive childhood experiences, such as childhood social support and positive psychosocial resources,

have been shown to lower the risk of mortality through adulthood. [83,85,139,140] Limited studies have examined the buffering effect of perceived social support on the ACEs–health relationship, especially on mental health outcomes. [141-143] However, 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 the current limitations of evidence on the relationship between ACEs, perceived social support, and premature mortality in the US, we utilized data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) - a prospective cohort of a nationally representative sample of US adolescents followed through young adulthood. We examined direct and joint associations of ACEs and perceived social support with risks of premature mortality. Our specific hypotheses were: (i) ACEs and perceived social support are directly associated with greater all-cause mortality through young adulthood, and (ii) Childhood social support modifies the risk of mortality associated with ACEs, i.e., social support confers significantly greater protection against mortality risk among those exposed to ACEs compared to those non-exposed. We conducted a series of primary and sensitivity analyses to test our hypotheses and discussed potential public health and policy implications of the findings.

Methods

Study population and setting

We used data from the Add Health. In 1994, Add Health used a stratified school-based design to select a nationally representative sample of high schools and a paired feeder school (such as middle school feeding into the high school) from a list of all high schools in the US. An in-school questionnaire was administered to all students who attended the selected schools during 1994–95 school year. Then, from the school rosters, a core sample was selected for in-depth interviews at home with adolescents and one parent. A total of 20,745 adolescents, representative of the US adolescents enrolled in grade 7-12 (age 11-21 y) in 1995, constituted

the baseline sample/wave for this study. The exposure variables (ACEs and social support) and covariates for this study are drawn from the in-home interviews with adolescents and a parent at wave I. There were five waves of follow-up. Wave V (age 33-43 y) during 2016-19 included mortality surveillance that identified and tracked deceased participants. A total of 12,421 participants who had complete information on exposure, outcome, and covariates were included in our primary analysis. Those with missing sampling weights (*n* = 1,821), missing age (n=14), those 18 years of age or older at baseline (*n* = 3,216), those missing ACEs information (*n* = 2,129), those missing social support information (n=313), and those without complete covariate information (*n* = 831) were excluded (Figure 3.1).

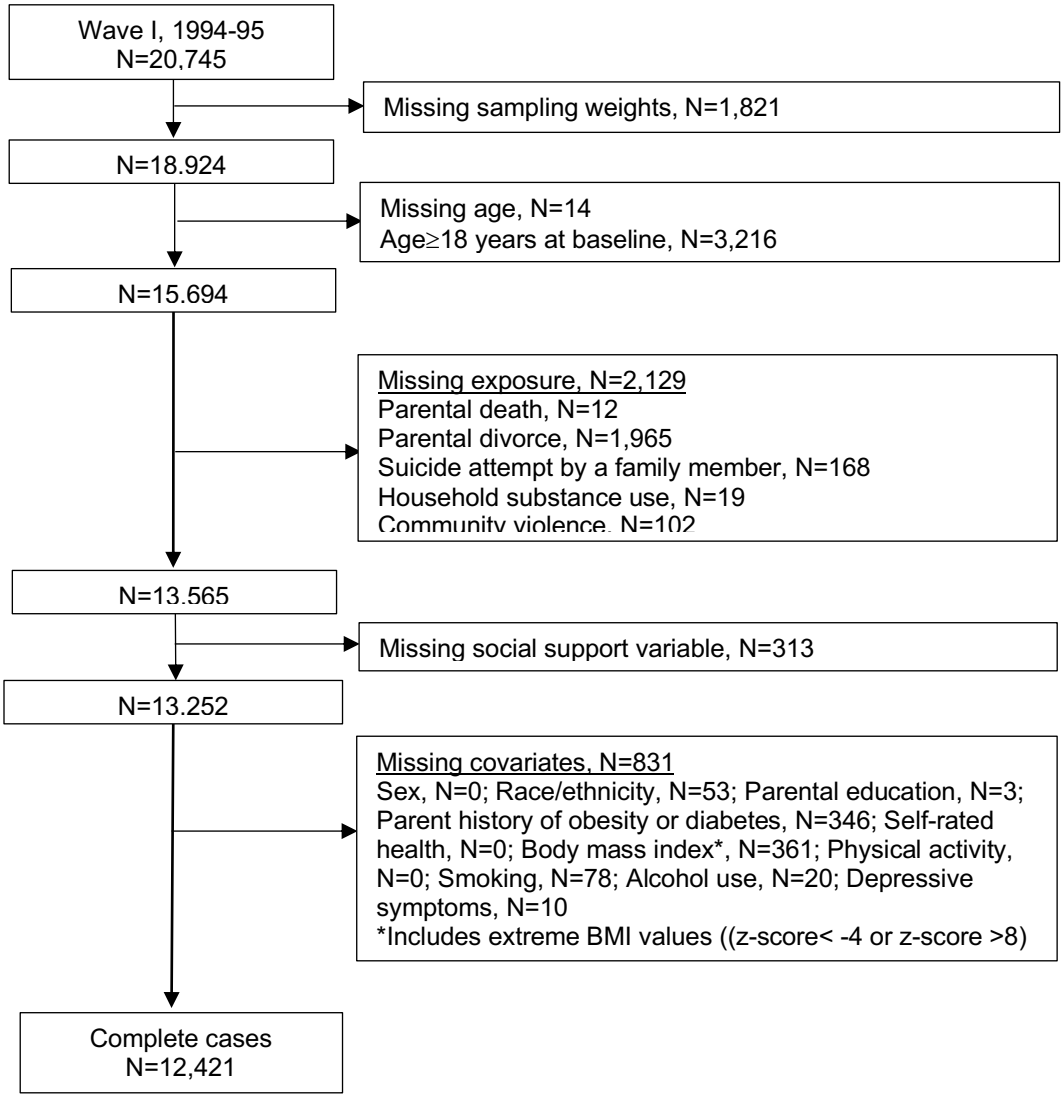


Figure 3.1 Study sample – Aim 2

Exposure measures

Adverse childhood experiences (ACEs)

We included five ACEs available in the Add Health experienced at the time of data collection in 1995. They included 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. [144,145] Data on parental divorce and parental substance use were obtained from a parent survey, while data on other ACE items were obtained from in-home interviews with the adolescent participant. All exposures were collected prospectively at baseline. The original questions and response options, including weighted prevalence of each ACEs, is presented in Table 3.1. We dichotomized the responses for each ACE item to indicate presence or absence of the ACE exposure. We then summed the dichotomous response to each item to create a cumulative ACEs score which ranged from 0 to 5, with higher value indicating exposure to higher number of ACEs. To test dose-response relationship between ACEs and mortality, we categorized ACEs into three categories: 0, 1, and ≥2 ACEs. For interaction analyses, to gain statistical power, we used a binary ACEs variable (0 and ≥1 ACEs).

Table 3.1 Measures and prevalence of adverse childhood experiences (Add health Wave I) – Aim 2

Variables	Question and response options	Response recategorization	Prevalence, weighted
ACE1: Parental death/bereavement	Is he [biological father] still living? (No/Yes) Is she [biological mother] still living? (No/Yes)	0= Both parents alive 1= Either one of the parents is not alive	4.03%
ACE2: Parental divorce or separation	*Parent Survey*: "What is your current marital status?" (Single, never	0 = Not divorced or separated	19.90%

	married/Married/Widowed/Divorced/Separated)	1 = Divorced or separated	
ACE3: Household substance use	*Parent Survey*: "Does respondent's biological father currently have the following health problem: Alcoholism" (No/Yes) *Parent Survey*: "Does respondent's biological mother currently have the following health problem: Alcoholism" (No/Yes) Are illegal drugs easily available to you in your home? (No/Yes)	0= No parental alcoholism or drug availability at home 1= Either alcohol use by one of the parents or drug availability at home	17.70%
ACE4: Suicide attempt by a family member	Have any of your family members tried to kill themselves during the past 12 months? (No/Yes)	0 = *No* 1 = Yes	4.53%
ACE5: Community violence	During the past 12 months, did the following happen? If so, how often? *You saw someone shoot or stab another person*. (Never/Once/More than once)	0 = Never witnessed, received threat, or victimized 1 = Either witnessed, received threat, or victimized once or more than once	18.80%
	During the past 12 months, how often did each of the following things happen? *Someone pulled a knife or gun on you.* (Never/Once/More than once)		
	During the past 12 months, how often did each of the following things happen? *Someone cut or stabbed you.* (Never/Once/More than once) During the past 12 months, how often did the following happen? *Someone shot you.* (Never/Once/More than once)		

Social support

We defined social support using a summary indicator involving nine items that asked respondents the perceived level of support from parents, family, and school at baseline (Table 3.2). 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 to 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. Respondents who fell in the top 75th percentile were coded as having

received ‘high social support’, those in the bottom 25th percentile as having ‘low social support’, and those falling in between 25th to 75th percentile were coded as having received ‘moderate social support’. The correlation matrix between ACEs items and social support items is presented in Appendix Table B.1.

Table 3.2 Measures and prevalence of perceived social support items (Add health Wave I) – Aim 2

Question and response options	Response recategorization	% respondents reporting 4 or 5, weighted
SS1: Most of the time, your mother/father is warm and loving toward you.	1= Strongly disagree; 2= Disagree; 3= Neither agree nor disagree; 4= Agree; 5= Strongly agree	56.1%
SS2: You are satisfied with the way your mother/father and you communicate with each other.		48.3%
SS3: You are satisfied with your relationship with your mother/father.		55.4%
SS4: Your family understands you.	1= Not at all; 2= Very little; 3= Somewhat; 4= Quite a bit; 5= Very much	56.3%
SS5: You have fun with your family.		63.8%
SS6: Your family pays attention to you.		72.1%
SS7: You feel close to people at your school.	1= Strongly disagree; 2= Disagree; 3= Neither agree nor disagree; 4= Agree; 5= Strongly agree	68.4%
SS8: You feel like you are a part of your school.		75.5%
SS9: You are happy to be at your school.		67.7%

SS1, SS2, and SS3 were each derived from two items – each item relating to paternal and maternal support separately. Those reporting support from either of the parents were coded as receiving 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 15th day of the month as the universal day of death for all deceased. Information about ascertainment of decedents in the Add health cohort can be found elsewhere [146]. We assigned the start date of

follow-up as the date of baseline interview (wave I). Participants were censored at death or the end of the study period. For all deceased, we calculated the follow-up time or the survival time, which is the outcome of interest, in number of days (converted to years dividing by 365), using the difference between the baseline interview date in 1994-95 and the date of death. For those alive, we calculated the follow-up time as the difference between the baseline interview date and NDI matching date, i.e., December 31, 2019. There was effectively no loss to follow-up. For analysis purposes, we coded the death indicator as 1 for respondents who died during follow-up and as 0 for respondents alive at follow-up.

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 the multivariable analyses. This included sociodemographic variables and health-related behavioral variables. The socio-demographic variables included participant's age at interview (wave I), sex, race/ethnicity, and parental educational attainment and parental history of chronic condition also from wave I. The age of the participant was measured in years (continuous), as the difference between baseline interview date and the date of birth. Because only the month and year of birth were available, we assigned 15th day of the month as the universal day of birth for those who reported their month and year of birth. We also divided age into 11-14 years and 15-17 years to estimate potential effect of the timing of exposure to ACEs. Sex of the participant was coded as male or female. Race/ethnicity was coded as Hispanic, non-Hispanic White, non-Hispanic Black, or other. Parental education, defined as the highest level of education attained by either father or mother, was coded as less than high school, high school or GED, some college or 2-y college, or 4-y college or greater. Missing parental education in the in-home survey with adolescent participant was replaced with parent survey responses at Wave I. Parental history of chronic condition was coded as yes for those who reported having obesity or diabetes in the parent survey, and no for those who did not.

We also included the following bio- behavioral variables collected at baseline: body mass index (BMI), physical activity, smoking, alcohol use, and depressive symptoms among the adolescent participant. BMI was calculated from the 2000 age and sex-standardized CDC growth charts and newer metrics that more accurately characterize BMIs above the CDC 97th percentile using self-reported height and weight. BMI was classified into underweight ($<5^{th}$ percentile), normal weight (5^{th}–85^{th} percentile), overweight (85^{th}–95^{th} percentile), and obesity ($\geq 95^{th}$ percentile). Physical activity was measured using an index created by summing responses across three items that asked participants the frequency of engaging in different activities in the past week ('Not at all', '1 or 2 times', '3 or 4 times', and '5 or more times') (range 0-9). The responses were standardized between-subjects using z-scores, with higher number indicating higher physical activity level. Smoking status was coded as 'yes' for those who reported having smoked cigarette at least one day in the past month, and as 'no' otherwise. Alcohol use was coded as 'yes' if the participant reported drinking at least 3-12 times in the past 12 months, and as 'no' otherwise. Depressive symptoms were assessed using a shortened version of Center for Epidemiological Studies Depression (CES-D) scale involving five items with responses as 'never or rarely', 'sometimes', 'a lot of the time', and 'most of the time or all the time'. Responses were summed across items (range 0-15) and standardized using z-scores, with higher score indicating greater depressive symptoms.

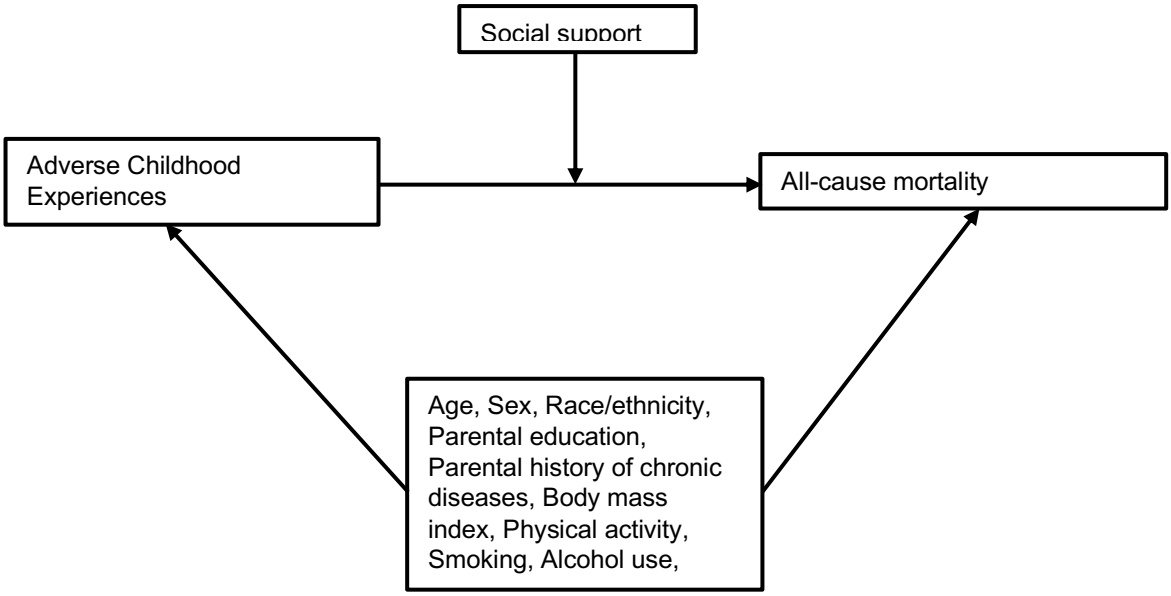


Figure 3.2 Directed Acyclic Graph (DAG) – Aim 2

Statistical analysis

We compared baseline characteristics of the respondents across ACEs exposure categories (0, 1, and ≥2 ACEs) using descriptive statistics. We then fitted Cox proportional hazards models and Aalen's additive hazards models to examine the effect of ACEs and social support, while adjusting for the confounding effect of each other and other variables (direct effect). In Cox models, hazard is modelled as an exponential function of explanatory variables thus providing a relative measure of mortality; in Aalen's additive models, hazard is modelled as a linear function of explanatory variables thus providing absolute measure of mortality. [147,148] Sociodemographic variables including bio-behavioral risk factors were evaluated as potential confounders. We presented the mortality hazard estimates without and with adjustment for bio-behavioral variables (body mass index, smoking, alcohol use, physical activity, and depressive symptoms) to respectively provide high and low estimates for HRs and HDs, as bio-behavioral factors may act as the mechanisms through which ACEs affect mortality. We modelled ACEs and social support both as continuous standardized scores (mean=0, SD=1) and as categorical variables

(0/1/≥2 ACEs; low/moderate/high social support). The estimates from Cox models were reported as hazard rate ratios (HRs), and the estimates from the Aalen models were reported as hazard rate differences (HDs) per 10,000 person-years associated with one-unit standard deviation change in ACEs or social support score (continuous), or in exposed group compared to a reference group (categorical). For interaction analyses, we first plotted unadjusted Kaplan-Meir survival curves for 0 vs ≥1 ACEs stratified by a categorical social support variable. To test for departures from multiplicativity of effects (statistical interaction) and additivity of effects (causal interaction), we included an interaction term between ACEs and social support variable in Cox and Aalen models respectively. The assumptions of proportional hazards for Cox models and time-invariant effects for Aalen models were satisfied. 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, stratification. We used 2-sided $P<0.05$ to infer statistical significance.

In sensitivity analyses, we reported mortality hazard estimates for males and females separately. For females, because number of deaths was less than 10 in a cell for joint effect analyses, we presented results using 50th percentile as a social support cutoff. We compared estimates limiting our sample to include only those who died after two years and after five years of baseline assessment to reduce potential confounding bias from pre-existing disease. Then, to evaluate if exposure to ACEs and/or social support after 18 years of age alters the mortality hazard estimates, we repeated the analyses without baseline age restriction.

Results

About 46% of the respondents were exposed to at least one ACEs, and 15% were exposed to at least 2 ACEs at baseline. Exposure to ACEs was higher among 16-18-year-olds, males, non-Hispanic Blacks, those whose parents had lower educational attainment, whose parents had a history of obesity or diabetes, those who smoked, used alcohol, those with higher CES-D

scores, and those with low level of social support. Perceived social support was lower among 16-18-year-olds, females, those whose parents had lower educational attainment, smokers, alcohol users, and those with greater exposure to ACEs; depressive symptom scores were higher among moderate social support group. (Table 3.3; Appendix Table B.2)

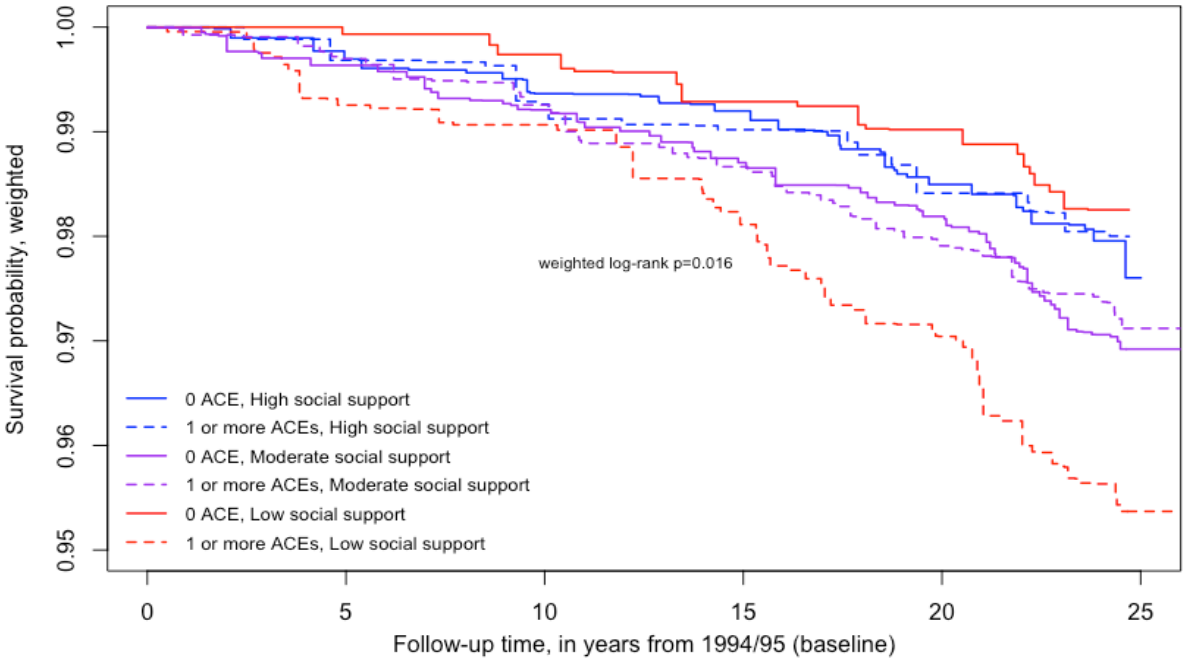
Table 3.3 Participant characteristics, overall and by ACEs exposure categories

	N[a] (%)	Exposure to ACEs		
		0 ACEs	1 ACEs	≥2 ACEs
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)
Age group				
11-15	4,163 (42.0%)	55.9%	29.3%	14.8%
16-18	8,258 (58.0%)	52.9%	31.2%	15.9%
Sex				
Female	6,267 (48.8%)	56.4%	29.3%	14.3%
Male	6,154 (51.2%)	52.1%	31.4%	16.5%
Race and ethnicity				
Hispanic	1,938 (10.9%)	48.2%	33.1%	18.7%
Non-Hispanic White	7,148 (71.1%)	57.8%	28.6%	13.6%
Non-Hispanic Black	2,569 (14.1%)	38.3%	38.3%	23.4%
Non-Hispanic Other	766 (3.9%)	62.3%	26.8%	10.9%
Parental education				
High school/ GED or less	5,204 (43.7%)	46.9%	33.4%	19.7%
Some college/2-y college	2,683 (22.0%)	52.7%	31.7%	15.7%
4-y college or greater	4,534 (34.3%)	64.4%	25.8%	9.8%
Parental obesity or diabetes				
Yes	3,220 (26.3%)	51.0%	31.1%	17.8%
No	9,201 (73.7%)	55.3%	30.1%	14.6%
Body Mass Index classification				
Underweight	379 (3.2%)	57.9%	31.8%	10.3%
Normal weight	9,105 (72.9%)	54.8%	30.3%	14.9%
Overweight	1,678 (13.9%)	52.3%	31.0%	16.8%
Obesity	1,259 (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)
Smoking				
Yes	3,022 (25.5%)	42.6%	34.4%	23.0%
No	9,399 (74.5%)	58.1%	29.0%	12.8%
Alcohol use				
Yes	3,426 (27.4%)	42.4%	34.5%	23.1%
No	8,995 (72.6%)	58.6%	28.8%	12.5%

Depressive symptoms, Mean CESD-5 score (SD)	2.3 (2.4)	2.0 (2.1)	2.6 (2.5)	3.0 (2.8)
Social support				
High	2,967 (25.2%)	65.2%	26.3%	8.5%
Moderate	6,126 (49.3%)	54.6%	30.6%	14.8%
Low	3,328 (25.4%)	42.5%	34.0%	23.5%

[a]Unweighted; Row percentages and mean values in the table are weighted estimates.

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 accident (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, 10.55]; 1 ACEs: 11.80 [95% CI: 9.82, 14.19]; ≥2 ACEs: 15.30 [95% CI: 12.15, 19.26]) and lower among those with greater perceived social support (high: 9.29 [95% CI: 7.32, 11.80]; moderate: 10.96 [95% CI: 9.41, 12.78]; low: 12.02 [95% CI: 9.86, 14.67]). Stratified survival curves indicated significantly lower overall survival over time among those exposed to ≥1 ACEs with low level of social support (log-rank p-value=0.016). (Figure 3.3)



	Deaths/No deaths, unweighted	Person-years, unweighted	Deaths per 10,000 individuals per year, unweighted	Unadjusted hazard ratio (95% CI), weighted	Unadjusted hazard difference per 10,000 individuals per year (95% CI), weighted
0 ACEs, High social support	43/1,812	45,070	9.5	1.00 (ref.)	0.00 (ref.)
≥1 ACEs, High social support	24/1,088	27,037	8.9	0.94 (0.53, 1.67)	-0.50 (-5.36, 4.35)
0 ACEs, Moderate social support	78/3,182	79,187	9.9	1.44 (1.05, 1.97)	3.83 (1.67, 5.98)
≥1 ACEs, Moderate social support	85/2,781	69,463	12.2	1.34 (0.85, 2.10)	2.95 (-1.08, 6.98)
0 ACEs, Low social support	21/1,388	34,373	6.1	0.82 (0.44, 1.97)	-1.57 (-6.80, 3.66)
≥1 ACEs, Low social support	76/1,843	46,288	16.4	2.18 (1.16, 4.11)	10.30 (2.65, 17.94)

Figure 3.3 Unadjusted Kaplan-Meier survival curves, stratified by ACEs and social support

In a co-adjusted model including ACEs, social support and socio-demographic variables, exposure to ≥2 ACEs was associated with 37% increased hazards for all-cause mortality through young adulthood ≤43 years compared to those not exposed to ACEs. This increased hazard corresponds to about 5.1 additional deaths per 10,000 individuals per year. Similarly, those with low and moderate level of perceived social support had 58% and 38% greater

hazards of premature mortality compared to those with low social support level – the increased hazards corresponding to about 4.4 and 3.2 additional deaths per 10,000 individuals per year respectively. Further adjustment for bio-behavioral factors resulted in a higher but non-significant effect associated with exposure to ≥2 ACEs, and moderate or low social support compared to their respective counterparts (Table 3.4). Among bio-behavioral factors, body mass index and smoking behavior were the major confounders of the association of ACEs and social support with mortality.

Table 3.4 Direct effect estimates for hazards of premature mortality associated with ACEs and social support.

ACEs	Without adjustment for bio-behavioral factors[a]		With adjustment for bio-behavioral factors[b]	
	HR [95% CI]; P	HD [95% CI]; P	HR [95% CI]; P	HD [95% CI]; P
Continuous				
Score (standardized)	1.11 [1.02, 1.20]; P=0.011	1.33 [0.13, 2.53]; P=0.030	1.06 [0.98, 1.15]; P=0.150	0.77 [-0.41, 1.95]; P=0.200
Categorical				
0 ACEs	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
1 ACEs	1.00 [0.83, 1.21]; P=0.970	-0.18 [-2.35, 2.00]; P=0.872	0.95 [0.78, 1.15]; P=0.620	-0.93 [-3.09, 1.22]; P=0.757
≥2 ACEs	1.37 [1.07, 1.76]; P=0.012	4.46 [0.62, 8.30]; P=0.023	1.22 [0.95, 1.55]; P=0.113	2.88 [-0.73, 6.49]; P=0.117
Social support				
Continuous				
Score (standardized)	0.87 [0.73, 1.03]; P=0.111	-1.60 [-3.60, 0.40; P=0.116	0.96 [0.85, 1.10]; P=0.592	-0.39 [-1.87, 1.08]; P=0.601
Categorical				
High	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
Moderate	1.38 [1.00, 1.91]; p=0.052	3.22 [0.61, 5.83]; P=0.016	1.25 [0.93, 1.67]; p=0.140	2.18 [-0.37, 4.73]; P=0.094
Low	1.58 [1.01, 2.47]; P=0.044	4.96 [0.84, 9.08]; P=0.018	1.24 [0.87, 1.77]; P=0.230	2.08 [-0.96, 5.12]; P=0.180

HR= Hazard risk ratios from Cox model (multiplicative effect)
HD= Hazard rate differences from Aalen's model presented as per 10,000 individuals per year (additive effect)
[a]Adjusted for age, sex, race/ethnicity, parental education, and parental history of chronic condition, and social support or ACEs.

[b]Adjusted for age, race/ethnicity, parental education, parental history of chronic condition, BMI class, physical activity, smoking, alcohol use, depressive symptoms, and social support or ACEs.

On both additive and multiplicative models, we observed significant antagonistic interaction between continuous ACEs and continuous social support in relation to premature mortality, indicating that social support buffered the increased risk of premature mortality associated with ACEs. Categorical interaction analyses revealed that compared to those without ACEs exposure and high level of perceived social support, those reporting ≥1 ACEs and low level of social support had over twice greater hazards of premature mortality, corresponding to about 9.4 additional deaths per 10,000 individuals per year, after adjusting for baseline socio-demographic variables (aHR: 2.03 [95% CI: 1.10, 3.75]; aHD: 9.40 [95% CI: 1.89, 16.90]). Mortality hazard was also higher among those without ACEs and moderate social support, compared to those without ACEs and high social support. (Table 3.5)

Table 3.5 Joint effect estimates hazards of premature mortality associated with ACEs and social support

ACEs	Social support	Without adjustment for bio-behavioral factors[a]		With adjustment for bio-behavioral factors[b]	
		HR [95% CI]; P	HD [95% CI]; P	HR [95% CI]; P	HD [95% CI]; P
Continuous					
Standardized ACEs score	Standardized social support score	0.85 [0.83, 0.87]; P<0.001	-2.86 [-3.44, -2.28]; P<0.001	0.84 [0.82, 0.86]; P<0.001	-2.89 [-3.61, -2.17]; P<0.001
Categorical					
0 ACEs	High	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
	Moderate	1.41 [1.03, 1.94]; P=0.034	3.65 [1.43, 5.86]; P=0.001	1.27 [0.95, 1.70]; P=0.105	2.70 [0.82, 4.57]; P=0.005
	Low	0.86 [0.46, 1.60]; P=0.639	-1.11 [-6.20, 4.00]; P=0.671	0.65 [0.40, 1.06]; P=0.085	-4.05 [-7.97, -0.13]; P= 0.042

≥1 ACEs	High	0.84 [0.45, 1.55]; P=0.566	-1.94 [-7.44, 3.56]; P=0.489	0.76 [0.41, 1.43]; P=0.398	-2.80 [-8.48, 2.88]; P=0.333
	Moderate	1.23 [0.82, 1.82]; P=0.313	1.76 [-1.74, 5.27]; P=0.326	1.01 [0.72, 1.41]; P=0.970	-0.35 [-3.23, 2.53]; P=0.810
	Low	2.03 [1.10, 3.75]; P=0.023	9.40 [1.89, 16.90]; P=0.014	1.47 [0.89, 2.42]; P=0.133	5.50 [-0.44, 11.44] P=0.069

HR= Hazard Risk Ratios from Cox model (additive effect)
HD= Hazard rate differences presented as per 10,000 individuals per year (multiplicative effect)
aAdjusted for age, sex, race/ethnicity, parental education, and parental history of chronic condition.
bAdjusted for age, race/ethnicity, parental education, parental history of chronic condition, BMI class, physical activity, smoking, alcohol use, and depressive symptoms

Sex-stratified analyses revealed that among males, those with ≥1 ACEs and poor social support were associated with twice the mortality hazard (aHR: 2.05, 95% CI: 1.21, 3.50) compared to those without ACEs and high social support. That aHR corresponded to 11.4 additional deaths per 10,000 individuals per year (aHD: 11.40, 95% CI: 3.99, 18.81). Among females, the relative hazard for the comparison was 1.9 (aHR: 1.92, 95% CI: 0.84, 4.40), representing 7.6 additional deaths per 10,000 individuals per year (aHD: 7.61, 95% CI: -1.76, 17.00) (Appendix Table B.3 & B.4). Excluding participants who died within two years or five years of baseline assessment did not significantly change the findings, nor did the inclusion of participants aged 18 years or above at baseline (Appendix Table B.5 & B.6).

Discussion

In this cohort analysis of a nationally representative sample of adolescents in the US, we found that ACEs increased the risk of premature mortality prior to age 43 while social support reduced the risk of premature mortality. We observed significant effect modification by social support. While in the low social support group, exposure to ACEs was associated with significantly greater risk of mortality, in the moderate and high social support group, exposure to ACEs was associated with lower, non-significant mortality hazards, especially exposure to one ACEs

compared to no ACEs. These findings potentially lend support to the differential susceptibility hypothesis[149] suggesting that low level of stress may confer protection against mortality in the presence of high social support but confer risk in the presence of low social support.

To our knowledge, this is the first study that examined the direct and joint associations of prospectively collected ACEs and childhood social support with the risk of premature mortality through young adulthood from a US representative sample. Our findings suggest that exposure to family and community stressors during sensitive periods of childhood without a positive, secure, and nurturing relationships and support systems can activate body's immune, neuroendocrine, and metabolic pathways, thus acting as causal precursors to unhealthful behaviors, increased morbidity, and early mortality. [150,151] Early experiences, both positive and negative, may become "hardwired" into an individual's biology. [12,124,152,153] Past studies have shown the moderating and mediating role of social support in the relationship between ACEs and allostatic load among adult participants. [38,154] Similarly, perceived social support may also bolster psychological and socio-emotional resilience to negative emotional stimuli, in turn, hindering the psychological pathways from ACEs to mortality.

Our findings emphasize that critical attention is needed to strengthen ACEs prevention efforts along with the promotion of policy and programs that promote social support among US adolescents. Our joint effects findings particularly suggest that enhancing perceived social support among those experiencing or with a history of ACEs may have added benefit in reducing the burden of mortality among young adults. Prevention of ACEs and subsequent early mortality can occur through enacting or strengthening policy, ensuring needed supports are in place, and adopting early screening processes. Few policy-based research show the positive impact of safety net policies that support families reduce ACEs or related outcomes. [155,156]

Policy and public health efforts to prevent ACEs must be complemented by policies to enhance social support. Population-level interventions focusing on systems approach that target families and educational settings to improve the availability, accessibility, and quality of perceived social

support among vulnerable school-aged youths is critical to reduce premature deaths. Moreover, finding ways to strengthen existing safety-net policies that provide economic support to low-income families may help, as these groups are more likely to experience ACEs and have poor social support systems. [156,157] The Centers for Disease Control and Prevention (CDC)'s framework focuses on ensuring children have lived and felt experiences of having "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. [158,159] Moreover, training clinicians and health professionals to identify toxic stress, ACEs and their deleterious health consequences may serve better at the primary care level. [160] Existing ACEs screening programs may benefit by incorporating social support tools and resources to identify or refer at-risk children.

This study has several strengths. It fills several conceptual and methodological gaps in the current epidemiological literature investigating the link between ACEs, social support, and mortality. Most studies did not have a clear temporal sequence between exposure and outcome because they retrospectively assessed ACEs exposure among participants who were well into their adulthood and among which morbidity and mortality rates are relatively high. And even when ACEs information was prospectively collected, there may be a risk of misclassification bias resulting from the use of proxy responses from a parent/caregiver or teacher who may not have complete or accurate information about the child's experiences. Similarly, the study sample in most of the prior studies was non-representative, limiting generalizability to the target population. This study addresses the concern of temporality and uses adolescents-reported data from a representative US sample. Further, we provide relative and absolute risks of mortality while controlling for the important confounders (socio-demographic and bio-behavioral) of the relationship between ACEs, social support, and mortality.

Despite the advantages of the nationally representative prospective cohort with long-term follow-up, our study did not capture some of the most severe forms of ACEs such as sexual

abuse and domestic violence, and aspect of structural support. We only examined a limited number and types of stressors, however, the prevalence of cumulative ACEs in our study is comparable to prior studies. [161,162] Due to limited number of ACEs information available at baseline, we could not examine the interrelationships among different types of childhood ACEs and/or social support, and how they may cluster to affect premature mortality. Further, confounding from unmeasured contextual factors such as neighborhood socioeconomic disadvantage and structural racism is possible. Statistical power was somewhat limited for sex-stratified analyses owing to relatively few deaths during follow-up due to a young cohort.

Conclusions

We show the health-deteriorating effect of high ACEs independent of perceived social support, and health-promoting effect of high social support independent of ACEs. Poor social support exacerbated the risk of all-cause mortality associated with ACEs. Interventions that target to increase perceived social support among those with ACEs may have the greatest public health benefit in reducing premature deaths among young adults in the US. Additionally, to strengthen existing ACEs prevention efforts, the clinicians and educators need high quality ACEs prevention and mitigation education to effectively be supportive.

CHAPTER IV: NATIONAL AND STATE-LEVEL PREVALENCE OF ADVERSE CHILDHOOD EXPERIENCES AND POSITIVE CHILDHOOD EXPERIENCES IN THE US: NATIONAL SURVEY OF CHILDREN'S HEALTH 2016-2020

Thapa, K., Shen, Y., Vall, E.A., Cordero, J., & Rajbhandari-Thapa, J. To be Submitted to the Journal of Child and Family Studies.

Abstract

Substantial evidence demonstrates that positive protective factors (PPFs) mitigate the negative physical and psychological outcomes resulting from the toxic stress and trauma associated with adverse childhood experiences (ACEs). PPFs also have independent, dose-response effect on child health and flourishing even in the absence of ACEs. Understanding the prevalence of PPFs across levels of ACEs among children and adolescents in the United States (US) can inform public health programs seeking to promote PPFs and prevent and mitigate impacts of ACEs. Using the population-based National Survey of Children's Health (NSCH) data from 2016 to 2020, we estimated the national and across-state prevalence of ACEs, PPFs and PFFs across ACEs among US children aged 6-17 years. Nine ACEs (parental divorce, parental death, economic hardship, household incarceration, household substance abuse, household mental illness, domestic violence witness, neighborhood violence witness, and racial/ethnic discrimination) and seven PPFs (having an adult mentor, family resilience, participating in after-school activities, parent-child communication, safe neighborhood, supportive neighborhood, community volunteering) were included. The weighted national prevalence of >5 PPFs, >2 ACEs, and >5 PPFs among those with >2 ACEs were 37.6% [95% CI: 37.0%, 38.2%], 12.7% [95% CI: 12.2%, 13.1%], and 21.0% [95% CI: 19.4%, 22.6%] respectively. Across-state prevalence of ACEs, PPFs, and PFFs across ACEs varied considerably, with prevalence of >5 PPFs among children with >2 ACEs ranging from 12.3% [95% CI: 7.1%, 17.5%; p=0.001] in Nevada to 29.4% [95% CI: 20.5%, 38.3%; p=0.063] in Virginia. Joint assessment of ACEs and PPFs may inform public health interventions to better promote positive child wellbeing.

Introduction

Experiences during early months and years of human life shape brain development, and trigger physiological, psychological, and behavioral changes.[13,163,164] Decades of epigenetic, neurobiological, and epidemiological research on adversity and chronic stress lend support to

the notion that early life experiences become biologically embedded to affect developmental and health outcomes across the life course.[150,153] Adverse childhood experiences (ACEs) refer to potentially traumatic experiences that occur during childhood, particularly below 18 years of age, and within a family and community environment and that can result in toxic stress.[158] Specifically, ACEs can lead to physical or emotional trauma and, if not mitigated, to toxic stress, by disrupting the safety, stability and nurturance provided by caregivers and in other relationships and the community environment, which shape a child's healthy development and thriving.

The landmark ACEs study conducted at Kaiser Permanente in 1995-96 asked adult respondents about their history of adversity (abuse by parent/caregiver and dysfunction in the household) and found a dose-response relationship between number of ACEs and adult health risk behaviors such as smoking, alcoholism, and drug abuse and illnesses like depression, heart diseases and cancer.[19] These findings have been consistently replicated across large numbers of studies. The cross-cutting mechanism of effect is the experience of toxic stress that extends beyond what is tolerable and how this accumulates over time to worsen health outcomes through neural, physiologic, behavioral, and genetic mechanisms.[38,93] The assessment of ACEs has evolved over the past decades, and multiple studies in the last decade have studied ACEs among children and youth in the United States using the population-based National Survey of Children's Health (NSCH).[165,166] The ACEs measure in the NSCH included experiences such as experiences of discrimination due to race/ethnicity and witnessing or experiencing community violence.[167] Others also include experiences like bullying and living in foster care as potentially traumatic exposures, however, the strongest evidence is that linking family and household ACEs with future health problems pointing to the protective effect of nurturing relationships in daily life.[145,168,169] More recent studies have led to Positive Childhood Experiences (PCEs) measures that also demonstrate a similar dose-response relationship with adult health outcomes as ACEs.[170] Positive protective factors (PPFs) that can result in PCEs, such as family

resilience and positive parent-child communication, have been shown to mitigate the impacts of ACEs for children and youth.[171]

Scientific research continues to demonstrate the negative effects of ACEs such as physical or emotional abuse and neglect, sexual abuse, family conflict, and maternal psychopathology on a child's healthy development including academic achievement and their wellbeing into adulthood.[163,165,172] Large economic costs are associated with ACEs, pointing to economic benefits that could be reaped from preventing or reducing ACEs.[35,137,173,174] While the deleterious impact of ACEs is increasingly understood, the independent importance and protective impact of positive childhood experiences (PCEs) has gained greater attention in recent years partly due to the emergence of studies documenting impact on a population level.[123,170,175] Such studies and those on more targeted samples seek to understand the wide variation in health impacts among adults and children exposed to ACEs by identifying the resiliency and protective factors at play to lessen the deleterious effect of ACEs.

Positive protective factors (PPFs) are protective factors in the child's immediate environment (family, school, community) such as active engagement in learning and extracurricular activities and having a safe neighborhood that can foster or encompass PCEs (e.g., child experience of feeling safe and protected at home, positive caregiver-child interactions, feeling supported a sense of belonging with friends and in school, etc.).[171,176] The Centers for Disease Control and Prevention (CDC) has not advanced an emphasis on promoting PCEs with a focus on ensuring children have lived and felt experiences of having "safe, stable, and nurturing relationships and environments" during childhood.[158] Like ACEs research, PCEs and other PPFs that foster such relationships and environments have shown to independently and cumulatively predict positive health behaviors and outcomes, and have preventative as well as buffering effects against the toxic stress and negative health impacts of ACEs.[123,150,170,171,175-182] Examination of these modifiable factors generates insights into resilience among children and may provide policy targets for parenting or school interventions to improve socioemotional development.

ACEs and PCEs are often assessed retrospectively, such as among adults in the Behavioral Risk Factor Surveillance System (BRFSS) survey.[161,170,173,183] While retrospective evaluation provides valuable associative information between how adults recall the cumulative experiences from their childhood regarding ACEs, PCEs and health outcomes, it does not allow assessment of the current scenario among children and youths. Moreover, contemporaneous assessment of ACEs and PPFs during early life allows greater understanding of potentially harmful and beneficial experiences and stimuli in the developing child's ecological environment and thus inform population-based interventions to reduce burden of ACEs and promote positive lifelong health and development. Past studies using data from the National Survey of Children's Health used single or two-year data to examine disparities in exposure to ACEs and PPFs.[162,165,170,171,184-187] While some of these studies examined across state prevalence and variations, more years of NSCH data are required to examine prevalence of PPFs by ACEs more carefully among children and adolescents at the state level. For example, several studies have demonstrated the buffering effect of PPFs showing positive associations of PPFs with child flourishing and school engagement[171], adult mental and relational health[170], adult cardiovascular health[123], and adult family health[188] among US populations. Therefore, it is more informative to simultaneously examine ACEs and PPFs, with some across-state reporting on ACEs and/or PPFs separately. However, due to sample size limitations, to estimate and compare state-level prevalence of ACEs and PFFs subgroups (e.g., high ACEs, high PFFs or low ACEs, low PFFs) it is essential to combine more years of data. In this study, using data from a nationally representative survey, we assessed five-year estimates of the (i) prevalence of ACEs and PPFs among US children, and (ii) prevalence of PPFs across ACEs at the national and the state level.

Methods

Data

Data from a 2016-2020 combined National Survey of Children's Health (NSCH) (Child and Adolescent Health Measurement Initiative, 2021) were used (Appendix Table C.1). The NSCH is a mail- and web-based survey of civilian, non-institutionalized children aged 0-17 years led by the Maternal and Child Health Bureau of the Health Resources and Services Administration (MCHB HRSA). The NSCH collects data on health and well-being of children and adolescents from all 50 states and the District of Columbia (DC), including family and neighborhood, school and community experiences and characteristics, ACEs and PPFs. The NSCH employs a complex sampling design and data is weighted to represent children nationally and within each state. The NSCH randomly selects one child from households with at least one child between the ages of 0–17. The survey is completed by a parent/adult caregiver with knowledge of the child's health and health care. Additional information regarding the survey and sample methodology can be found elsewhere (www.childhealthdata.org).

Study sample and missing values

This study focused on children aged 6-17 years because data on four of seven PPFs assessed in this study was not collected for children aged 0-5 years. This resulted in a total sample size of 125,005. In addition, children with missing data on any ACEs or PPFs questions were set to missing during variable construction and analysis (n=12,827). A total of 112,178 6-17-year-old participants who had complete set of responses to ACEs and PPFs measures were represented in the final analyses conducted. The non-response to any ACEs as well as PPFs questions was significantly greater for non-Hispanic Black respondents (~11%) compared to other race/ethnicity groups (~7%), and for low-income groups (~9%) compared to higher income groups (~6%) (Appendix Table C.2).

Key Measures

Measures of Adverse Childhood Experiences (ACEs)

The NSCH collects information on nine types of ACEs. This includes the five items that make up the conventional household dysfunction domain of the ACEs from the original ACE study[19] – household mental illness, household substance abuse, household incarceration, witnessing domestic violence, and parental divorce. The four additional items include parental death, economic hardship, experience of discrimination due to child's race/ethnicity, and experiencing or witnessing neighborhood violence. These additional items were developed by a Technical Expert Panel based on a review of evidence-based life course stressors on children's lives.[167] In our analytic sample, parental/guardian divorce or separation (29%) and economic hardship (19%) were the most prevalent types of ACEs among children and adolescents (Appendix Figure C.1). All ACEs items except economic hardship have a 'Yes/No' dichotomous response option, and the wording of questions were consistent across the study years except for the economic hardship item, where the response options and wording were changed starting in 2018 which resulted in lower number of children being identified as experiencing economic hardship and, subsequently, lower overall ACEs prevalence (Table 4.1 & Appendix Table C.3). Specifically, item and response option changes for assessing economic hardship resulted in significant decrease in the number of respondents reporting 'somewhat often' and 'very often' experiencing economic hardship (12.3% in 2018-20 versus 19.8% in 2016-17). However, the impact of this change is uniform across child and geographic subgroups evaluated allowing for comparison in findings across these groups. Further, the national prevalence of PFFs by ACEs among those exposed to at least one ACE remained similar after the removal of economic hardship item (Appendix Table C.4). For the ACEs measure used in the study, each child was categorized based on their cumulative ACEs score using 0, 1-2, and >2 categories.

Measures of Positive Protective Factors (PPFs)

The NSCH PPFs items are distributed across relevant sections of the survey. Based on prior studies examining PPFs using NSCH data[185,189], the following seven items were included to assess exposure to PPFs for school age children (aged 6-17): 1) the child volunteers in the community; 2) child participates in after-school activities; 3) child has an adult mentor, other than their parent/caregiver; 4) caregiver reports positive communication with the child whereby they share about ideas and things that really matter; (5) child's family demonstrates resilience;6) child lives in a supportive neighborhood; and 7) child lives in a safe neighborhood (Table 4.1). These items also align with four categories of protective factors set forth in the HOPE (Health Outcomes from Positive Experiences) framework – a guiding framework that synthesizes a range of PPFs and places them into four categories: (1) being in nurturing, supportive relationships; (2) living, developing, playing, and learning in safe, stable, protective, and equitable environments; (3) having opportunities for constructive social engagement and to develop a sense of connectedness; and (4) learning social and emotional competencies.[181] There were no substantial changes in the wording of PPF items across the study years. Having a mentor for advice or guidance (89%), family resilience (81%), and after school activities (79%) were the most common types of PPFs (Appendix Figure C.1). All PPFs items were categorized as binary (yes/no) and children were categorized based on the cumulative scoring using ≤2, 3-5, and 6-7 categories.

Table 4.1 ACEs and PPFs measures used in the study

Item	**NSCH question**	**Original response options**	**Recoded options**
ACEs items			
Parental divorce	To the best of your knowledge, has this child EVER experienced any of the following? *Parent or guardian divorced or separated*	○ Yes ○ No	○ Yes ○ No

Parental death	To the best of your knowledge, has this child EVER experienced any of the following? *Parent or guardian died*	○ Yes ○ No	○ Yes ○ No
Household incarceration	To the best of your knowledge, has this child EVER experienced any of the following? *Parent or guardian served time in jail*	○ Yes ○ No	○ Yes ○ No
Witnessing domestic violence	To the best of your knowledge, has this child EVER experienced any of the following? *Saw or heard parents or adults slap, hit, kick, punch one another in the home*	○ Yes ○ No	○ Yes ○ No
Witnessing neighborhood violence	To the best of your knowledge, has this child EVER experienced any of the following? *Was a victim of violence or witnessed violence in their neighborhood*	○ Yes ○ No	○ Yes ○ No
Household mental illness	To the best of your knowledge, has this child EVER experienced any of the following? *Lived with anyone who was mentally ill, suicidal, or severely depressed*	○ Yes ○ No	○ Yes ○ No
Household substance abuse	To the best of your knowledge, has this child EVER experienced any of the following? *Lived with anyone who had a problem with alcohol or drugs*	○ Yes ○ No	○ Yes ○ No
Racial mistreatment	To the best of your knowledge, has this child EVER experienced any of the following? *Treated or judged unfairly because of their race or ethnic group*	○ Yes ○ No	○ Yes ○ No
Economic hardship	SINCE THIS CHILD WAS BORN, how often has it been very hard to cover the basics, like food or housing, on your family's income?*	○ Never ○ Rarely ○ Somewhat often ○ Very often	○ Never/Rarely ○ Somewhat often/Very often
PPFs items			

Child volunteers in the community, school, or place of worship	*During the past 12 months, did this child participate in any type of community service or volunteer work at school, place of worship[#], or in the community?*	○ Yes ○ No	○ Yes ○ No
Child participates in after school activities	*During the past 12 months, did this child participate in a sports team or did they[^] take sports lessons after school or on weekends?* *Any clubs or organizations after school or on weekends?* *Any other organized activities or lessons, such as music, dance, language, or arts?*	○ Yes ○ No	○ Yes ○ No
Child has an adult mentor other than parent/caregiver	*Other than you or other adults in your home, is there at least 1 adult in this child's school, neighborhood, or community who knows this child well and who they[^] can rely on for advice or guidance?*	○ Yes ○ No	○ Yes ○ No
Child lives in a resilient family	*When your family faces problems, how often are you likely to do each of the following?* *1) talk together about what to do,* *2) work together to solve our problems,* *3) know we have strengths to draw on, and* *4) stay hopeful even in difficult times.*	○ None of the time ○ Some of the time ○ Most of the time ○ All the time	○ None of the time/Some of the time ○ Most of the time/All the time (to all 4 sub-items)
Child lives in a connected family	*How well can you and this child share ideas or talk about things that really matter?*	○ Very well ○ Somewhat well ○ Not very well ○ Not very well at all	○ Very well ○ Somewhat well/Not very well/Not very well at all
Child lives in a supportive neighborhood	*To what extent do you agree with these statements about your*	○ Definitely agree ○ Somewhat agree	○ Definitely agree to at least one sub-item and

	neighborhood or community… *1) people in this neighborhood help each other out,* *2) we watch out for each other's children in this neighborhood, and* *3) when we encounter difficulties, we know where to go for help in our community.*	○ Somewhat disagree ○ Definitely disagree	Definitely agree/Somewhat agree on other two sub-items ○ Other combinations
Child lives in a safe neighborhood	*To what extent do you agree with this statement about your neighborhood or community: the child is safe in our neighborhood.*	○ Definitely agree ○ Somewhat agree ○ Somewhat disagree ○ Definitely disagree	○ Definitely agree ○ Somewhat agree/Somewhat disagree/Definitely disagree

*The wording of the item prior to NSCH 2018 was as follows 'SINCE THIS CHILD WAS BORN, how often has it been very hard to get by on your family's income – hard to cover the basics like food or housing?'
#Changed in 2017 from 'church' to 'place of worship'
^Changed in 2020 from 'he or she' to 'they'

Socio-demographic characteristics

Four socio-demographic characteristics were used in this study: (1) age group (6-11 years, and 12-17 years); (2) sex of the child (Male, Female); (3) race/ethnicity of the child (Non-Hispanic White, Non-Hispanic Black, Hispanic, Non-Hispanic Asian, and Non-Hispanic Other (American Indian or Alaska Native, Native Hawaiian or Other Pacific Islander, and multi-racial) and (4) the poverty level of the child's household as determined by family income and family size and divided into four groups based on federal poverty level (FPL) 0-199% FPL, 200-299% FPL, 300-399% FPL, and 400% FPL or greater).

Analytic Methods

National and state (including DC) level prevalence estimates were calculated for each ACEs and PPFs item. Two categorical ACEs and PPFs cumulative count variables were created indicating whether a child had 0, 1-2 or 3-9 ACEs and/or 0-2, 3-5 or 6-7 PPFs. Bivariate

analyses were used to calculate the prevalence of cumulative ACEs and PPFs for each socio-demographic subgroup nationally and for each state. The statistical significance of differences across socio-demographic subgroups was assessed using chi-square tests of differences using a 95% confidence level. Next, we stratified the ACEs cumulative score categorical variable with the PPFs cumulative score categorical variable for the nation and each state/DC and similarly evaluated the presence of significant differences across subgroups using chi-square tests of significance. State/DC differences from the nation in prevalence of ACEs, PPFs, and PPFs for each ACEs cumulative score category were evaluated using nested t-test and a 95% confidence level. All analyses used weighting, stratification, and clustering to account for complex survey design and multiyear analysis. Analyses were conducted using R v4.0.3 software using 'srvyr' package.

Results

The mean age of study analytic sample (age 6-17) was 11.5 years (SD=3.4). About 50% were 6-11 years old; 49% females; 52% non-Hispanic White, 25% Hispanic, 13% non-Hispanic Black, and less than 5% were non-Hispanic Asian; about 19% had household income level between 0-99% FPL, 22% between 100-199% FPL, and 28% between 200-399% FPL. The national prevalence of 0, 1-2 and 3-9 ACEs were 52.3% [95% CI: 51.7%, 53.0%], 35.0% [95% CI: 34.4%, 35.6%] and 12.7% [95% CI: 12.2%, 13.1%] respectively, with significant differences across age group, race/ethnicity, and poverty level. Similarly, the national prevalence of 0-2, 3-5 and 6-7 PPFs were 9.5% [95% CI: 9.0%, 10.0%], 52.9% [95% CI: 52.2%, 53.5%] and 37.6% [95% CI: 37.0%, 38.2%] respectively, with significant differences across age group, sex, race/ethnicity, and poverty level (Table 4.2).

Across ACEs categories, the prevalence of children with 6-7 PPFs ranged from 21% [95% CI: 19.4%, 22.6%] among those with 3-9 ACEs to 46.8% [95% CI: 45.9%, 47.7%] among those with no ACEs. The prevalence of PPFs across ACEs levels varied by subgroups. Overall, the

prevalence of children with 6-7 PPFs was significantly greater among 12–17-year-olds, females, Non-Hispanic Whites, and high-income households across all levels of ACEs (Appendix Table C.5).

Table 4.2 Prevalence of ACEs [95% CI] and PPFs [95% CI] by socio-demographic characteristics, NSCH 2016-2020

	Respondent distribution (%)[a]	ACEs (%)			p-value[c]	PPFs (%)			p-value[c]
		0 ACEs [95% CI][b]	1-2 ACEs [95% CI][b]	3-9 ACEs [95% CI][b]		0-2 PCEs [95% CI][b]	3-5 PCEs [95% CI][b]	6-7 PCEs [95% CI][b]	
Overall	100%	52.3 [51.7, 53.0]	35.0 [34.4, 35.6]	12.7 [12.2, 13.1]	-	9.5 [9.0, 10.0]	52.9 [52.2, 53.5]	37.6 [37.0, 38.2]	-
Age group									
6-11 years	49.7%	56.8 [55.8, 57.8]	32.6 [31.7, 33.6]	10.6 [10.0, 11.2]	<0.001	9.4 [8.7, 10.1]	54.6 [53.7, 55.6]	36.0 [35.1, 36.9]	<0.001
12-17 years	50.3%	48.0 [47.0, 48.9]	37.3 [36.4, 38.2]	14.7 [14.0, 15.4]		9.6 [8.9, 10.3]	51.1 [50.2, 52.1]	39.3 [38.4, 40.1]	
Gender									
Female	48.9%	52.2 [51.2, 53.2]	35.2 [34.3, 36.2]	12.6 [11.9, 13.3]	0.8	8.5 [7.8, 9.1]	52.2 [51.2, 53.2]	39.3 [38.4, 40.2]	<0.001
Male	51.1%	52.5 [51.6, 53.4]	34.8 [33.9, 35.7]	12.7 [12.1, 13.3]		10.5 [9.8, 11.1]	53.5 [52.6, 54.4]	36.0 [35.2, 36.8]	
Race/ethnicity									
Non-Hispanic White	51.7%	56.7 [56.0, 57.3]	32.0 [31.4, 32.7]	11.3 [10.8, 11.7]	<0.001	5.7 [5.4, 6.1]	47.7 [47.1, 48.4]	46.5 [45.9, 47.2]	<0.001
Hispanic	25.1%	49.6 [47.7, 51.5]	37.8 [35.9, 39.6]	12.6 [11.4, 13.8]		14.6 [13.2, 16.0]	59.7 [57.8, 61.5]	25.7 [24.1, 27.3]	
Non-Hispanic Black	12.9%	36.5 [34.5, 38.5]	45.4 [43.4, 47.4]	18.1 [16.5, 19.7]		12.9 [11.4, 14.5]	59.0 [57.0, 61.0]	28.1 [26.3, 29.8]	
Non-Hispanic Asian	4.4%	73.9 [71.4, 76.4]	23.0 [20.6, 25.3]	3.1 [1.9, 4.4]		14.5 [12.2, 16.7]	51.6 [48.8, 54.4]	33.9 [31.4, 36.4]	
Non-Hispanic Other	5.9%	44.5 [42.3, 46.7]	35.5 [33.4, 37.6]	20.0 [18.0, 22.0]		9.5 [8.2, 10.9]	56.7 [54.5, 58.9]	33.8 [31.7, 35.9]	
Household income level									

0-99% FPL	18.5%	35.2 [33.3, 37.1]	43.4 [41.5, 45.3]	21.4 [19.9, 22.8]	<0.001	17.5 [16.0, 19.1]	59.5 [57.6, 61.4]	23.0 [21.4, 24.5]	<0.001
100-199% FPL	21.7%	41.7 [40.0, 43.4]	40.1 [38.5, 41.7]	18.2 [17.0, 19.4]		12.9 [11.6, 14.1]	60.0 [58.4, 61.6]	27.2 [25.8, 28.5]	
200-399% FPL	28.3%	52.1 [50.9, 53.3]	36.6 [35.5, 37.8]	11.3 [10.5, 12.0]		8.2 [7.4, 9.0]	53.0 [51.8, 54.1]	38.8 [37.7, 39.9]	
400% FPL or greater	31.6%	69.9 [69.0, 70.7]	25.1 [24.3, 25.9]	5.0 [4.6, 5.4]		3.6 [3.3, 4.0]	44.1 [43.2, 45.0]	52.3 [51.4, 53.2]	

Abbreviation: ACEs: Adverse Childhood Experiences; PPFs: Positive Protective Factors; CI: Confidence Interval; NSCH: National Survey of Children's Health; FPL: Federal Poverty Level [a]The denominator is the total weighted number of respondents. [b]The denominator is the total weighted number of respondents for the respective category of age, sex, race/ethnicity, or household poverty level. [c]Chi-squared test with Rao & Scott's second-order correction

The mean number of ACEs and PPFs varied across states (Appendix Figure C.2). In several states, the prevalence of cumulative ACEs and PPFs varied significantly from the national prevalence. For example, the prevalence of 3-9 ACEs ranged from 7.2% [95% CI: 5.6%, 8.9%, p<0.001] to 20.2% [95% CI: 17.5%, 22.8%, p<0.001] while the prevalence of 6-7 PPFs ranged from 26.9% [95% CI: 24.3%, 29.6%, p<0.001] to 50.8% [95% CI: 47.8%, 53.7%, p<0.001] (Table 4.3).

Table 4.3 Across-state prevalence [95% CI] of ACEs and PPFs among school-aged children in the US, NSCH 2016-2020

	Total N, unweighted	ACEs score (range: 0-9)			PPFs score (range: 0-7)		
		Weighted % (95% CI)			Weighted % (95% CI)		
		0	1-2	3-9	0-2	3-5	6-7
United States	112,178	52.3 [51.7, 53.0]	35.0 [34.4, 35.6]	12.7 [12.2, 13.1]	9.5 [9.0, 10.0]	52.9 [52.2, 53.5]	37.6 [37.0, 38.2]
State							
Alabama	2,115	47.0 [44.0, 50.0][#]	41.2 [38.1, 44.2][*]	11.8 [9.7, 13.8]	7.8 [5.7, 9.8]	52.6 [49.6, 55.7]	39.5 [36.6, 42.4][*]
Alaska	1,910	49.3 [46.1, 52.5]	32.6 [29.4, 35.7]	18.0 [15.5, 20.4][*]	10.5 [8.3, 12.7]	54.0 [50.8, 57.2]	35.5 [32.5, 38.5]

Arizona	2,151	49.9 [46.8, 53.1]	35.3 [32.3, 38.3]	14.7 [12.4, 17.0]	11.0 [8.9, 13.1]	55.3 [52.2, 58.4]	33.6 [30.7, 36.4]#
Arkansas	2,103	41.4 [38.4, 44.5]#	38.3 [35.1, 41.5]*	20.2 [17.5, 22.8]*	9.3 [7.3, 11.3]	52.9 [49.7, 56.1]	37.8 [34.8, 40.8]
California	2,101	59.5 [56.3, 62.6]*	29.9 [27.0, 32.8]#	10.5 [8.5, 12.6]#	13.1 [10.8, 15.5]*	56.6 [53.5, 59.8]*	30.2 [27.4, 33.0]#
Colorado	2,799	52.8 [49.8, 55.6]	33.0 [30.2, 35.7]	14.2 [12.0, 16.4]	7.4 [5.6, 9.2]#	53.0 [50.1, 55.8]	39.5 [36.8, 42.2]*
Connecticut	2,313	57.1 [54.1, 60.1]*	32.3 [29.4, 35.2]	10.5 [8.4, 12.5]#	8.4 [6.5, 10.3]	49.4 [46.4, 52.3]	42.2 [39.3, 45.0]*
Delaware	2,035	48.5 [45.4, 51.5]#	38.0 [34.9, 41.0]	13.5 [11.2, 15.8]	10.2 [8.2, 12.3]	54.2 [51.1, 57.2]	35.6 [32.7, 38.4]
District of Columbia	1,622	45.3 [41.4, 49.2]#	38.3 [34.4, 42.2]	16.3 [13.1, 19.6]*	8.5 [6.2, 10.7]	62.2 [58.4, 65.9]*	29.3 [25.9, 32.7]#
Florida	2,119	47.2 [44.1, 50.2]#	39.4 [36.4, 42.4]*	13.3 [11.1, 15.6]	9.3 [7.4, 11.2]	56.8 [53.8, 59.8]*	33.8 [31.1, 36.6]#
Georgia	2,130	50.4 [47.3, 53.4]	36.7 [33.7, 39.7]	12.8 [10.7, 15.0]	10.4 [8.2, 12.6]	54.3 [51.3, 57.3]	35.3 [32.5, 38.0]#
Hawaii	2,188	54.5 [51.5, 57.4]	35.0 [32.1, 37.9]	10.4 [8.6, 12.2]#	8.6 [7.0, 10.1]	57.2 [54.3, 60.1]*	34.2 [31.4, 37.0]#
Idaho	2,244	50.5 [47.6, 53.4]	33.9 [31.1, 36.6]	15.5 [13.3, 17.8]*	6.2 [4.7, 7.7]#	49.6 [46.7, 52.5]#	44.2 [41.4, 47.0]*
Illinois	2,180	56.2 [53.1, 59.3]*	32.9 [29.1, 35.8]	10.8 [8.8, 12.8]	7.2 [5.7, 8.8]#	55.5 [52.5, 58.4]	37.3 [34.4, 40.1]
Indiana	2,162	49.4 [46.5, 52.3]#	36.9 [34.1, 39.7]	13.6 [11.5, 15.7]	8.8 [6.8, 10.7]	51.9 [49.0, 54.8]	39.3 [36.5, 42.0]
Iowa	2,230	53.8 [51.0, 56.6]	32.9 [30.2, 35.5]	13.3 [11.1, 15.4]	6.7 [5.1, 8.2]#	46.9 [44.1, 49.8]#	46.4 [43.6, 49.1]*
Kansas	2,253	52.6 [49.6, 55.5]	33.4 [30.5, 36.3]	13.9 [11.7, 16.1]	6.8 [5.1, 8.5]#	50.0 [47.1, 52.9]	43.1 [40.3, 46.0]*
Kentucky	2,121	47.3 [44.4, 50.3]#	36.3 [33.4, 39.2]	16.3 [13.9, 18.6]*	6.8 [5.3, 8.2]#	52.7 [49.8, 55.6]	40.5 [37.7, 43.4]*
Louisiana	2,006	45.1	41.4	13.4	9.9	52.7	37.4

		[42.1, 48.1][#]	[38.3, 44.5][*]	[11.3, 15.5]	[7.9, 11.9]	[49.6, 55.7]	[34.5, 40.3]
Maine	2,114	46.9 [44.1, 49.6][#]	37.3 [34.6, 40.1]	15.7 [13.5, 17.9][*]	6.1 [4.6, 7.5][#]	50.7 [47.9, 53.5]	43.2 [40.5, 46.0][*]
Maryland	2,237	52.6 [49.6, 55.7]	36.5 [33.5, 39.5]	10.8 [8.7, 13.0]	9.1 [7.0, 11.2]	51.0 48.0, 54.0]	39.8 [37.0, 42.7][*]
Massachusetts	2,274	57.9 [54.9, 60.9][*]	33.4 [30.6, 36.3]	8.6 [6.7, 10.5][#]	7.0 [5.3, 8.6][#]	50.8 [47.9, 53.7]	42.2 [39.4, 45.0][*]
Michigan	2,238	51.1 [48.1, 54.1]	34.8 [31.8, 37.8]	14.0 [11.7, 16.3]	7.0 [5.3, 8.7][#]	53.1 [50.1, 56.1]	40.0 [37.1, 42.8][*]
Minnesota	2,267	58.7 [55.7, 61.7][*]	30.4 [27.6, 33.2][#]	10.8 [8.6, 12.9]	8.1 [6.1, 10.1]	45.7 [42.7, 48.6][#]	46.2 [43.3, 49.1][*]
Mississippi	2,016	42.4 [39.5, 45.4][#]	41.3 [38.4, 44.4][*]	16.1 [13.9, 18.4][*]	9.3 [7.3, 11.3]	55.1 [52.2, 58.1]	35.5 [32.8, 38.2][#]
Missouri	2,190	49.2 [46.3, 52.1][#]	35.3 [32.5, 38.2]	15.4 [13.1, 17.8][*]	8.3 [6.5, 10.1]	50.4 [47.5, 53.4][#]	41.2 [38.4, 44.0][*]
Montana	2,241	46.0 [43.1, 48.9][#]	34.4 [31.6, 37.3]	19.5 [17.0, 22.0][*]	5.9 [4.4, 7.3][#]	50.1 [47.2, 53.0][#]	44.0 [41.2, 46.9][*]
Nebraska	2,323	55.9 [52.9, 58.9][*]	31.7 [28.9, 34.6][#]	12.3 [10.2, 14.4]	6.5 [4.9, 8.0][#]	45.0 [42.0, 48.1][#]	48.5 [45.5, 51.5][*]
Nevada	1,996	45.9 [42.7, 49.1][#]	40.0 [36.8, 43.2][*]	14.1 [11.7, 16.4]	15.0 [12.4, 17.6][*]	58.0 [54.8, 61.2][*]	26.9 [24.3, 29.6][#]
New Hampshire	2,276	56.6 [53.9, 59.2][*]	34.2 [31.6, 36.8]	9.2 [7.6, 10.8][#]	6.5 [5.0, 7.9][#]	47.8 [45.1, 50.5][#]	45.7 [43.0, 48.3][*]
New Jersey	2,282	59.4 [56.3, 62.4][*]	33.3 [30.4, 36.3]	7.2 [5.6, 8.9][#]	9.2 [6.9, 11.4]	47.7 [44.7, 50.8][#]	43.1 [40.1, 46.0][*]
New Mexico	2,103	43.3 [40.1, 46.6][#]	38.3 [35.0, 41.6][*]	18.3 [15.8, 20.8][*]	11.3 [9.1, 13.5]	57.9 [54.7, 61.1][*]	30.7 [27.8, 33.6][#]
New York	2,083	54.8 [51.6, 58.1]	36.4 [33.2, 39.6]	8.7 [6.7, 10.7][#]	10.0 [7.7, 12.3]	56.1 [53.0, 59.3]	33.8 [30.9, 36.8][#]
North Carolina	2,115	52.3 [49.1, 55.6]	35.2 [32.0, 38.4]	12.4 [10.1, 14.7]	10.5 [8.2, 12.8]	49.8 [46.6, 53.0]	39.6 [36.6, 42.7]
North Dakota	2,143	57.2 [54.2, 60.2][*]	30.0 [27.2, 32.7][#]	12.8 [10.5, 15.0]	4.4 [3.3, 5.6][#]	45.9 [42.9, 48.9][#]	49.6 [46.7, 52.6][*]

Ohio	2,214	47.7 [44.8, 50.6][#]	36.5 [33.7, 39.4]	15.6 [13.3, 18.0][*]	9.2 [7.1, 11.2]	49.2 [46.2, 52.1][#]	41.6 [38.8, 44.5][*]
Oklahoma	2,050	44.7 [41.8, 47.7][#]	37.2 [34.2, 40.1]	18.0 [15.6, 20.4][*]	11.7 [9.5, 14.0][*]	53.7 [50.7, 56.7]	34.5 [31.8, 37.2][#]
Oregon	3,719	49.3 [46.4, 52.2][#]	35.0 [32.2, 37.8]	15.6 [13.3, 17.8][*]	9.1 [7.2, 10.9]	55.3 [52.4, 58.2][*]	35.6 [32.9, 38.3]
Pennsylvania	2,197	55.3 [52.3, 58.3][*]	32.8 30.0, 35.7]	11.8 [9.7, 13.8]	7.5 [5.7, 9.3]	50.0 [47.0, 53.0]	42.4 [39.5, 45.3][*]
Rhode Island	2,036	52.7 [49.6, 55.8]	34.9 [31.9, 38.0]	12.3 [10.2, 15.5]	8.9 [7.0, 10.8]	53.1 [50.0, 56.1]	38.0 [35.1, 40.9]
South Carolina	2,148	48.9 [45.9, 51.9][#]	36.9 [33.9, 39.9]	14.2 [11.9, 16.5]	7.9 [6.0, 9.7]	50.4 [47.4, 53.4]	41.7 [38.8, 44.6][*]
South Dakota	2,181	50.7 [47.8, 53.7]	33.2 [30.4, 36.0]	16.0 [13.6, 18.5][*]	5.3 [3.8, 6.8][#]	48.3 [45.3, 51.3][#]	46.4 [43.5, 49.3][*]
Tennessee	2,033	47.7 [44.7, 50.6][#]	36.7 [33.7, 39.6]	15.6 [13.2, 18.0][*]	9.6 [7.6, 11.5]	49.3 [46.3, 52.3]	41.1 [38.3, 44.0][*]
Texas	2,007	50.0 [46.7, 53.4]	36.6 [33.4, 39.9]	13.2 [11.1, 15.5]	11.9 [9.5, 14.3][*]	52.9 [49.6, 56.2]	35.2 [32.1, 38.2]
Utah	2,158	57.9 [55.0, 60.8][*]	30.9 [28.2, 33.6][#]	11.2 [9.2, 13.1]	5.6 [4.2, 7.1][#]	43.6 [40.6, 46.6][#]	50.8 [47.8, 53.7][*]
Vermont	2,313	51.1 [48.3, 54.0]	34.4 [31.7, 37.1]	14.4 [12.2, 16.6]	5.5 [3.9, 7.1][#]	48.9 [46.1, 51.8][#]	45.6 [42.8, 48.3][*]
Virginia	2,211	53.3 [50.3, 56.3]	35.5 [32.7, 38.4]	11.1 [9.1, 13.2]	6.1 [4.6, 7.6][#]	52.1 [49.2, 55.0]	41.8 [38.9, 44.6][*]
Washington	2,209	54.6 [51.5, 57.7]	33.6 [30.6, 36.6]	11.8 [9.8, 13.9]	8.0 [6.2, 10.0]	54.6 [51.5, 57.7]	37.3 [34.4, 40.3]
West Virginia	2,023	42.0 [39.2, 44.8][#]	39.3 [36.4, 42.2][*]	18.7 [16.0, 21.3][*]	8.4 [6.6, 10.2]	53.5 [50.6, 56.4]	38.1 [35.3, 40.8]
Wisconsin	2,982	52.4 [49.6, 55.2]	32.7 [30.1, 35.3]	14.9 [12.5, 17.2]	6.9 [5.2, 8.5][#]	50.6 [47.8, 53.3]	42.5 [39.9, 45.2][*]
Wyoming	2,017	48.4 [45.5, 51.4][#]	33.6 [30.8, 36.5]	17.9 [15.4, 20.3][*]	6.7 [5.0, 8.3][#]	47.9 [44.9, 50.9][#]	45.5 [42.5, 48.4][*]

[*]Prevalence significantly higher than national prevalence at 0.05 level of significance;
[#]Prevalence significantly lower than national prevalence at 0.05 level of significance

The prevalence of PPFs across ACEs categories also varied from the national prevalence in several states. For example, the prevalence of 6-7 PPFs among those with 3-9 ACEs ranged from 12.3% [95% CI: 7.1%, 17.5%, p<0.001] to 29.4% [95% CI: 20.5%, 38.3%, p=0.063] (Figure 4.1), while the prevalence of 6-7 PPFs among those with no ACEs ranged from 35.4% [95% CI: 31.7%, 39.2%, p<0.001] to 62.5% [95% CI: 58.6%, 66.3%, p<0.001] (Appendix Table C.6).

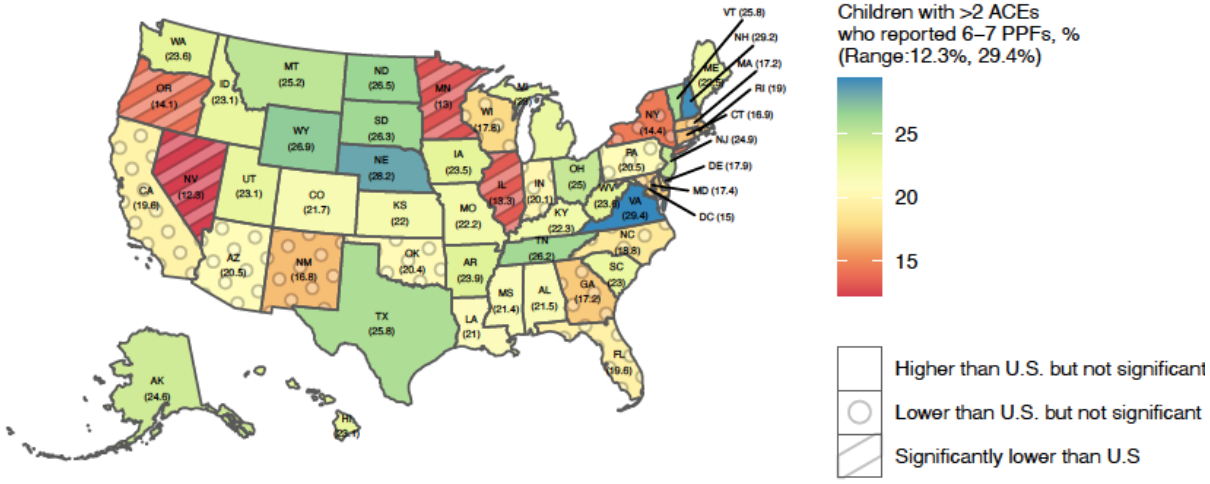


Figure 4.1 Percentage of school-aged children with >2 ACEs who reported >5 PPFs, NSCH 2016-2020

The prevalence of individual ACEs and individual PPFs varied across states (Appendix Table C.7, Appendix Table C.8).

Discussion

In this study, we found that the prevalence of ACEs, protective factors, and protective factors among children experiencing ACEs vary considerably across US states Our findings on ACEs prevalence and disparities are largely consistent with the past literature utilizing NSCH data, the majority of which report that more than four in ten children are exposed to at least one ACE, the

prevalence is higher among older age groups, ethnic minorities especially among Non-Hispanic Blacks, and low-income populations, and there is considerable variations in prevalence across states.[30,162,166,186,190,191] Our study adds further evidence on the prevalence of protective factors and its variations across socio-economic characteristics and across states. Prior studies have also reported disparities with regards to household educational level, sexual orientation, rurality status, non-immigration status, special healthcare needs status, family structure, and so on.[30,185,189,192,193] Family's access to economic opportunities and resources has consistently demonstrated a profound effect on a child's exposure to ACEs or PPFs. The complex interplay between socioeconomic status and ACEs has been studied[157,190], but how poverty affects children's exposure to PPFs both within and outside the context of ACEs needs further exploration. This is especially important since many children experiencing poverty do also experience many PPFs and factors contributing to this are important to understand to promote PPFs among children facing adversity. We also found that children with a higher number of ACEs also experience a lower number of PPFs in a dose-dependent and cumulative manner, suggesting the co-occurring nature of high ACEs and low PPFs or vice versa. Yet, many children with higher ACEs do experience higher levels of PPFs and understanding this is important to supporting children facing adversity. This calls for systems change approach to childhood development which identifies the crucial roles of the parents/caregivers, family relationships, schools, and the community in developing and maintaining positive life-course outcomes, as well as reducing the negative outcomes.

Our state-level analyses of prevalence of ACEs, PPFs, and PPFs across ACEs can guide state actions to prevent ACEs and promote trauma-informed care. The CDC's evidence-based resource tool focuses on family-centered approach to preventing ACEs such as strengthening economic supports for families, promoting social norms that protect against violence and adversity, teaching skills, and connecting youths to caring adults.[158,159] Several states have passed legislation and launched initiatives ranging from primary prevention of ACEs through

improved prevention and health promotion services and well child care visits in health care, public education campaigns, government assistance programs and parenting interventions focusing on building healthy families and relationships to screening and treatment for ACE and its immediate and long-term consequences such as psychotherapy and substance abuse treatment.[194] The PPFs explored in this study reflect the CDC's ACEs prevention approaches, suggesting that low prevalence of PPFs at the state level can be an indicator for greater need for state support to ACEs prevention and mitigation and promotion of PPFs. In this regard, standard indicators to track ACEs and PPFs at the state-level may be necessary to inform efforts to promote healthy child development and lower physical and mental health conditions associated with ACEs. Likewise, examining states with children with higher ACEs also experiencing higher PPFs is informative. The Improving Data Collection for Adverse Childhood Experiences Act authorizes the CDC to collect longitudinal data on ACEs in cooperation with the states to broaden our understanding of adverse and positive childhood experiences.

Recognizing the importance of protective factors during early life, some states have made efforts to include measures of positive experiences in their annual health surveys.[195] For instance, Tennessee's inclusion of a PCEs in their 2021 BRFSS[196], based on the PCEs measure used in the 2019 study using BRFSS data.[170] Massachusetts has similarly included PCEs and other PPF questions in their 2021 youth health survey.[159] Including measures of PCEs and other PPFs in addition to ACEs in routinely collected public health surveillance systems, such as the NSCH, BRFSS, and YRBS may help advance the scientific knowledge on children's exposure to safe, stable, and nurturing environments and communities.

This study utilizes the strength of a nationally representative survey of children's health to identify key vulnerabilities and opportunities in different aspects of the family and community environment essential for healthy child development. However, there are a few limitations to this study. First, the ACEs and PPFs are not definitive – their assessment is limited to the categories included in our study and therefore may not be generalizable to other types of ACEs or PPFs.

Due to lack of standardized metrics, caution should be made to compare findings across studies due to variations in measurement of ACEs and PPFs and their individual components. Second, PPFs differ from assessments of child flourishing and resilience in that the former focuses on protective factors across multiple levels of the child environment while the latter focuses on the positive health outcomes which have been shown to be associated with exposure to PPFs. Third, the responses to NSCH questions are parent/caregiver-reported, which may be subject to recall or other biases, including effects associated with a parent/caregivers' own childhood experiences.[195,197] As a result, the ACEs and PPFs exposures may be under- or overreported. Fourth, due to lack of validity of parent/caregiver report, the NSCH does not directly collect information on two conventional domains of ACEs – experience of abuse and experience of neglect, however questions about witnessing or experiencing violence in the home or community were found in cognitive testing of the NSCH ACEs items to identify children experiencing abuse as well.[167] Due to the co-occurring and clustering nature of ACEs, the cumulative prevalence of children evaluated here may not be greatly affected. Lastly, because participation in the NSCH is limited to those with an address, the sample also does not include transient or homeless children, children living within institutions or who are incarcerated who may have greater risk of ACEs exposure and lower levels of PPFs exposure.

Conclusions

ACEs continue to be prevalent among US children and adolescents, with significant variations across states. The prevalence of PPFs varied, with about 4 in 10 children overall and 1 in 5 children with more than 2 ACEs experiencing six or more PPFs nationally, along with state-level differences. Due to lifelong health impact of early life experiences, states should implement, strengthen, and evaluate programs that focus on preventing and reducing exposure to ACEs and increasing exposure to PPFs. National efforts to prevent ACEs must continue with specific efforts to promote state and national efforts to promote PPFs. The NSCH provides unique

opportunity to incorporate and track both ACEs and PPFs measures as children are increasingly exposed to new forms of stressors such as community violence and racism. Inclusion of such information should be considered in other national surveys, like the Medical Expenditures Panel Survey, the National Health Interview Survey as well standard content in the Behavioral Risk Factor Surveillance System and the Youth Risk Behavior Surveillance System, among others.[198,199]

CONCLUDING SUMMARY

Adverse childhood experiences (ACEs) continue to be widely prevalent among children and adolescents in the United States, with significant positive associations with biological risk as assessed from objectively assessed clinical biomarkers, cardio-metabolic risk factors, and all-cause premature mortality through young adulthood. This is an important finding because these results are based on the analysis of a young cohort who were in their mid 30s or early 40s, and therefore, sheds light on the need for early detection and intervention to address poor health outcomes associated with ACEs. Further, the results show that early life social support did not modify the relationship between ACEs and overall cardiometabolic risk but modified the relationship between ACEs and all-cause mortality. This suggests that among young adults, early life social support reduced premature mortality associated with ACEs through mechanism other than biological mechanisms. Other plausible mechanisms through which social support may modify increased mortality risk is psychological or behavioral pathways. It is crucial to note that resilience to trauma can take many forms including having a safe, supportive, and nurturing relationships; it needs to be studied from a multilevel and life-course perspective that incorporates a holistic approach to ACEs prevention and early identification of poor health outcomes. Future research should explore the interdependence between psychological resilience and communal resilience, and how they contribute to health outcomes across life. Healthcare interventions should prioritize early identification of population at risk, trauma-informed care, support systems, and targeted health education to mitigate the long-term health consequences of ACEs and promote resilience across the lifespan. Building and/or strengthening healthcare systems that integrates trauma awareness and prevention strategies can potentially help foster healthier health trajectories for young adults and reduce the societal burden of preventable chronic diseases and premature death. The results also open new avenues for future research on the role of social experiences and determinants in cardiovascular disease risk prediction to improve health equity, and the identification of

interventions that foster positive psychological adaptation following the experience of trauma or adversity to reduce early deaths in the young population. Advanced epidemiological methods that incorporate causal inference approaches can help correct for additional biases in estimating causal effects of social exposures. As youths today are increasingly exposed to changing family, school, and socio-political environment, public health surveillance is fundamental to identify both the detrimental and beneficial health effects of these experiences.

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APPENDICES

APPENDIX A

Associations with cardiometabolic risk factors

ACEs, social support, and general obesity

ACEs was associated with general obesity independently of social support, and even after adjustment for potential baseline confounders and young adulthood mediators (aOR: 1.22 [95% CI: 1.09, 1.36]; p<0.001) (Table A.1). There was statistically significant interaction of ACEs with social support at p<0.1 for general obesity (Table A.2). We observed significant effect modification by sex between ACEs and general obesity (Table A.3), but not by race/ethnicity.

ACEs categories	General obesity	Abdominal obesity	Hypertension	Diabetes	Hyperlipidemia
0 ACE	Ref.	Ref.	Ref.	Ref.	Ref.
1 ACE	1.21 (0.96, 1.51); p=0.100	1.19 (0.96, 1.48); p=0.113	1.34 (1.06, 1.69); p=0.012	1.27 (0.85, 1.90); p=0.241	1.14 (0.85, 1.51); p=0.383
2 ACEs	1.21 (0.90, 1.63); p=0.212	1.32 (0.99, 1.76); p=0.058	1.22 (0.90, 1.64); p=0.202	1.04 (0.59, 1.76); p=0.886	0.99 (0.66, 1.45); p=0.953
3 ACEs	1.81 (1.22, 2.71); p=0.003	1.57 (1.06, 2.32); p=0.023	0.67 (0.43, 1.02); p=0.069	2.43 (1.38, 4.14); p=0.001	1.14 (0.69, 1.82); p=0.587
4 or more ACEs	2.21 (1.39, 3.55); p<0.001	2.02 (1.28, 3.23); p=0.003	1.43 (0.89, 2.26); p-0.129	2.36 (1.17, 4.52); p=0.012	1.14 (0.61, 2.00); p=0.665

	General obesity	
ACEs categories	Males	Females
0 ACE	Ref.	Ref.
1 ACE	1.01 (0.70, 1.45); p=0.947	1.38 (1.02, 1.86); p=0.035
2 ACEs	1.14 (0.70, 1.84); p=0.586	1.23 (0.84, 1.82); p=0.285
3 ACEs	1.42 (0.71, 2.85); p=0.317	2.22 (1.35, 3.68); p=0.002
4 or more ACEs	2.09 (0.88, 5.03); p=0.094	2.49 (1.41, 4.46); p=0.002

	Abdominal obesity	
ACEs categories	Males	Females
0 ACE	Ref.	Ref.
1 ACE	1.09 (0.76, 1.57); p=0.633	1.36 (1.02, 1.82); p=0.035
2 ACEs	1.28 (0.78, 2.06); p=0.317	1.43 (0.97, 2.10); p=0.068
3 ACEs	1.42 (0.72, 2.79); p=0.304	1.67 (1.01, 2.82); p=0.047
4 or more ACEs	3.10 (1.32, 7.36); p=0.009	1.64 (0.93, 2.94); p=0.091

	Hypertension	
ACEs categories	Males	Females
0 ACE	Ref.	Ref.
1 ACE	1.35 (0.96, 1.88); p=0.080	1.31 (0.94, 1.82); p=0.109
2 ACEs	1.13 (0.72, 1.76); p=0.593	1.27 (0.82, 1.93); p=0.269
3 ACEs	0.75 (0.38, 1.42); p=0.394	0.70 (0.38, 1.23); p=0.229
4 or more ACEs	2.98 (1.32, 6.89); p=0.009	1.08 (0.57, 1.99); p=0.794

	Diabetes	
ACEs categories	Males	Females
0 ACE	Ref.	Ref.
1 ACE	1.41 (0.72, 2.76); p=0.305	1.17 (0.69, 1.95); p=0.559
2 ACEs	0.92 (0.35, 2.20); p=0.862	1.06 (0.51, 2.05); p=0.871
3 ACEs	1.99 (0.71, 5.16); p=0.169	2.50 (1.25, 4.86); p=0.007
4 or more ACEs	11.17 (3.61, 33.49); p<0.001	1.11 (0.39, 2.72); p=0.827

	Hyperlipidemia	
ACEs categories	Males	Females
0 ACE	Ref.	Ref.
1 ACE	1.12 (0.74, 1.68); p=0.585	1.11 (0.72, 1.69); p=0.625
2 ACEs	1.04 (0.60, 1.76); p=0.882	0.88 (0.47, 1.55); p=0.670
3 ACEs	0.71 (0.29, 1.55); p=0.423	1.62 (0.86, 2.93); p=0.122
4 or more ACEs	1.67 (0.64, 4.01); p=0.269	0.96 (0.40, 2.07); p=0.921

Table A.1: Main effects of ACEs and social support on general obesity

	Model 1	**Model 2**	**Model 3**
Characteristic	**OR (95% CI); p**	**OR (95% CI); p**	**OR (95% CI); p**
Number of ACEs	1.24 (1.12, 1.38); p<0.001	1.26 (1.13, 1.41); p<0.001	1.22 (1.09, 1.36); p<0.001
Social support			
high	—	—	—

low	0.907 (0.671, 1.23); p=0.524	0.889 (0.647, 1.22); p=0.463	0.869 (0.635, 1.19); p=0.376

Model 1: adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.
Model 2: further adjusted for baseline self-rated health, BMI class, physical activity, smoking, alcohol use, and depressive symptoms.
Model 3: further adjusted for participant educational attainment, marital status, and health insurance status.

Table A.2: Interaction effects of ACEs and social support on general obesity

Characteristic	**OR**	**95% CI**	**p-value**
Number of ACEs	1.03	0.822, 1.28	0.817
Social support			
high	—	—	
low	0.763	0.533, 1.09	0.138
Number of ACEs * Social support			
Number of ACEs * low	1.27	0.994, 1.63	0.056

Model 1: adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

Table A.3: Interaction effects of ACEs and sex on general obesity

Characteristic	**OR**	**95% CI**	**p-value**
Number of ACEs	1.39	1.22, 1.59	<0.001
Sex			
female	—	—	
male	1.35	0.918, 1.98	0.126
Number of ACEs * Sex			
Number of ACEs * male	0.794	0.643, 0.981	0.033

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

ACEs, social support, and central obesity

Similar to general obesity, ACEs predicted central obesity independently of social support, even after adjustment for potential young adulthood mediators (aOR: 1.19 [95% CI: 1.05, 1.35]; p=0.007) (Table A.4). Sex and social support did not modify the relationship between ACEs and central obesity. There was some evidence of effect modification by race/ethnicity, with non-Hispanic Others group having significantly greater odds compared to Hispanics at p<0.1 (Table A.5).

Table A.4: Main effects of ACEs and social support on central obesity

	Model 1	Model 2	Model 3
Characteristic	**OR (95% CI); p**	**OR (95% CI); p**	**OR (95% CI); p**
Number of ACEs	1.21 (1.08, 1.36); p=0.001	1.22 (1.07, 1.38); p=0.002	1.19 (1.05, 1.35); p=0.007
Social support			
high	—	—	—
low	0.872 (0.666, 1.14); p=0.316	0.828 (0.621, 1.10); p=0.196	0.806 (0.603, 1.08); p=0.143

Model 1: adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.
Model 2: further adjusted for baseline self-rated health, BMI class, physical activity, smoking, alcohol use, and depressive symptoms.
Model 3: further adjusted for participant educational attainment, marital status, and health insurance status.

Table A.5: Interaction effects of ACEs and race/ethnicity on central obesity

Characteristic	**OR**	**95% CI**	**p-value**
Number of ACEs	1.07	0.777, 1.49	0.661
Race/ethnicity			
Hispanic	—	—	
Non-Hispanic Black	1.84	0.907, 3.72	0.091
Non-Hispanic Other	0.453	0.178, 1.15	0.096
Non-Hispanic White	1.51	0.870, 2.61	0.142
Number of ACEs * Race/ethnicity			
Number of ACEs * Non-Hispanic Black	1.16	0.776, 1.74	0.462
Number of ACEs * Non-Hispanic Other	2.06	0.949, 4.46	0.067
Number of ACEs * Non-Hispanic White	1.11	0.813, 1.52	0.501

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

ACEs, social support, and hypertension

ACEs did not significantly predict the odds of hypertension (Table A.6). There was some evidence of effect modification by race/ethnicity, with ACEs exposed non-Hispanic Blacks having significantly greater odds compared to Hispanics at $p<0.1$ (Table A.7). Sex did not modify the association.

Table A.6: Main effects of ACEs and social support on hypertension

Characteristic	**OR**	**95% CI**	**p-value**

Number of ACEs	1.03	0.930, 1.15	0.535
Social support			
high	—	—	
low	0.989	0.753, 1.30	0.936

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

Table A.7: Interaction of ACEs and race/ethnicity on hypertension

Characteristic	**OR**	**95% CI**	**p-value**
Number of ACEs	0.774	0.533, 1.13	0.179
Race/ethnicity			
Hispanic	—	—	
Non-Hispanic Black	1.19	0.533, 2.66	0.669
Non-Hispanic Other	0.981	0.306, 3.15	0.974
Non-Hispanic White	0.909	0.449, 1.84	0.790
Number of ACEs * Race/ethnicity			
Number of ACEs * Non-Hispanic Black	1.49	0.942, 2.36	0.088
Number of ACEs * Non-Hispanic Other	1.05	0.482, 2.30	0.897
Number of ACEs * Non-Hispanic White	1.36	0.908, 2.03	0.135

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

ACEs, social support, and diabetes

ACEs was not associated with diabetes, after adjustment for baseline socio-demographic confounders (Table A.8). However, there was significant interaction between ACEs and sex on diabetes, with males exposed to ACEs having significantly greater odds of diabetes compared to females (Table A.9).

Table A.8: Main effects of ACEs and social support on diabetes

Characteristic	**OR**	**95% CI**	**p-value**
Number of ACEs	1.20	0.937, 1.53	0.149
Social support			
high	—	—	
low	1.31	0.779, 2.22	0.304

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

Table A.9: Interaction of ACEs and sex on diabetes

Characteristic	OR	95% CI	p-value
Number of ACEs	0.970	0.770, 1.22	0.798
Sex			
female	—	—	
male	0.661	0.335, 1.30	0.229
Number of ACEs * Sex			
Number of ACEs * male	1.47	1.01, 2.15	0.045

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

ACEs, social support, and hyperlipidemia

After adjusting for baseline sociodemographic variables along with social support, ACEs was not associated with hyperlipidemia in the main effects model (Table A.10). However, in the model with interaction term between ACEs and race, ACEs was significantly negatively associated with hyperlipidemia. There was significant interaction between ACEs and race/ethnicity on hyperlipidemia, with non-Hispanic Blacks, non-Hispanic Whites and non-Hispanic Others exposed to ACEs having significantly greater odds of diabetes compared to Hispanics (Table A.11).

Table A.10: Main effects of ACEs and social support on hyperlipidemia

Characteristic	OR	95% CI	p-value
Number of ACEs	1.06	0.902, 1.24	0.487
Social support			
high	—	—	
low	0.963	0.644, 1.44	0.851

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

Table A.11: Interaction of ACEs and race/ethnicity on hyperlipidemia

Characteristic	OR	95% CI	p-value
Number of ACEs	0.618	0.381, 1.00	0.050
Race/ethnicity			
Hispanic	—	—	
Non-Hispanic Black	0.469	0.186, 1.18	0.107
Non-Hispanic Other	1.05	0.331, 3.33	0.933
Non-Hispanic White	0.582	0.256, 1.32	0.194
Number of ACEs * Race/ethnicity			

Number of ACEs * Non-Hispanic Black	1.71	0.972, 3.00	0.063
Number of ACEs * Non-Hispanic Other	1.91	0.919, 3.99	0.082
Number of ACEs * Non-Hispanic White	1.79	1.05, 3.04	0.032

Model adjusted for baseline age, sex, race/ethnicity, parental education and parental history of obesity or diabetes.

APPENDIX B

Table B.1: Correlation among ACEs items and social support items.

	ACE 1	ACE 2	ACE 3	ACE 4	ACE 5	SS 1	SS 2	SS 3	SS 4	SS 5	SS 6	SS 7	SS 8	SS 9
ACE 1	1.00													
ACE 2	-0.02	1.00												
ACE 3	0.03	0.17	1.00											
ACE 4	0.01	0.05	0.07	1.00										
ACE 5	0.01	0.06	0.07	0.08	1.00									
SS1	-0.03	-0.05	-0.06	-0.05	-0.08	1.00								
SS2	-0.02	-0.02	-0.05	-0.06	-0.06	0.52	1.00							
SS3	-0.02	-0.03	-0.05	-0.05	-0.06	0.58	0.73	1.00						
SS4	-0.01	-0.04	-0.07	-0.06	-0.10	0.29	0.36	0.33	1.00					
SS5	-0.00	-0.03	-0.07	-0.05	-0.09	0.32	0.37	0.36	0.45	1.00				
SS6	-0.02	-0.04	-0.06	-0.06	-0.10	0.35	0.37	0.36	0.50	0.54	1.00			
SS7	-0.01	-0.05	-0.05	-0.02	-0.08	0.14	0.13	0.13	0.17	0.15	0.15	1.00		
SS8	-0.01	-0.04	-0.05	-0.03	-0.10	0.18	0.16	0.18	0.18	0.20	0.20	0.51	1.00	
SS9	-0.01	-0.06	-0.06	-0.03	-0.11	0.17	0.17	0.19	0.19	0.20	0.19	0.39	0.47	1.00

Numbers represent phi coefficients (for correlation among ACEs items) or Kendall's tau (for correlation between ACEs items and social support items and among social support items).

Table B.2: Participant characteristics by social support

	Social support level		
	High social support (n=2,967)	Moderate social support (n=6,126)	Low social support (n=3,328)
Overall	25.2%	49.4%	25.4%
Age, Mean (SD)	15.0 (1.5)	15.8 (1.4)	15.4 (1.5)
Age group			
11-15	32.6%	49.0%	18.5%
16-18	19.9%	49.6%	30.5%
Sex			
Female	24.8%	47.6%	27.6%
Male	25.7%	51.0%	23.4%

Race and ethnicity			
Hispanic	24.9%	49.5%	25.6%
Non-Hispanic White	25.6%	49.0%	25.4%
Non-Hispanic Black	24.0%	50.7%	25.3%
Non-Hispanic Other	23.6%	50.4%	26.0%
Parental education			
High school/ GED or less	23.8%	48.9%	27.3%
Some college/2-y college	24.3%	49.9%	25.8%
4-y college or greater	27.7%	49.5%	22.8%
Parental obesity or diabetes			
Yes	25.3%	50.1%	24.6%
No	25.0%	47.3%	27.8%
Body Mass Index classification			
Underweight	25.6%	47.0%	27.5%
Normal weight	25.5%	49.0%	25.5%
Overweight	24.3%	48.9%	26.8%
Obesity	24.5%	53.4%	22.1%
Physical activity, Mean score (SD)	4.4 (2.2)	3.5 (2.1)	3.9 (2.1)
Smoking			
Yes	13.8%	45.2%	41.1%
No	29.1%	50.8%	20.1%
Alcohol use			
Yes	14.0%	46.4%	39.6%
No	29.5%	50.4%	20.1%
Depressive symptoms, Mean CESD-5 score (SD)	1.4 (1.6)	3.7 (3.1)	2.1 (2.0)
ACEs			
0 ACEs	30.3%	49.7%	20.0%
1 ACEs	21.9%	49.7%	28.4%
2 or more ACEs	14.0%	47.3%	38.7%

Row percentages and mean values in the table are weighted estimates.

Table B.3: Joint effect estimates hazards of premature mortality associated with ACEs and social support among males.

ACEs	Social support	Deaths/No deaths	Without adjustment for bio-behavioral factors[a]		With adjustment for bio-behavioral factors[b]	
			HR [95% CI]; P	HD [95% CI]; P	HR [95% CI]; P	HD [95% CI]; P
Standardized ACEs score	Standardized social support score	-	0.86 [0.81, 0.91]; P<0.001	-3.15 [-4.03, -2.27]; P<0.001	0.84 [0.77, 0.91]; P<0.001	-3.43 [-4.88, -1.98]; P<0.001
0 ACEs	High	25/880	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)

	Moderate	54/1,542	1.65 [1.37, 1.99]; P<0.001	7.15 [4.54, 9.76]; P<0.001	1.51 [1.27, 1.78]; P<0.001	6.00 [3.43, 8.57]; P<0.001
	Low	14/553	1.33 [0.93, 1.89]; P=0.114	3.70 [0.13, 7.27]; P=0.042	1.04 [0.76, 1.42]; P=0.811	0.28 [-4.32, 4.88]; P= 0.904
≥1 ACEs	High	18/585	1.13 [0.55, 2.32]; P=0.747	1.28 [-7.42, 9.98]; P=0.774	1.04 [0.51, 2.11]; P=0.912	0.38 [-8.01, 8.76]; P=0.930
	Moderate	61/1,506	1.40 [1.26, 1.54]; P<0.001	4.28 [2.20, 6.36]; P<0.001	1.17 [1.06, 1.30]; P=0.001	1.86 [-0.35, 4.07]; P=0.101
	Low	48/868	2.05 [1.21, 3.50]; P=0.008	11.40 [3.99, 18.81]; P=0.002	1.60 [1.00, 2.57]; P=0.049	7.69 [1.61, 13.77] P=0.013

HDs are presented as per 10,000 individuals per year.
[a]Adjusted for age, race/ethnicity, parental education, and parental history of chronic condition.
[b]Adjusted for age, race/ethnicity, parental education, parental history of chronic condition, BMI class, physical activity, smoking, alcohol use, and depressive symptoms.

Table B.4: Joint effect estimates hazards of premature mortality associated with ACEs and social support among females.

ACEs	Social support	Deaths/No deaths	Without adjustment for bio-behavioral factors[a]		With adjustment for bio-behavioral factors[b]	
			HR_{upper} [95% CI]; P	HD_{upper} [95% CI]; P	HR_{lower} [95% CI]; P	HD_{lower} [95% CI]; P
Standardized ACEs score	Standardized social support score	-	0.85 [0.76, 0.95]; P=0.005	-2.57 [-3.50, -1.64]; P<0.001	0.85 [0.77, 0.95]; P=0.003	-2.53 [-3,50, -1.56]; P<0.001
0 ACEs	Top 50th percentile	30/1,723	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
	Bottom 50th percentile	19/1,684	0.79 [0.36, 1.72]; P=0.549	-1.65 [-6.31, 3.01]; P=0.490	0.59 [0.31, 1.15]; P=0.123	-3.81 [-7.32, -0.30]; P=0.033
≥1 ACEs	Top 50th percentile	19/1,011	1.10 [0.53, 2.32]; P=0.795	0.66 [-4.67, 5.99]; P=0.808	0.96 [0.51, 1.82]; P=0.911	-0.43 [-4.98, 4.12]; P=0.854
	Bottom 50th percentile	39/1,742	1.34 [0.82, 2.20]; P=0.247	2.54 [-1.32, 6.40]; P=0.195	0.87 [0.60,	-1.09 [-3.66,

					1.23]; P=0.422	1.48]; P=0.405

For females, we used median split comparing those falling above median with those falling at or below median to protect confidentiality of the data (due to less than 10 individuals in a cell)
HDs are presented as per 10,000 individuals per year.
[a]Adjusted for age, race/ethnicity, parental education, and parental history of chronic condition.
[b]Adjusted for age, race/ethnicity, parental education, parental history of chronic condition, BMI class, physical activity, smoking, alcohol use, and depressive symptoms.

Table B.5: Joint effect estimates hazards of premature mortality associated with ACEs and social support after excluding deaths that occurred within two and five years of exposure assessment.

ACEs	Social support	Excluding deaths within the first 2 years of baseline assessment (n=12,409; deaths=315)		Excluding deaths within the first 5 years of baseline assessment (n=12,378; deaths=284)	
		HR [95% CI]; P	HD [95% CI]; P	HR [95% CI]; P	HD [95% CI]; P
0 ACEs	High	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
	Moderate	1.31 [1.10, 1.57]; P=0.002	2.77 [1.90, 3.63]; P<0.001	1.45 [1.07, 1.98]; P=0.017	3.44 [1.87, 5.00]; P<0.001
	Low	0.87 [0.48, 1.57]; P=0.648	-1.05 [-5.99, 3.89]; P=0.677	0.98 [0.52, 1.83]; P=0.937	-1.92 [-4.92, 4.53]; P=0.937
≥1 ACEs	High	0.83 [0.46, 1.51]; P=0.543	-1.93 [-7.28, 3.42]; P=0.480	0.78 [0.44, 1.41]; P=0.413	-2.24 [-6.77, 2.29]; P=0.333
	Moderate	1.19 [0.79, 1.79]; P=0.408	1.48 [-2.24, 5.20]; P=0.436	1.23 [0.67, 2.25]; P=0.503	1.54 [-3.46, 6.54]; P=0.547
	Low	2.02 [1.12, 3.64]; P=0.020	9.24 [1.83, 16.65]; P=0.014	1.93 [0.98, 3.80]; P=0.055	7.34 [0.25, 14.43]; P=0.042

HDs are presented as per 10,000 individuals per year.
HR and HD are adjusted for age, race/ethnicity, parental education, and parental history of chronic condition.

Table B.6: Joint effect estimates hazards of premature mortality associated with ACEs and social support after including respondents 18 years or older at baseline (N*=14,320).

ACEs	Social support	Deaths/No deaths	Without adjustment for bio-behavioral factors[a]		With adjustment for bio-behavioral factors[b]	
			HR [95% CI]; P	HD [95% CI]; P	HR [95% CI]; P	HD [95% CI]; P
0 ACEs	High	45/2,027	1 (ref.)	0 (ref.)	1 (ref.)	0 (ref.)
	Moderate	85/3,668	1.52 [1.08, 2.14]; P=0.015	4.18 [1.83, 6.53]; P<0.001	1.40 [1.03, 1.90]; P=0.032	3.36 [1.34, 5.38]; P=0.001

	Low	25/1,669	0.94 [0.49, 1.82]; P=0.853	-0.31 [-5.37, 4.74]; P=0.904	0.72 [0.43, 1.22]; P=0.221	-2.92 [-6.64, 0.80]; P=0.124
≥1 ACEs	High	26/1,245	0.91 [0.53, 1.57]; P=0.744	-1.06 [-5.70, 3.59]; P=0.656	0.85 [0.49, 1.47]; P=0.566	-1.74 [-6.40, 2.92]; P=0.463
	Moderate	96/3,186	1.37 [0.93, 2.02]; P=0.108	2.85 [-0.21, 5.91]; P=0.068	1.16 [0.84, 1.62]; P=0.370	1.05 [-1.52, 3.62]; P=0.426
	Low	87/2,161	2.14 [1.48, 3.09]; P=0.003	9.71 [3.87, 15.55]; P=0.001	1.64 [1.08, 2.51]; P=0.021	6.32 [1.89, 10.75]; P=0.005

*Analytic sample obtained by removing missing sampling weights (*n* = 1,821), missing age (n=14), those missing ACEs information (*n* = 3,066), those missing social support information (n=513), and those without complete covariate information (*n* = 1,011) in subsequent steps. HDs are presented as per 10,000 individuals per year.
[a]Adjusted for age, race/ethnicity, parental education, and parental history of chronic condition.
[b]Adjusted for age, race/ethnicity, parental education, parental history of chronic condition, BMI class, physical activity, smoking, alcohol use, and depressive symptoms.

Table B.7: Hazards of premature mortality associated with individual ACEs, overall and across social support level.

	HR [95% CI]; P	HD [95% CI]; P
Parental death, overall	0.70 [0.35, 1.44]; P=0.338	-3.57 [-9.43, 2.29]; P=0.248
Parental death, moderate-high social support	0.58 [0.21, 1.62]; P=0.301	-4.84 [-11.6, 1.88]; P=0.161
Parental death, low social support	0.97 [0.38, 2.50]; P=0.951	-0.22 [-11.80, 11.40]; P=0.970
Parental divorce/separation, overall	1.28 [0.85, 1.91]; P=0.238	3.20 [-1.82, 8.22]; P=0.217
Parental divorce/separation, moderate-high social support	1.04 [0.65, 1.65]; P=0.871	0.32 [-5.12, 5.77]; P=0.913
Parental divorce/separation, low social support	1.74 [0.95, 3.18]; P=0.073	8.99 [-1.50, 19.50]; P=0.090
Household substance use, overall	1.10 [0.78, 1.54]; P=0.582	1.09 [-3.77, 5.95]; P=0.434
Household substance use, moderate-high social support	0.80 [0.47, 1.36]; P=0.417	-2.54 [-7.68, 2.60]; P=0.331
Household substance use, low social support	1.65 [0.87, 3.10]; P=0.123	7.91 [-2.63, 18.50]; P=0.134
Suicide attempt by a family member, overall	1.14 [0.53, 2.43]; P=0.737	1.72 [-7.84, 11.30]; P=0.723
Suicide attempt by a family member, moderate-high social support	0.50 [0.15, 1.67]; P=0.262	-5.53 [-12.50, 1.49]; P=0.127
Suicide attempt by a family member, low social support	1.94 [0.75, 5.02]; P=0.173	11.30 [-10.10, 32.70]; P=0.286
Community violence, overall	1.43 [0.99, 2.06]; P=0.057	4.83 [-0.68, 10.30]; P=0.086

Community violence, moderate-high social support	1.08 [0.70, 1.68]; P=0.726	0.82 [-4.93, 6.56]; P=0.780
Community violence, low social support	2.14 [1.10, 4.14]; P=0.025	12.60 [4.87, 24.70]; P=0.038

HRs and HDs are adjusted for age, sex, race/ethnicity, parental education, and parental history of chronic condition.

Table B.8: Independent effect estimates for hazards of premature mortality associated with sources of perceived social support.

	Model A	Model B	Model C	Model D
	HR [95% CI]; p	HR [95% CI]; p	HR [95% CI]; p	
Perceived support from parents				
Score (continuous)	0.90 [0.81, 1.00]; P=0.054	0.90 [0.81, 1.01]; P=0.084	0.93 [0.81, 1.06]; P=0.267	0.96 [0.85, 1.09]; P=0.556
Low	1 (ref.)	1 (ref.)	1 (ref.)	1 (ref.)
High	0.85 [0.63, 1.14]; P=0.272	0.85 [0.61, 1.17]; P=0.309	0.92 [0.65, 1.30]; P=0.637	1.02 [0.73, 1.43]; P=0.902
Perceived support from family				
Score (continuous)	0.93 [0.79, 1.10]; P=0.418	0.92 [0.77, 1.10]; P=0.360	0.94 [0.79, 1.11]; P=0.454	1.01 [0.85, 1.21]; P=0.873
Low	1 (ref.)	1 (ref.)	1 (ref.)	1 (ref.)
High	0.92 [0.66, 1.30]; P=0.659	0.91 [0.64, 1.30]; P=0.604	0.94 [0.66, 1.33]; P=0.723	1.10 [0.77, 1.58]; P=0.579
Perceived support at school				
Score (continuous)	0.80 [0.70, 0.91]; P<0.001	0.81 [0.71, 0.92]; P=0.002	0.82 [0.72, 0.93]; P=0.003	0.87 [0.75, 1.02]; P=0.083
Low	1 (ref.)	1 (ref.)	1 (ref.)	1 (ref.)
High	0.71 [0.53, 0.96]; P=0.024	0.73 [0.54, 0.99]; P=0.046	0.76 [0.56, 1.02]; P=0.071	0.86 [0.62, 1.20]; P=0.375
Number of social support sources				
0	1 (ref.)	1 (ref.)	1 (ref.)	1 (ref.)
1	0.79 [0.55, 1.15]; P=0.232	0.79 [0.54, 1.16]; P=0.232	0.82 [0.56, 1.19]; P=0.299	0.93 [0.63, 1.36]; P=703
2 or 3	0.69 [0.50, 0.95]; P=0.021	0.69 [0.50, 0.96]; P=0.027	0.73 [0.53, 1.01]; P=0.059	0.91 [0.63, 1.30]; P=602

Model A is unadjusted; Model B adjusts for age, sex, and race/ethnicity, parental education and parental history of diabetes or obesity; Model C adjusts for Model B estimators plus ACEs categories; Model D adjusts for Model C estimators plus BMI class, physical activity, smoking, alcohol use, and depressive symptoms.

APPENDIX C

Table C.1: Socio-demographic distribution of all participants by year of NSCH survey

	NSCH year of survey				
	2016	**2017**	**2018**	**2019**	**2020**
N, unweighted	50,212	21,599	30,530	29,433	42,777
N, weighted	73,350,040	73,424,383	73,433,138	73,133,076	72,774,300
Age in years, mean (SD)	8.60 (5.15)	8.61 (5.16)	8.64 (5.16)	8.66 (5.15)	8.69 (5.14)
Age <= 5 years	32.32%	32.27%	32.12%	32.10%	31.98%
Age 6 to 11 years	33.85%	33.87%	33.81%	33.57%	33.37%
Age 12 to 17 years	33.83%	33.86%	34.07%	34.33%	34.65%
Female	48.94%	48.87%	48.89%	48.87%	48.89%
Male	51.06%	51.13%	51.11%	51.13%	51.11%
Hispanic	24.54%	24.94%	25.24%	25.64%	25.67%
Non-Hispanic White	51.89%	50.96%	50.51%	50.21%	50.06%
Non-Hispanic Black	12.71%	13.40%	13.37%	13.29%	13.29%
Non-Hispanic Asian	4.52%	4.65%	4.75%	4.52%	4.62%
Non-Hispanic Other	6.34%	6.05%	6.14%	6.34%	6.36%
0-99% FPL	21.64%	20.80%	19.74%	18.98%	17.81%
100-199% FPL	22.10%	21.39%	21.82%	21.31%	21.62%
200-399% FPL	26.78%	27.13%	27.11%	28.90%	29.39%
400% FPL or greater	29.48%	30.68%	31.33%	30.81%	31.17%

Note: Percentages presented in the table are weighted percentages.

Table C.2: Distribution of non-response to ACEs and PPFs questions across participant characteristics, NSCH 2016-2020

	ACEs		PPFs	
	Missing (N=8,109)	Not missing (N=116,896)	Missing (N=8,041)	Not missing (N=116,964)
Age in years, mean (SD)	12.13 (3.41)	12.11 (3.45)	12.16 (3.46)	12.11 (3.45)
Age 6 to 11 years	6.42%	93.58%	6.40%	93.60%
Age 12 to 17 years	6.54%	93.46%	6.46%	93.54%
Female	6.57%	93.43%	6.41%	93.59%
Male	6.40%	93.60%	6.45%	93.55%

Hispanic	7.80%	92.20%	7.89%	92.11%
Non-Hispanic White	5.59%	94.41%	5.71%	94.28%
Non-Hispanic Black	11.20%	88.80%	11.01%	88.98%
Non-Hispanic Asian	8.92%	91.08%	7.60%	92.40%
Non-Hispanic Other	6.81%	93.19%	5.83%	94.17%
0-99% FPL	10.64%	89.36%	10.07%	89.93%
100-299% FPL	8.13%	91.87%	7.85%	92.15%
200-399% FPL	5.66%	94.34%	5.82%	94.18%
400% FPL or greater	5.33%	94.67%	5.34%	94.66%

Note: Percentages presented in the table are unweighted percentages.

Table C.3: Prevalence of 0, 1-2 and >2 ACEs before and after change in wording of economic adversity item

	2016-2017 (Unweighted N =47,463; Weighted N = 45,449,058)			2018-2020 (Unweighted N =69,433; Weighted N = 45,767,605)		
	0 ACEs [95% CI][b]	1-2 ACEs [95% CI][b]	>2 ACEs [95% CI][b]	0 ACEs [95% CI][b]	1-2 ACEs [95% CI][b]	>2 ACEs [95% CI][b]
All	50.0 [48.9, 51.0]	36.8 [35.8, 37.9]	13.2 [12.5, 13.9]	53.8 [53.0, 54.7]	33.9 [33.1, 34.7]	12.2 [11.7, 12.8]
6-11 years	54.4 [52.8, 56.0]	34.0 [32.5, 35.5]	11.6 [10.5, 12.7]	58.3 [57.1, 59.5]	31.9 [30.8, 33.0]	9.8 [9.1, 10.5]
12-17 years	45.5 [44.1, 47.0]	39.6 [38.2, 41.1]	14.8 [13.9, 15.8]	49.5 [48.3, 50.6]	35.9 [34.8, 37.0]	14.6 [13.7, 15.5]
Female	49.9 [48.4, 51.5]	37.0 [35.4, 38.5]	13.1 [12.1, 14.2]	53.5 [52.3, 54.7]	34.3 [33.2, 35.5]	12.2 [11.4, 13.1]
Male	50.0 [48.5, 51.5]	36.7 [35.3, 38.2]	13.3 [12.3, 14.3]	54.2 [53.0, 55.3]	33.6 [32.5, 34.6]	12.3 [11.5, 13.0]
Hispanic	47.8 [44.7, 50.9]	40.2 [37.3, 43.2]	12.0 [10.3, 13.6]	51.3 [48.9, 53.6]	36.1 [33.9, 38.4]	12.6 [11.0, 14.2]
Non-Hispanic White	54.7 [53.6, 55.7]	33.0 [32.0, 34.0]	12.4 [11.6, 13.2]	57.8 [57.0, 58.6]	31.5 [30.7, 32.3]	10.7 [10.2, 11.2]
Non-Hispanic Black	32.0 [28.8, 35.2]	50.0 [46.7, 53.4]	18.0 [15.5, 20.5]	39.6 [37.1, 42.0]	42.5 [40.2, 45.0]	17.8 [15.9, 19.8]

Non-Hispanic Asian	72.2 [68.0, 76.4]	24.2 [20.3, 28.2]	3.6 [1.5, 5.6]	75.5 [72.6, 78.5]	21.7 [19.0, 24.4]	2.8 [1.4, 4.2]
Non-Hispanic Other	42.0 [38.7, 45.4]	35.7 [32.5, 38.9]	22.2 [18.9, 25.6]	45.3 [42.5, 48.2]	35.7 [33.0, 38.3]	19.0 [16.7, 21.4]
0-99% FPL	32.1 [29.1, 35.1]	45.8 [42.9, 48.7]	22.1 [19.8, 24.3]	37.7 [35.4, 40.0]	41.4 [39.1, 43.7]	20.8 [19.0, 22.6]
100-299% FPL	38.4 [35.7, 41.1]	44.0 [41.3, 46.7]	17.6 [15.8, 19.4]	44.2 [42.1, 46.3]	37.8 [35.9, 39.7]	18.0 [16.5, 19.6]
200-399% FPL	50.1 [48.2, 52.0]	37.8 [36.0, 39.6]	12.1 [10.9, 13.3]	53.5 [52.1, 55.0]	35.9 [34.5, 37.3]	10.6 [9.7, 11.5]
400% FPL or greater	69.4 [68.1, 70.7]	25.2 [24.0, 26.4]	5.4 [4.6, 6.1]	70.0 [68.9, 71.1]	25.2 [24.1, 26.2]	4.8 [4.4, 5.3]

Note: Numbers presented in the table are weighted percentages.

Table C.4: Prevalence of PPFs across ACEs categories after exclusion of the economic adversity item

	2016-20 (Unweighted N =112,426; Weighted N = 43,711,487)								
	No ACEs			1-2 ACE			3-9 ACEs		
	0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	6-7 PPFs [95% CI][b]	0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	6-7 PPFs [95% CI][b]	0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	6-7 PPFs [95% CI][b]
Overall	7.1 [6.5, 7.7]	48.9 [48.0, 49.7]	44.0 [43.2, 44.9]	11.0 [10.2, 11.9]	58.4 [57.3, 59.6]	30.6 [29.6, 31.6]	19.0 [17.2, 20.8]	59.5 [57.3, 61.6]	21.5 [19.7, 23.3]
6-11 years	7.4 [6.6, 8.3]	51.1 [49.9, 52.4]	41.4 [40.3, 42.6]	11.3 [10.0, 12.6]	60.9 [59.2, 62.7]	27.8 [26.2, 29.3]	18.6 [15.8, 21.3]	60.7 [57.2, 64.1]	20.8 [17.9, 23.6]
12-17 years	6.7 [5.9, 7.6]	46.2 [44.9, 47.4]	47.1 [45.9, 48.3]	10.8 [9.7, 11.9]	56.4 [54.9, 57.9]	32.8 [31.5, 34.2]	19.3 [17.0, 21.7]	58.7 [55.9, 61.5]	22.0 [19.7, 24.3]
Female	6.1 [5.3, 6.9]	48.3 [47.0, 49.6]	45.6 [44.4, 46.8]	9.7 [8.5, 10.9]	57.4 [55.7, 59.1]	32.9 [31.3, 34.4]	18.6 [15.8, 21.3]	59.1 [55.8, 62.3]	22.4 [19.6, 25.1]
Male	8.1 [7.2, 8.9]	49.4 [48.2, 50.6]	42.5 [41.4, 43.7]	12.3 [11.1, 13.5]	59.4 [57.8, 60.9]	28.4 [27.0, 29.7]	19.5 [17.1, 21.8]	59.9 [57.0, 62.8]	20.6 [18.3, 22.9]
Non-Hispanic White	3.6 [3.2, 3.9]	42.7 [41.9, 43.6]	53.7 [52.9, 54.5]	7.4 [6.7, 8.0]	55.1 [53.9, 56.4]	37.5 [36.3, 38.7]	15.8 [14.0, 17.6]	59.0 [56.6, 61.4]	25.2 [23.2, 27.2]

Hispanic	12.6 [10.7, 14.5]	58.2 [55.6, 60.7]	29.2 [27.0, 31.5]	16.1 [13.6, 18.6]	61.7 [58.6, 64.8]	22.2 [19.7, 24.7]	21.7 [17.0, 26.3]	61.8 [56.3, 67.4]	16.5 [12.1, 20.9]
Non-Hispanic Black	10.9 [8.4, 13.4]	56.5 [53.2, 59.7]	32.6 [29.7, 35.6]	11.1 [9.1, 13.0]	63.0 [60.2, 65.8]	25.9 [23.5, 28.3]	25.3 [20.2, 30.4]	54.6 [48.8, 60.4]	20.1 [15.0, 25.2]
Non-Hispanic Asian	11.8 [9.4, 14.2]	51.9 [48.7, 55.0]	36.3 [33.4, 39.2]	25.2 [19.2, 31.3]	49.3 [43.4, 55.1]	25.5 [21.0, 30.0]	15.9 [4.0, 27.7]	62.8 [38.9, 86.6]	21.4 [0, 45.1]
Non-Hispanic Other	5.7 [4.4, 7.1]	51.6 [48.6, 54.7]	42.6 [39.6, 45.6]	11.8 [9.4, 14.2]	60.5 [56.7, 64.2]	27.7 [24.2, 31.2]	16.5 [11.5, 21.5]	64.4 [58.5, 70.4]	19.1 [14.6, 23.5]
0-99% FPL	14.9 [12.6, 17.3]	57.4 [54.4, 60.5]	27.6 [24.9, 30.3]	17.0 [14.6, 19.3]	62.3 [59.4, 65.1]	20.8 [18.5, 23.0]	27.0 [22.9, 31.0]	58.0 [53.7, 62.3]	15.1 [12.3, 17.8]
100-199% FPL	11.5 [9.6, 13.3]	56.9 [54.5, 59.4]	31.6 [29.4, 33.8]	12.3 [10.5, 14.1]	63.5 [61.1, 65.9]	24.2 [22.3, 26.1]	19.3 [16.0, 22.5]	62.9 [58.8, 66.9]	17.9 [14.7, 21.0]
200-399% FPL	6.6 [5.5, 7.6]	49.7 [48.2, 51.2]	43.7 [42.2, 45.2]	9.5 [8.1, 10.8]	57.1 [55.1, 59.2]	33.4 [31.5, 35.2]	14.1 [11.2, 17.0]	59.5 [55.3, 63.7]	26.4 [22.3, 30.5]
400% FPL or greater	2.5 [2.2, 2.8]	41.1 [40.0, 42.2]	56.5 [55.4, 57.5]	5.8 [4.8, 6.8]	51.2 [49.2, 53.1]	43.0 [41.1, 44.9]	11.5 [8.3, 14.6]	55.0 [50.7, 59.4]	33.5 [29.4, 37.6]

Note: Numbers presented in the table are weighted percentages.

Table C.5: Prevalence of PPFs across ACEs categories [95% CI] by socio-demographic characteristics, NSCH 2016-2020 (Unweighted n =112,178; Weighted n = 43,605,030)

	Respondent distribution (%)[a]	0 ACEs			P-value[c]	1-2 ACEs			P-value[c]	>2 ACEs			P-value[c]
		0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	>5 PPFs [95% CI][b]		0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	>5 PPFs [95% CI][b]		0-2 PPFs [95% CI][b]	3-5 PPFs [95% CI][b]	>5 PPFs [95% CI][b]	
Overall	100%	5.8 [5.3, 6.4]	47.3 [46.4, 48.2]	46.8 [45.9, 47.7]	-	11.4 [10.6, 12.3]	58.7 [57.5, 59.8]	29.9 [28.9, 30.9]	-	19.1 [17.5, 20.7]	59.9 [58.0, 61.8]	21.0 [19.4, 22.6]	-
6-11 years	49.7%	6.3 [5.4, 7.1]	49.4 [48.1, 50.7]	44.3 [43.0, 45.5]	<0.001	11.8 [10.6, 13.1]	61.6 [60.0, 63.3]	26.6 [25.2, 28.0]	<0.001	18.5 [16.2, 20.9]	61.1 [58.1, 64.1]	20.3 [17.8, 22.8]	0.6
12-17 years	50.3%	5.3 [4.6, 6.1]	44.9 [43.6,	49.7 [48.5,		11.1 [9.9,	56.1 [54.6,	32.8 [31.5,		19.5 [17.4,	59.0 [56.6,	21.5 [19.5,	

			46.2]	51.0]		12.3]	57.6]	34.2]		21.6]	61.5]	23.5]	
Female	48.9%	4.8 [4.0, 5.5]	46.5 [45.2, 47.9]	48.7 [47.3, 50.0]	<0.001	10.4 [9.1, 11.7]	57.9 [56.3, 59.6]	31.7 [30.2, 33.1]	0.002	18.3 [15.9, 20.6]	59.7 [56.8, 62.5]	22.1 [19.7, 24.4]	0.3
Male	51.1%	6.9 [6.0, 7.7]	48.1 [46.8, 49.3]	45.0 [43.8, 46.3]		12.4 [11.2, 13.6]	59.4 [57.8, 60.9]	28.2 [26.9, 29.5]		19.9 [17.8, 22.0]	60.1 [57.5, 62.7]	20.0 [17.9, 22.1]	
Non-Hispanic White	51.7%	2.8 [2.5, 3.1]	41.2 [40.3, 42.0]	56.0 [55.2, 56.9]	<0.001	7.3 [6.6, 7.9]	55.1 [53.9, 56.3]	37.6 [36.5, 38.8]	<0.001	15.9 [14.3, 17.6]	59.9 [57.7, 62.0]	24.2 [22.4, 26.0]	<0.001
Hispanic	25.1%	11.1 [9.1, 13.1]	57.0 [54.2, 59.7]	31.9 [29.5, 34.4]		16.9 [14.4, 19.3]	62.3 [59.3, 65.2]	20.9 [18.5, 23.2]		21.7 [17.7, 25.7]	62.3 [57.4, 67.2]	16.0 [12.2, 19.7]	
Non-Hispanic Black	12.9%	8.6 [6.4, 10.8]	55.5 [52.0, 59.0]	35.9 [32.6, 39.2]		11.6 [9.4, 13.8]	63.3 [60.5, 66.1]	25.1 [22.7, 27.4]		25.0 [20.6, 29.4]	55.2 [50.2, 60.2]	19.8 [15.5, 24.1]	
Non-Hispanic Asian	4.4%	10.6 [8.4, 12.7]	51.4 [48.2, 54.7]	38.0 [35.0, 41.0]		26.3 [20.1, 32.5]	51.3 [45.6, 57.1]	22.4 [18.4, 26.3]		20.2 [8.3, 32.2]	58.2 [38.4, 77.9]	21.6 [2.2, 41.0]	
Non-Hispanic Other	5.9%	4.7 [3.5, 6.0]	50.4 [47.1, 53.7]	44.9 [41.6, 48.1]		11.9 [9.6, 14.3]	60.9 [57.6, 64.3]	27.1 [24.2, 30.0]		15.9 [11.6, 20.1]	63.1 [57.4, 68.9]	21.0 [15.8, 26.2]	
0-99% FPL	18.5%	12.9 [10.2, 15.6]	56.7 [53.3, 60.2]	30.3 [27.2, 33.5]	<0.001	17.1 [14.8, 19.4]	62.5 [59.8, 65.3]	20.4 [18.2, 22.5]	<0.001	26.1 [22.7, 29.5]	57.8 [54.1, 61.6]	16.1 [13.5, 18.7]	<0.001

100-199% FPL	21.7%	9.4 [7.6, 11.1]	56.3 [53.5, 59.0]	34.3 [31.8, 36.8]		13.6 [11.5, 15.8]	62.2 [59.8, 64.6]	24.2 [22.3, 26.0]		19.2 [16.4, 21.9]	63.6 [60.1, 67.0]	17.3 [14.6, 19.9]	
200-399% FPL	28.3%	5.9 [4.8, 7.0]	47.7 [46.1, 49.4]	46.4 [44.8, 48.0]		9.7 [8.5, 11.1]	58.2 [56.3, 60.1]	32.0 [30.3, 33.8]		13.9 [11.4, 16.4]	60.0 [56.3, 63.7]	26.1 [22.5, 29.6]	
400% FPL or greater	31.6%	2.4 [2.0, 2.7]	40.6 [39.5, 41.7]	57.1 [56.0, 58.1]		5.5 [4.6, 6.4]	51.5 [49.6, 53.3]	43.1 [41.2, 44.9]		12.0 [9.0, 15.1]	55.7 [51.5, 59.8]	32.3 [28.5, 36.1]	

Abbreviation: ACEs: Adverse Childhood Experiences; PPFs: Positive Protective Factors; CI: Confidence Interval; NSCH: National Survey of Children's Health; FPL: Federal Poverty Level
[a]The denominator is the total weighted number of respondents. [b]The denominator is the total weighted number of respondents for the respective category of age, sex, race/ethnicity, or household poverty level. [c]Chi-squared test with Rao & Scott's second-order correction

Table C.6: Across-state prevalence [95% CI] of PPFs across ACEs categories among school-age children in the US, NSCH 2016-2020 (N=112,178)

	0 ACE			1-2 ACEs			3-9 ACEs		
	Weighted % (95% CI)			Weighted % (95% CI)			Weighted % (95% CI)		
	0-2 PPFs	3-5 PPFs	6-7 PPFs	0-2 PPFs	3-5 PPFs	6-7 PPFs	0-2 PPFs	3-5 PPFs	6-7 PPFs
United States	5.8 [5.3, 6.4]	47.3 [46.4, 48.2]	46.8 [45.9, 47.7]	11.4 [10.6, 12.3]	58.7 [57.5, 59.8]	29.9 [28.9, 30.9]	19.1 [17.5, 20.7]	59.9 [58.0, 61.8]	21.0 [19.4, 22.6]
State									
Alabama	2.9 [1.5, 4.3][#]	44.7 [40.7, 48.7]	52.4 [48.4, 56.4][*]	10.7 [6.9, 14.6]	59.2 [54.1, 64.2]	30.1 [25.6, 34.6]	17.2 [8.7, 25.6]	61.3 [51.7, 71.0]	21.5 [13.5, 29.6]
Alaska	6.4 [3.8, 8.9]	50.1 [45.8, 54.5]	43.5 [39.3, 47.8]	11.3 [7.1, 15.4]	59.4 [53.4, 65.3]	29.4 [24, 34.8]	20.3 [13.6, 27]	55.1 [47.6, 62.7]	24.6 [18.1, 31]
Arizona	6.3 [3.9, 8.7]	50.4 [46.1, 54.7]	43.3 [39.1, 47.5]	11.2 [7.9, 14.6]	63.5 [58.3, 68.6]	25.3 [20.8, 29.9][#]	26.8 [18.9, 34.7]	52.7 [44.2, 61.2]	20.5 [13.9, 27.1]
Arkansas	4.2 [2.2, 6.2]	48.1 [43.5, 52.7]	47.7 [43.2, 52.2]	10.7 [7.3, 14.1]	54.9 [49.5, 60.3]	34.4 [29.3, 39.5]	17.1 [10.9, 23.3]	59.1 [51.7, 66.4]	23.9 [17.7, 30.1]
California	9.8 [7, 12.6][*]	54.8 [50.7, 58.8][*]	35.4 [31.7, 39.2][#]	15.9 [11.3, 20.6][*]	60.6 [54.9, 66.2]	23.5 [19, 28][#]	24.2 [15.5, 32.8]	56.2 [45.9, 66.6]	19.6 [11.1, 28.1]

Colorado	3.9 [2.3, 5.5][#]	47.8 [44, 51.7]	48.3 [44.5, 52]	8.9 [5.7, 12.1]	57.8 [52.8, 62.7]	33.3 [28.8, 37.9]	17.2 [9.6, 24.9]	61.1 [52.4, 69.7]	21.7 [14.7, 28.8]
Connecticut	4.9 [3.3, 6.5]	43.5 [39.8, 47.2][#]	51.6 [47.9, 55.3][*]	11.5 [7.3, 15.7]	54.7 [49.2, 60.3]	33.8 [28.7, 38.9]	18.3 [9.9, 26.7]	64.8 [55.1, 74.4]	16.9 [10.5, 23.4]
Delaware	7 [4.5, 9.6]	49.7 [45.6, 53.8]	43.3 [39.3, 47.3]	9.7 [6.5, 12.9]	58.2 [53, 63.5]	32.1 [27.2, 37]	23.2 [14.7, 31.7]	58.9 [49.8, 68.1]	17.9 [11.4, 24.3]
District of Columbia	4.3 [2.4, 6.3]	58 [52.8, 63.2][*]	37.7 [32.7, 42.7][#]	10 [6.2, 13.8]	64.6 [58.3, 70.8]	25.5 [19.9, 31.1]	16.6 [8.1, 25]	68.4 [58.1, 78.7]	15 [7.4, 22.7]
Florida	5 [2.8, 7.1]	51.6 [47.3, 55.9][*]	43.5 [39.3, 47.6]	10.7 [7.6, 13.9]	62.1 [57.5, 66.7]	27.2 [23.1, 31.3]	20.6 [12.9, 28.4]	59.8 [50.6, 68.9]	19.6 [12.3, 26.9]
Georgia	7.4 [4.5, 10.3]	47.5 [43.4, 51.6]	45.1 [41.2, 49.1]	10.2 [7, 13.4]	61.7 [56.8, 66.7]	28.1 [23.7, 32.4]	23 [14.6, 31.4]	59.8 [50.8, 68.8]	17.2 [10.9, 23.5]
Hawaii	6.9 [5, 8.8]	55.6 [51.8, 59.5][*]	37.4 [33.7, 41.1][#]	10.4 [7.6, 13.2]	57 [51.8, 62.1]	32.6 [27.7, 37.6]	11 [6, 16][#]	65.9 [57.3, 74.5]	23.1 [15.2, 31.1]
Idaho	3.3 [1.3, 5.3][#]	40.5 [36.4, 44.5][#]	56.2 [52.2, 60.3][*]	7.3 [4.6, 10.1][#]	56.7 [51.8, 61.6]	36 [31.3, 40.6][*]	13.2 [8.7, 17.6][#]	63.7 [56.5, 70.9]	23.1 [16.8, 29.4]
Illinois	3.9 [2.4, 5.4][#]	46.6 [42.7, 50.6]	49.5 [45.6, 53.4]	8.9 [6.2, 11.5]	66.9 [62.1, 71.7][*]	24.3 [20.1, 28.5][#]	19.9 [11.7, 28.1]	66.9 [57.5, 76.2]	13.3 [7.1, 19.5][#]
Indiana	4.7 [2.7,6.6]	45.7 [41.7, 49.7]	49.6 [45.7, 53.6]	10.5 [7, 14.1]	57 [52.2, 61.7]	32.5 [28.2, 36.7]	19.2 [11.7, 26.6]	60.7 [52.5, 69]	20.1 [14, 26.2]
Iowa	3.1 [1.9, 4.4][#]	40.2 [36.7, 43.8][#]	56.7 [53.1, 60.2][*]	8 [4.7, 11.4]	53.2 [48.1, 58.3][#]	38.8 [33.9, 43.7][*]	17.7 [11.2, 24.2]	58.8 [50.5, 67.1]	23.6 [17.1, 30]
Kansas	3.2 [1.8, 4.7][#]	42.3 [38.6, 46.1][#]	54.5 [50.7, 58.3][*]	8.5 [5.3, 11.6]	57.4 [52.1, 62.6]	34.2 [29.4, 38.9]	16.5 [9.5, 23.6]	61.5 [53.1, 69.9]	22 [15.5, 28.5]
Kentucky	2.9 [1.8, 4.1][#]	41.2 [37.1, 45.2][#]	55.9 [51.8, 60][*]	8.5 [5.7, 11.3][#]	62.9 [58.2, 67.5]	28.6 [24.4, 32.9]	14.1 [8.9, 19.3]	63.6 [56.2, 71]	22.3 [16.1, 28.5]
Louisiana	5.3 [2.9, 7.7]	48.6 [44.2, 52.9]	46.2 [41.9, 50.4]	10.6 [7.5, 13.7]	56.2 [51.2, 61.3]	33.2 [28.3, 38]	23.2 [15.7, 30.7]	55.8 [47.6, 64]	21 [15, 27]
Maine	3.3 [1.6, 4.9][#]	42.8 [38.9, 46.6][#]	54 [50.1, 57.9][*]	6.8 [4.5, 9.1][#]	54.8 [50.1, 59.5]	38.5 [33.9, 43][*]	12.7 [7.6, 17.9][#]	64.7 [57.4, 72]	22.6 [16.2, 28.9]
Maryland	4.4 [2.3, 6.6]	45.9 [42, 49.8]	49.7 [45.8, 53.6]	11.8 [7.8, 15.9]	55.9 [50.6, 61.1]	32.3 [27.6, 37]	23 [14.3, 31.8]	59.6 [49.7, 69.5]	17.4 [10.9, 23.9]

Massachusetts	2.9 [1.5, 4.4]#	46.3 [42.7, 49.9]	50.8 [47.3, 54.4]*	10.5 [7.1, 13.8]	55.8 [50.4, 61.1]	33.8 [28.8, 38.8]	20.5 [10.4, 30.6]	62.3 [50.9, 73.7]	17.2 [9.2, 25.1]
Michigan	3.9 [2.4, 5.5]#	46.2 [42.3, 50.1]	49.8 [46, 53.7]	8.2 [5.1, 11.3]#	59.5 [54.1, 64.8]	32.3 [27.3, 37.3]	14.9 [7.8, 21.9]	62.1 [53.3, 71]	23 [15.6, 30.5]
Minnesota	4.9 [2.7, 7]	37.3 [33.9, 40.7]#	57.8 [54.3, 61.4]*	8.5 [5.4, 11.6]	56 [50.4, 61.5]	35.6 [30.3, 40.8]*	24.7 [13.9, 35.5]	62.3 [51.6, 73]	13 [7.9, 18.1]#
Mississippi	6.4 [3.5, 9.3]	48.7 [44.4, 53.1]	44.8 [40.7, 49]	10.2 [7.1, 13.2]	58.4 [53.7, 63.2]	31.4 [27.1, 35.8]	15 [9.1, 20.9]	63.7 [56.5, 70.8]	21.4 [16, 26.8]
Missouri	4.8 [3.1, 6.6]	41.9 [38, 45.8]#	53.3 [49.3, 57.2]*	9.6 [6.4, 12.9]	57.6 [52.7, 62.6]	32.7 [28.2, 37.3]	16.4 [9.6, 23.2]	61.4 [53.2, 69.5]	22.2 [15.7, 28.7]
Montana	3.4 [1.6, 5.1]#	40.2 [36.5, 44]#	56.4 [52.5, 60.2]*	4 [2.5, 5.5]#	57.7 [52.6, 62.8]	38.3 [33.2, 43.3]*	14.9 [9.4, 20.5]	59.9 [52.7, 67.1]	25.2 [19, 31.4]
Nebraska	4.2 [2.5, 5.8]	36.5 [32.7, 40.3]#	59.3 [55.5, 63.2]*	8.3 [5.1, 11.4]	54.5 [49.1, 59.9]	37.2 [32, 42.4]*	12.3 [7, 17.7]#	59.4 [50.7, 68.1]	28.2 [20.5, 36]
Nevada	11.6 [7.9, 15.3]*	53 [48.4, 57.7]*	35.4 [31.1, 39.6]#	15.3 [11.4, 19.1]	62.3 [57.4, 67.3]	22.4 [18.5, 26.3]#	25.5 [17, 34]	62.2 [53.3, 71.1]	12.3 [7.1, 17.5]#
New Hampshire	2.9 [1.7, 4.1]#	43.2 [39.8, 46.7]#	53.9 [50.5, 57.4]*	9.7 [6.8, 12.6]	53.8 [49.1, 58.5]#	36.5 [32, 41]*	16.7 [9, 24.4]	54.2 [45.1, 63.3]	29.2 [20.8, 37.6]
New Jersey	4.4 [2.6, 6.2]	45.3 [41.5, 49.2]	50.3 [46.4, 54.1]	16.2 [10.8, 21.5]	49.6 [44.1, 55.2]#	34.2 [29.2, 39.2]	16.1 [7.2, 25]	59 [46.8, 71.2]	24.9 [13.5, 36.2]
New Mexico	6.2 [3.6, 8.7]	53.6 [48.9, 58.4]*	40.2 [35.6, 44.8]#	9.7 [6.3, 13]	63.7 [58.4, 68.9]	26.7 [22, 31.4]	27.2 [20, 34.4]*	56.1 [48.7, 63.5]	16.8 [11.9, 21.7]
New York	6.7 [4.2, 9.3]	52 [47.9, 56.1]*	41.3 [37.4, 45.3]#	12.7 [8.4, 16.9]	60.1 [54.5, 65.6]	27.3 [22.4, 32.1]	19.7 [9.1, 30.3]	65.9 [54.3, 77.4]	14.5 [7, 21.9]
North Carolina	6.8 [4.2, 9.5]	42.5 [38.3, 46.7]#	50.7 [46.5, 54.9]	11.8 [7.8, 15.7]	57.7 [52.1, 63.3]	30.6 [25.5, 35.6]	22.9 [13.7, 32]	58.3 [48.4, 68.2]	18.8 [12.1, 25.5]
North Dakota	2.1 [1.2, 3]#	40.6 [36.9, 44.3]#	57.3 [53.6, 61]*	5.9 [3.4, 8.4]#	49.3 [43.6, 54.9]#	44.9 [39.2, 50.5]*	11.6 [6.3, 16.9]#	62 [52.8, 71.2]	26.5 [18, 34.9]
Ohio	4.7 [2.8, 6.7]	43.2 [39.3, 47]#	52.1 [48.2, 55.9]*	9.2 [6, 12.3]	55.7 [50.6, 60.8]	35.2 [30.3, 40]*	22.8 [14.8, 30.8]	52.2 [43.8, 60.7]	25 [17.9, 32]
Oklahoma	8.1 [5.3, 11]	43.6 [39.4, 47.8]	48.3 [44.1, 52.5]	11.9 [7.9, 15.9]	63.2 [58.3, 68.1]	24.9 [21, 28.8]#	20.4 [14.4, 26.3]	59.2 [51.9, 66.5]	20.4 [14.4, 26.4]

Oregon	3.7 [2.3, 5.2][#]	49.4 [45.4, 53.3]	46.9 [43, 50.8]	12 [8.4, 15.6]	58.7 [53.7, 63.6]	29.4 [24.9, 33.9]	19.4 [12.8, 26]	66.5 [59.1, 73.9]	14.1 [9.2, 19][#]
Pennsylvania	5.1 [2.9, 7.2]	44.1 [40.2, 48.1]	50.8 [46.9, 54.8][*]	10 [6.4, 13.6]	53.8 [48.5, 59.1]	36.2 [31.2, 41.3][*]	12.1 [5.8, 18.3][#]	67.5 [59.2, 75.8]	20.5 [13.9, 27.1]
Rhode Island	4.7 [2.9, 6.4]	46.9 [42.9, 51]	48.4 [44.4, 52.4]	9.1 [5.5, 12.7]	61.8 [56.4, 67. 2]	29.1 [24.3, 34]	26.5 [18.3, 34.7]	54.5 [45.3, 63.7]	19 [12.3, 25.8]
South Carolina	3.5 [1.9, 5][#]	42.4 [38.5, 46.3][#]	54.1 [50.2, 58.1][*]	10.1 [6.8, 13.5]	57.3 [52.1, 62.5]	32.5 [27.7, 37.4]	17.1 [9.6, 24.6]	60 [51.2, 68.7]	23 [16.1, 29.9]
South Dakota	2.8 [0.9, 4.7][#]	39.7 [35.8, 43.5][#]	57.5 [53.6, 61.5][*]	5.9 [3.4, 8.4][#]	54.9 [49.7, 60]	39.3 [34.2, 44.3][*]	11.8 [6.7, 16.9][#]	62 [54, 69.9]	26.3 [19.3, 33.3]
Tennessee	7.1 [4.5, 9.6]	42.3 [38.3, 46.2][#]	50.7 [46.7, 54.6]	9 [6.2, 11.8]	55.9 [50.8, 61]	35.1 [30.3, 39.9][*]	18.6 [11.5, 25.7]	55.2 [46.7, 63.7]	26.2 [18.8, 33.7]
Texas	8.1 [5.1, 11.1]	47.5 [42.8, 52.2]	44.4 [39.9, 48.9]	16 [11.3, 20.7][*]	58.1 [52.5, 63.7]	25.9 [21.3, 30.5]	14.9 [9.2, 20.7]	59.3 [50.6, 68]	25.8 [17.8, 33.8]
Utah	2.5 [1, 4][#]	35 [31.2, 38.8][#]	62.5 [58.6, 66.3][*]	5.9 [3.9, 8][#]	55.2 [50, 60.4]	38.9 [34, 43.8][*]	20.9 [12.9, 28.8]	56.1 [46.7, 65.4]	23.1 [14.9, 31.3]
Vermont	2.5 [0.7, 4.2][#]	40.8 [37.1, 44.5][#]	56.7 [53, 60.5][*]	6 [3.1, 8.9][#]	56.8 [52, 61.6]	37.2 [32.7, 41.8][*]	15.1 [8.8, 21.5]	59.1 [50.7, 67.5]	25.8 [18.4, 33.2]
Virginia	3.3 [2, 4.6][#]	47.3 [43.5, 51.2]	49.4 [45.5, 53.2]	7.3 [4.7, 9.8][#]	58.5 [53.5, 63.5]	34.3 [29.5, 39.1]	16 [8, 24]	54.6 [44.7, 64.5]	29.4 [20.5, 38.3]
Washington	5.4 [3.1, 7.7]	48.8 [44.7, 52.9]	45.8 [41.8, 49.8]	11.6 [7.7, 15.5]	59.9 [54.5, 65.3]	28.5 [23.8, 33.2]	10.3 [6, 14.6][#]	66.1 [57.6, 74.7]	23.6 [15.5, 31.6]
West Virginia	3.4 [1.9, 4.9][#]	46.3 [42.2, 50.4]	50.3 [46.3, 54.4]	8.7 [6.1, 11.4]	59.4 [54.8, 64]	31.9 [27.7, 36.1]	19.2 [12.6, 25.8]	57.2 [49.3, 65.2]	23.6 [17, 30.2]
Wisconsin	2.6 [1.7, 3.5][#]	44.6 [41, 48.3]	52.9 [49.2, 56.5][*]	8.4 [5.3, 11.5]	54.4 [49.6, 59.2]	37.2 [32.7, 41.7][*]	18.8 [11.3, 26.2]	63.4 [55.2, 71.7]	17.8 [12.4, 23.2]
Wyoming	2.1 [0.9, 3.3][#]	40 [35.9, 44][#]	57.9 [53.9, 62][*]	8.5 [5.4, 11.5]	54.1 [48.9, 59.3]	37.4 [32.5, 42.4][*]	15.6 [9.6, 21.7]	57.5 [49.7, 65.3]	26.9 [19.9, 33.9]

[*]Prevalence significantly higher than national prevalence at 0.05 level of significance;
[#]Prevalence significantly lower than national prevalence at 0.05 level of significance

Table C.7: Across-state prevalence [95% CI] of individual ACEs among school-age children in the US, NSCH 2016-2020 (N=112,178)

State name	Economic	Parental divorce	Parental death	Parental	Witnessing domes	Witnessing neighb	Household	Household substa	Discrimination

	hardship			incarceration	tic violence	orhood violence	mental illness	nce use	due to race/ethnicity
Alabama	20.6 [17.8,23.3]	34.3 [31.2,37.3]	4.7 [3.3,6.2]	9.5 [7.5,11.5]	7.2 [5.4,9]	3.7 [2.6,4.8]	8.1 [6.5,9.6]	10 [8.2,11.8]	6.5 [4.8,8.2]
Alaska	19.5 [16.6,22.4]	32.8 [29.7,35.8]	4.3 [3,5.7]	13.7 [11.4,15.9]	8.6 [6.8,10.4]	8.8 [6.9,10.7]	14.5 [12.3,16.6]	17.5 [15.1,19.9]	5.6 [4.2,7.1]
Arizona	19.3 [16.8,21.9]	31.9 [29,34.9]	3.9 [2.6,5.2]	11.9 [9.6,14.2]	8.1 [6.4,9.9]	6.5 [4.8,8.2]	10.4 [8.5,12.2]	12.4 [10.3,14.5]	5.3 [4,6.6]
Arkansas	22.4 [19.7,25.1]	39 [35.8,42.2]	5.9 [4.3,7.4]	16.6 [14,19.2]	11 [8.9,13.1]	7.5 [5.8,9.3]	11.1 [9.3,13]	16.1 [13.7,18.4]	5.3 [3.8,6.7]
California	15.5 [13,18]	25.2 [22.4,28.1]	2.3 [1.5,3]	6.4 [4.8,8]	5.5 [4,7.1]	4.3 [2.9,5.7]	7 [5.5,8.6]	9.3 [7.4,11.3]	5.3 [4,6.7]
Colorado	18 [15.5,20.5]	30.1 [27.4,32.8]	3.2 [2.2,4.1]	9.9 [7.9,11.8]	6.5 [4.9,8.2]	5.1 [3.7,6.4]	11.9 [10,13.8]	12.8 [10.8,14.8]	5.6 [4.3,7]
Connecticut	17.2 [14.7,19.8]	26.3 [23.5,29.1]	3.6 [2.3,4.9]	6.8 [5,8.6]	4.8 [3.5,6.2]	4 [2.9,5.2]	8.7 [6.9,10.5]	8.2 [6.5,9.9]	4.3 [3,5.6]
Delaware	18.2 [15.5,20.8]	31.4 [28.4,34.4]	4.5 [3.1,5.9]	10.3 [8.2,12.5]	8 [6.1,10]	7 [5.1,8.8]	10 [8.1,11.9]	10.8 [8.8,12.8]	6.4 [4.8,8]
District of Columbia	17.6 [14.2,21]	29.6 [26,33.3]	7.6 [5.3,10]	11.1 [8.3,14]	9 [6.4,11.6]	14.4 [11.2,17.6]	7.9 [5.5,10.2]	9.6 [6.9,12.3]	10.5 [7.8,13.1]
Florida	20.6 [18,23.1]	33.5 [30.5,36.4]	5 [3.7,6.3]	9.8 [7.7,12]	7.5 [5.6,9.3]	5.8 [4.2,7.4]	9 [7.3,10.8]	9.7 [7.9,11.5]	5.1 [3.8,6.4]
Georgia	19.4 [16.8,22]	30.6 [27.7,33.5]	5 [3.6,6.4]	11.6 [9.5,13.8]	6.3 [4.7,7.9]	5.6 [4,7.3]	7.9 [6.3,9.5]	10.3 [8.4,12.3]	7.8 [5.9,9.6]
Hawaii	17 [14.6,19.4]	25.1 [22.6,27.6]	4.4 [3.1,5.8]	5.5 [4.1,6.9]	8.4 [6.7,10]	7.1 [5.4,8.7]	6.7 [5.3,8.1]	9.9 [8.2,11.6]	4.1 [3,5.2]
Idaho	20.9 [18.4,23.4]	28.9 [26.2,31.5]	3.2 [2.1,4.3]	11.1 [9.1,13.1]	7.8 [6,9.5]	4.1 [3,5.2]	16.2 [14,18.4]	13.5 [11.5,15.5]	2.8 [1.8,3.8]
Illinois	19.4 [16.8,22]	23.7 [21,26.4]	3.5 [2.3,4.7]	8.1 [6.1,10]	5.7 [4.2,7.2]	5.2 [3.6,6.7]	8.7 [6.8,10.5]	8.7 [7,10.3]	6.2 [4.5,7.9]
Indiana	17.9 [15.4,20.3]	31 [28.3,33.6]	5.6 [3.9,7.3]	10.9 [8.9,12.9]	8.1 [6.5,9.7]	5.9 [4.4,7.4]	11.2 [9.4,12.9]	11.9 [10,13.9]	4.8 [3.4,6.2]

Iowa	16.8 [14.5,19.2]	28.4 [25.8,31]	3.6 [2.6,4.7]	8 [6.4,9.6]	7 [5.4,8.6]	5.7 [4,7.5]	11 [9.2,12.8]	12.5 [10.5,14.4]	3.3 [2.4,4.3]
Kansas	19.2 [16.6,21.7]	30.4 [27.6,33.3]	3.5 [2.5,4.6]	10.2 [8.2,12.1]	7.7 [5.8,9.5]	5.5 [3.9,7.1]	10.4 [8.5,12.2]	11.9 [10,13.8]	3.8 [2.5,5.1]
Kentucky	20.9 [18.3,23.6]	35.1 [32.2,37.9]	4.5 [3.4,5.7]	13.4 [11.3,15.6]	8.1 [6.3,9.9]	5.8 [4.3,7.4]	11.2 [9.2,13.2]	12.5 [10.5,14.5]	4 [2.6,5.4]
Louisiana	20.5 [18,23.1]	35.7 [32.7,38.7]	5.3 [3.8,6.8]	12.1 [9.9,14.3]	7.3 [5.8,8.9]	4.7 [3.3,6]	9.1 [7.3,10.9]	10.5 [8.8,12.3]	4.5 [3.5,5.6]
Maine	21.2 [18.8,23.6]	35.3 [32.5,38.1]	3.3 [2.3,4.3]	8.1 [6.4,9.8]	8 [6.3,9.6]	5.4 [4.1,6.8]	15.3 [13.2,17.3]	13.9 [12,15.8]	2.1 [1.2,2.9]
Maryland	17.4 [14.8,20]	27.2 [24.4,30.1]	3 [2,4]	6 [4.4,7.6]	5.3 [3.9,6.6]	4.8 [3.5,6.1]	8 [6.3,9.6]	7.8 [6.3,9.3]	9 [6.9,11]
Massachusetts	16.1 [13.6,18.6]	23.6 [20.9,26.2]	3.5 [2.5,4.6]	3.9 [2.4,5.3]	4.7 [3.2,6.2]	4.8 [3.1,6.5]	9.4 [7.6,11.1]	8 [6.3,9.6]	3.6 [2.5,4.6]
Michigan	18.7 [16.1,21.4]	30.4 [27.5,33.4]	4.8 [3.3,6.3]	9.6 [7.6,11.7]	6.5 [4.9,8.1]	6.6 [4.8,8.4]	9.8 [8.1,11.5]	10.7 [8.8,12.6]	6.7 [5,8.4]
Minnesota	14.7 [12.4,17.1]	25.1 [22.4,27.9]	2.7 [1.6,3.8]	7 [5,8.9]	5.5 [3.9,7.1]	5.5 [3.9,7.2]	10.7 [8.8,12.6]	10.1 [8.4,11.7]	5.4 [3.8,7.1]
Mississippi	23.1 [20.4,25.7]	36.1 [33.2,39]	6.7 [5.1,8.3]	12.9 [10.6,15.1]	10.2 [8.2,12.1]	5.6 [4.2,7]	9.9 [8.1,11.7]	11.7 [9.8,13.6]	7 [5.5,8.5]
Missouri	21.4 [18.7,24.1]	31.3 [28.5,34.1]	3.3 [2.1,4.6]	11.9 [9.6,14.3]	7.9 [6.2,9.7]	5.9 [4.2,7.6]	10.9 [9.2,12.5]	12.5 [10.5,14.5]	4.2 [3,5.5]
Montana	22.6 [19.9,25.3]	35.1 [32.3,38]	3.6 [2.5,4.8]	12.1 [9.9,14.3]	10.5 [8.5,12.6]	7.2 [5.5,8.8]	16.4 [14.3,18.6]	17.6 [15.2,20]	4.3 [2.9,5.7]
Nebraska	17.7 [15.1,20.2]	27.1 [24.4,29.8]	3.1 [1.9,4.2]	9.1 [7.2,11]	6.6 [5.1,8.1]	3.9 [2.7,5.1]	9.4 [7.8,11]	10.6 [8.7,12.5]	4.4 [3.1,5.7]
Nevada	20.3 [17.4,23.2]	32.7 [29.6,35.7]	3.9 [2.7,5.1]	10 [7.7,12.2]	7.3 [5.5,9]	6.8 [5,8.5]	8.7 [6.9,10.5]	12.2 [10,14.3]	6.8 [5.2,8.3]
New Hampshire	15.5 [13.4,17.5]	26.7 [24.3,29.1]	4.2 [3.1,5.3]	4.8 [3.5,6]	4.5 [3.3,5.7]	2.8 [1.9,3.7]	11.6 [9.9,13.4]	9.7 [8.1,11.3]	1.6 [1,2.1]
New Jersey	18.4 [15.8,21]	21 [18.5,23.6]	2.7 [1.7,3.8]	3.6 [2.4,4.8]	2.6 [1.6,3.5]	3.3 [1.9,4.6]	5.3 [4.2,6.5]	6 [4.6,7.4]	5.4 [3.9,6.9]

New Mexico	20 [17.3,22.7]	35.9 [32.8,39.1]	4.6 [3.2,6.1]	13 [10.7,15.3]	11.5 [9.3,13.7]	8 [6.2,9.7]	10.8 [9,12.6]	14.5 [12.3,16.7]	8 [6.2,9.9]
New York	19.3 [16.5,22]	24.9 [22,27.7]	3.7 [2.4,5]	3.5 [2.3,4.7]	3.7 [2.6,4.7]	5 [3.2,6.7]	7 [5.5,8.6]	5.6 [4.2,7]	5.8 [3.9,7.6]
North Carolina	19.1 [16.4,21.8]	28.9 [25.8,31.9]	3 [2,4]	7.5 [5.7,9.4]	7.1 [5.1,9]	4.1 [2.7,5.4]	8.9 [7.1,10.6]	8.9 [7.1,10.8]	7.8 [5.8,9.8]
North Dakota	16.8 [14.3,19.3]	25.5 [22.9,28.2]	3 [2,4.1]	9.7 [7.7,11.8]	6.2 [4.6,7.8]	5.2 [3.6,6.8]	11.9 [9.8,14.1]	12.1 [9.9,14.2]	2.3 [1.4,3.2]
Ohio	20.8 [18.3,23.4]	31 [28.3,33.8]	5.4 [3.9,6.9]	12.9 [10.5,15.3]	8.7 [6.8,10.5]	6.7 [4.9,8.4]	11.4 [9.5,13.2]	11.3 [9.5,13.2]	5.2 [3.7,6.6]
Oklahoma	21.4 [18.9,23.9]	35.8 [32.9,38.7]	5.6 [4.3,7]	14.4 [12.1,16.7]	8.6 [6.9,10.4]	6.4 [4.8,8]	11.7 [9.8,13.6]	14 [11.9,16.1]	5.6 [4.1,7]
Oregon	21.5 [18.9,24.1]	29.8 [27.1,32.5]	3 [2.1,4]	7.9 [6.4,9.5]	7.7 [6.1,9.3]	5.4 [4,6.9]	14.1 [12,16.2]	15.1 [13,17.3]	5.9 [4.4,7.4]
Pennsylvania	16.6 [14.2,19]	24.4 [21.9,27]	5 [3.6,6.3]	9 [7,10.9]	6.2 [4.6,7.8]	6.6 [4.8,8.4]	8.3 [6.8,9.8]	8.4 [6.9,10]	4.4 [3,5.8]
Rhode Island	20.3 [17.5,23]	29.4 [26.5,32.3]	4.5 [3,5.9]	5.4 [3.9,6.9]	6.2 [4.7,7.7]	5.8 [4.2,7.4]	10.3 [8.5,12.1]	10.1 [8.2,11.9]	5 [3.5,6.5]
South Carolina	19.9 [17.2,22.5]	33.5 [30.5,36.5]	6 [4.5,7.6]	11.7 [9.5,14]	6.7 [4.9,8.5]	5.2 [3.7,6.7]	9 [7.3,10.7]	9.1 [7.4,10.7]	6 [4.5,7.6]
South Dakota	20.5 [17.9,23]	30.7 [27.9,33.6]	2.6 [1.5,3.7]	11.1 [9,13.1]	8 [6.1,9.8]	5.5 [4.1,7]	10.8 [8.9,12.7]	15.4 [13.1,17.6]	5.7 [4.1,7.3]
Tennessee	20.6 [17.9,23.3]	32.5 [29.6,35.4]	5.7 [4.1,7.4]	13.6 [11.3,15.9]	8.9 [6.8,11]	5 [3.6,6.5]	10.1 [8.4,11.9]	12.1 [10.1,14.1]	5.1 [3.6,6.6]
Texas	19.9 [17.1,22.8]	29.5 [26.5,32.4]	4.6 [3,6.1]	8.5 [6.8,10.3]	7.4 [5.7,9.1]	5 [3.5,6.4]	9.3 [7.4,11.2]	11.5 [9.4,13.7]	6.6 [5,8.1]
Utah	15.9 [13.7,18.1]	22.3 [19.8,24.9]	2.6 [1.6,3.7]	6.6 [4.9,8.4]	5.8 [4.3,7.3]	5.1 [3.4,6.8]	14.8 [12.8,16.7]	9.1 [7.3,10.9]	3.7 [2.5,5]
Vermont	18.6 [16.3,20.9]	32.7 [29.9,35.4]	2 [1.3,2.7]	7.4 [5.6,9.2]	7 [5.3,8.8]	5 [3.6,6.4]	12.8 [10.9,14.8]	16.6 [14.3,18.9]	3 [1.9,4]
Virginia	17.4 [14.9,20]	28.3 [25.5,31.1]	4.8 [3.3,6.3]	8.1 [6.2,10]	5.2 [3.8,6.5]	3.2 [2.1,4.3]	8.7 [7.2,10.3]	8.7 [7.1,10.4]	5.1 [3.8,6.4]

Washington	17.2 [14.6,19.7]	26.4 [23.7,29.1]	1.9 [1,2.7]	6.5 [4.9,8]	4.7 [3.4,5.9]	5.1 [3.7,6.6]	11.4 [9.5,13.4]	11.6 [9.7,13.5]	6.2 [4.5,8]
West Virginia	23.1 [20.4,25.9]	41 [38,43.9]	4.6 [3.4,5.7]	12.7 [10.5,14.9]	9.5 [7.7,11.4]	7.2 [5.3,9.1]	12.3 [10.3,14.2]	17 [14.5,19.4]	3 [2,4]
Wisconsin	16.8 [14.6,19]	32.3 [29.6,35.1]	3.9 [2.7,5.2]	10.8 [8.6,12.9]	7.9 [6.1,9.7]	5.3 [3.8,6.8]	11.5 [9.6,13.3]	11.7 [9.8,13.7]	5.6 [3.9,7.3]
Wyoming	21.3 [18.6,23.9]	33.3 [30.4,36.2]	3.6 [2.5,4.8]	12.5 [10.4,14.7]	11.1 [9,13.2]	6.7 [5,8.4]	14.9 [12.8,17.1]	16.7 [14.4,19.1]	3.2 [2.1,4.3]

Table C.8: Across-state prevalence [95% CI] of individual PPFs among school-age children in the US, NSCH 2016-2020 (N=112,178)

State name	Community volunteer	After school activities	Mentor for advice	Family resilience	Connected caregiver	Supportive neighborhood	Safe neighborhood
Alabama	48 [44.9,51]	76.2 [73.4,79]	89.3 [86.9,91.6]	83.1 [80.7,85.5]	67.5 [64.6,70.4]	58.1 [55.1,61.2]	66.7 [63.7,69.7]
Alaska	41.8 [38.7,44.9]	78.5 [75.6,81.5]	91.9 [89.9,93.8]	81 [78.3,83.7]	63.7 [60.6,66.7]	55 [51.8,58.2]	61.1 [58,64.3]
Arizona	39.6 [36.6,42.6]	77.2 [74.4,80.1]	86.9 [84.6,89.3]	81.3 [78.9,83.6]	65.5 [62.6,68.4]	50.9 [47.8,54.1]	61.2 [58.1,64.3]
Arkansas	44.9 [41.8,48.1]	74 [70.9,77.1]	92.2 [90.5,93.9]	79.4 [76.9,82]	65.2 [62.1,68.4]	55.9 [52.7,59.1]	65.5 [62.4,68.5]
California	36.3 [33.4,39.3]	75.5 [72.6,78.5]	84.4 [81.9,86.8]	80.3 [77.8,82.8]	64.1 [61,67.2]	51.1 [48,54.3]	59.3 [56.1,62.5]
Colorado	44.2 [41.3,47]	81.9 [79.3,84.5]	89.8 [87.8,91.8]	81.6 [79.3,83.9]	66.6 [63.9,69.4]	57.5 [54.6,60.4]	67.1 [64.3,69.9]
Connecticut	44 [41.1,46.9]	86.7 [84.5,89]	89.7 [87.6,91.7]	79.5 [77.1,82]	64.6 [61.7,67.4]	58.2 [55.2,61.3]	69.9 [66.9,72.9]
Delaware	42.5 [39.5,45.5]	79.4 [76.7,82.2]	87.1 [84.7,89.5]	78.1 [75.4,80.8]	64.1 [61,67.1]	51.6 [48.5,54.7]	62.4 [59.3,65.4]
District of Columbia	47 [43.1,51]	81 [77.5,84.5]	89.4 [86.9,91.8]	78.3 [74.9,81.7]	69.4 [65.8,73]	47 [43,50.9]	46 [42,49.9]
Florida	40.4 [37.4,43.3]	76 [73.3,78.7]	83.4 [81,85.8]	84 [81.7,86.3]	71.5 [68.8,74.3]	50.7 [47.7,53.8]	63.1 [60.1,66.2]

Georgia	44.9 [41.9,47.9]	74 [71,77]	88.2 [86.1,90.2]	81.8 [79.3,84.4]	68.5 [65.6,71.4]	51 [47.9,54]	64.9 [61.9,67.8]
Hawaii	48 [45,50.9]	83.7 [81.4,86]	89.8 [88.1,91.5]	81.8 [79.7,83.9]	62.9 [60.1,65.7]	52.5 [49.6,55.5]	58.3 [55.4,61.2]
Idaho	48.7 [45.8,51.5]	79.6 [77.1,82.2]	92.7 [90.8,94.6]	82.4 [80,84.7]	65.4 [62.7,68.2]	65 [62.2,67.9]	72.7 [70.1,75.4]
Illinois	41.6 [38.7,44.6]	83 [80.5,85.5]	90 [88,92]	80.5 [78.1,82.9]	63.3 [60.3,66.3]	59.8 [56.8,62.9]	65.4 [62.3,68.5]
Indiana	41.6 [38.9,44.4]	75.8 [73,78.5]	92.7 [90.9,94.5]	79.8 [77.4,82.3]	64.1 [61.3,67]	62 [59.2,64.9]	70.2 [67.5,72.9]
Iowa	48.6 [45.8,51.4]	83.2 [80.8,85.7]	94.4 [92.9,95.9]	80.3 [78.1,82.5]	62.3 [59.5,65]	66.1 [63.4,68.9]	74.9 [72.2,77.5]
Kansas	47.5 [44.6,50.4]	80.4 [77.6,83.1]	92.3 [90.5,94.1]	81.8 [79.3,84.2]	63 [60.1,65.8]	61.5 [58.6,64.4]	74.1 [71.4,76.8]
Kentucky	46 [43.1,49]	74.7 [72,77.4]	94.1 [92.7,95.6]	82.2 [79.9,84.6]	67.7 [64.9,70.4]	59.8 [56.8,62.8]	68.6 [65.8,71.4]
Louisiana	40.6 [37.6,43.5]	74.2 [71.3,77]	91.1 [89.3,92.8]	80.7 [78.2,83.2]	67.8 [65,70.6]	59.1 [56.1,62.1]	66 [63.1,69]
Maine	42.1 [39.3,44.8]	85.7 [83.6,87.8]	94.6 [93.1,96]	81.8 [79.7,84]	65.7 [63.1,68.4]	60.8 [58.1,63.6]	73 [70.4,75.7]
Maryland	47.6 [44.6,50.6]	81.9 [79,84.7]	89.7 [87.7,91.6]	79.4 [76.9,81.9]	65.8 [62.9,68.7]	54 [50.9,57.1]	66 [63,69]
Massachusetts	42 [39.2,44.8]	86.2 [83.8,88.5]	89.3 [87.3,91.2]	79.6 [77.1,82]	63.7 [60.8,66.5]	58.8 [55.8,61.8]	74.2 [71.4,77]
Michigan	43.3 [40.4,46.3]	80.1 [77.4,82.8]	91.9 [90.1,93.7]	80.8 [78.4,83.3]	67.8 [65,70.5]	59.8 [56.8,62.7]	69.3 [66.4,72.2]
Minnesota	47.2 [44.3,50.1]	84.1 [81.5,86.6]	91.9 [90.1,93.7]	81.7 [79.5,84]	63.5 [60.6,66.3]	65.2 [62.3,68.2]	74.8 [72,77.5]
Mississippi	46.3 [43.3,49.2]	70.2 [67.3,73.1]	90.8 [88.9,92.7]	78 [75.3,80.6]	68 [65.3,70.8]	55.6 [52.6,58.6]	63.4 [60.5,66.4]
Missouri	45.7 [42.8,48.6]	81.3 [78.6,84]	92.9 [91.3,94.5]	79.1 [76.7,81.6]	66.1 [63.3,68.8]	60.2 [57.3,63.1]	67.5 [64.7,70.4]

Montana	47.9 [44.9,50.8]	84.8 [82.7,86.9]	93.8 [92.2,95.4]	84.4 [82.3,86.5]	64.4 [61.5,67.2]	60.9 [58,63.9]	69.2 [66.4,72]
Nebraska	48.9 [45.9,51.9]	85.7 [83.4,88.1]	93.2 [91.3,95.2]	83.7 [81.7,85.7]	64.2 [61.3,67.1]	66.5 [63.6,69.3]	74.8 [72.1,77.4]
Nevada	34.9 [31.9,37.9]	72.2 [69,75.4]	82.5 [79.9,85.1]	81.1 [78.5,83.7]	64.4 [61.2,67.6]	42.4 [39.2,45.6]	55.9 [52.7,59.2]
New Hampshire	41.5 [38.9,44.1]	88.1 [86.3,89.9]	93.6 [92.2,95.1]	80.6 [78.4,82.8]	65.8 [63.2,68.3]	62.7 [60.1,65.3]	76.4 [74.1,78.7]
New Jersey	45.4 [42.4,48.4]	84 [81.4,86.6]	88.4 [86.3,90.4]	79.7 [77,82.4]	66.7 [63.8,69.6]	61.7 [58.7,64.8]	69.6 [66.6,72.6]
New Mexico	38.7 [35.6,41.8]	74.2 [71.1,77.3]	86.3 [83.7,88.9]	81.1 [78.4,83.7]	67.2 [64.1,70.3]	47.4 [44.2,50.7]	57.3 [54.1,60.6]
New York	41.5 [38.3,44.6]	82.4 [79.7,85.2]	88.6 [86.3,90.9]	79.6 [76.8,82.3]	66.1 [63.1,69.2]	52.5 [49.3,55.8]	57.3 [54.1,60.6]
North Carolina	47.9 [44.7,51.1]	76.9 [73.9,79.8]	86.8 [84.3,89.4]	81.8 [79.3,84.3]	62.4 [59.2,65.6]	56.6 [53.4,59.9]	68.1 [64.9,71.3]
North Dakota	50.3 [47.3,53.3]	86.1 [83.8,88.4]	94 [92.3,95.6]	81 [78.5,83.4]	66.1 [63.3,68.8]	69.5 [66.8,72.3]	77.2 [74.7,79.7]
Ohio	45.8 [42.9,48.7]	79.4 [76.7,82.1]	93 [91.3,94.6]	79.9 [77.4,82.4]	64.9 [62.1,67.7]	61.6 [58.7,64.5]	66.2 [63.4,69.1]
Oklahoma	41.6 [38.7,44.5]	75.4 [72.7,78.1]	91.6 [89.7,93.5]	77.9 [75.3,80.5]	60.6 [57.7,63.6]	53 [50,56]	63.3 [60.3,66.2]
Oregon	41.5 [38.7,44.3]	79.7 [77.1,82.3]	90.9 [89.1,92.6]	80 [77.5,82.4]	64 [61.2,66.8]	52.1 [49.1,55]	65.1 [62.4,67.9]
Pennsylvania	41.5 [38.6,44.3]	79.4 [76.4,82.3]	93.1 [91.6,94.6]	82.3 [80,84.7]	67 [64.2,69.7]	59.3 [56.3,62.3]	69.4 [66.4,72.3]
Rhode Island	40.5 [37.5,43.5]	82.3 [79.7,85]	91 [89,92.9]	81.3 [78.9,83.7]	63.9 [60.8,66.9]	56.3 [53.2,59.4]	64 [60.8,67.1]
South Carolina	46.5 [43.5,49.5]	79 [76.4,81.7]	92 [90.2,93.8]	83.2 [80.8,85.6]	68 [65.2,70.8]	57.1 [54,60.1]	68.3 [65.5,71.1]
South Dakota	51 [48.1,54]	82.6 [80.1,85.1]	93.4 [91.7,95.2]	79.4 [77,81.8]	65.2 [62.3,68]	64.6 [61.6,67.5]	75.2 [72.5,77.9]

Tennessee	43.3 [40.4,46.2]	76.7 [73.8,79.5]	89.8 [87.7,92]	81.5 [79,84.1]	70.1 [67.4,72.7]	57.9 [54.9,60.9]	68.6 [65.7,71.4]
Texas	38.9 [35.9,42]	76.5 [73.4,79.6]	84.8 [82.2,87.5]	81.5 [78.9,84]	68.8 [65.7,71.8]	52.6 [49.3,55.9]	62.8 [59.6,66.1]
Utah	55.8 [52.9,58.8]	82.8 [80.4,85.3]	92.9 [91.1,94.7]	82.7 [80.5,84.9]	64.3 [61.5,67.1]	72.3 [69.5,75.1]	72.9 [70.1,75.6]
Vermont	41.3 [38.6,44.1]	86.7 [84.6,88.9]	95.8 [94.7,96.9]	81 [78.6,83.3]	66.7 [64,69.4]	64.5 [61.8,67.3]	74.9 [72.2,77.5]
Virginia	46.9 [43.9,49.8]	82.9 [80.4,85.4]	90.4 [88.6,92.3]	82.1 [79.9,84.3]	66.5 [63.8,69.3]	61.3 [58.4,64.2]	73.3 [70.7,75.9]
Washington	41.5 [38.5,44.5]	83.2 [80.5,85.9]	90.2 [88,92.3]	81.7 [79.3,84]	64.4 [61.5,67.4]	55.9 [52.8,59]	61.2 [58,64.3]
West Virginia	44.4 [41.6,47.3]	75.2 [72.5,77.9]	92.2 [90.5,93.9]	81.2 [78.8,83.6]	65.7 [62.9,68.4]	60.2 [57.3,63.1]	69 [66.3,71.8]
Wisconsin	45.1 [42.4,47.9]	84.1 [81.8,86.4]	94.7 [93.6,95.8]	79.7 [77.3,82.1]	62.1 [59.3,64.8]	62.5 [59.7,65.2]	71.5 [68.8,74.1]
Wyoming	44.4 [41.4,47.3]	84.9 [82.5,87.2]	93.3 [91.6,95]	83.1 [80.9,85.3]	64.7 [61.8,67.6]	62.5 [59.6,65.4]	71.3 [68.6,74.1]

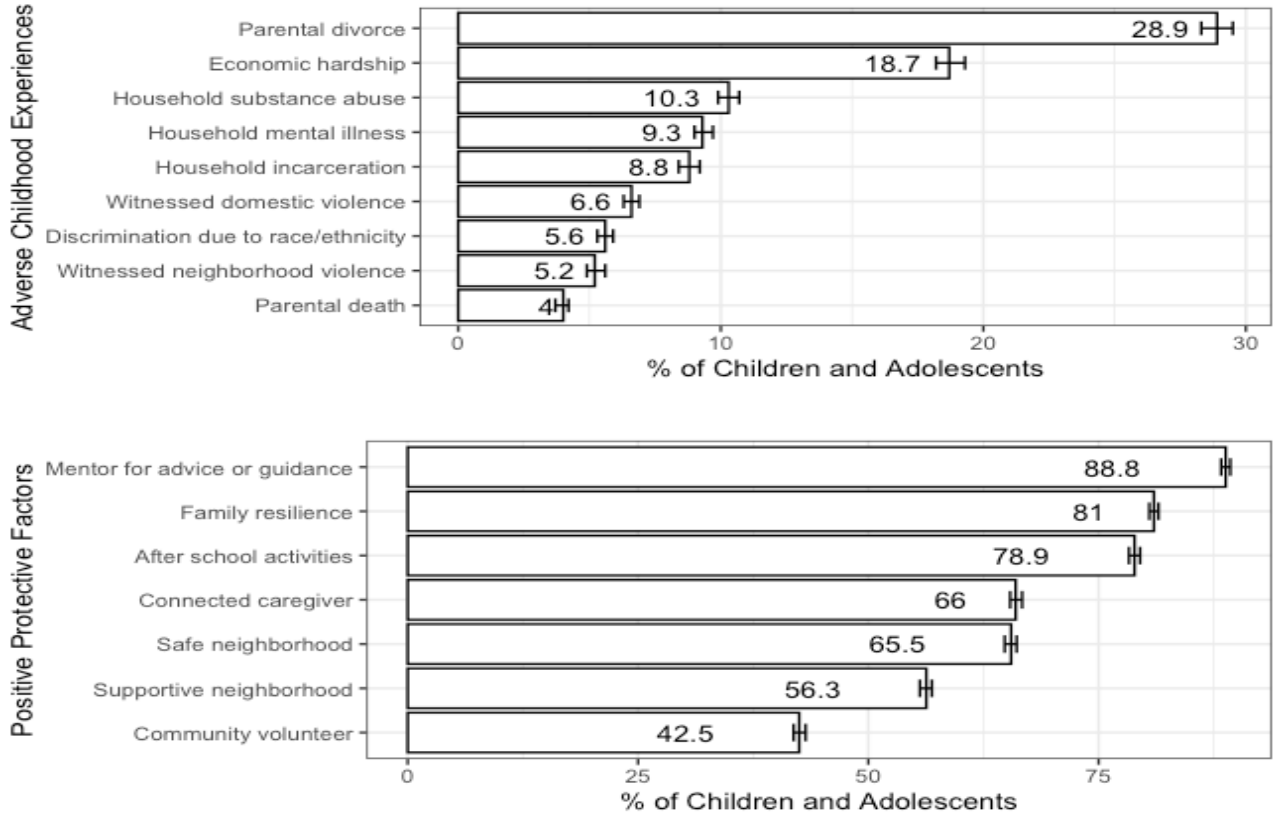

Figure C.1: National prevalence of individual ACEs and PPFs assessed in the study

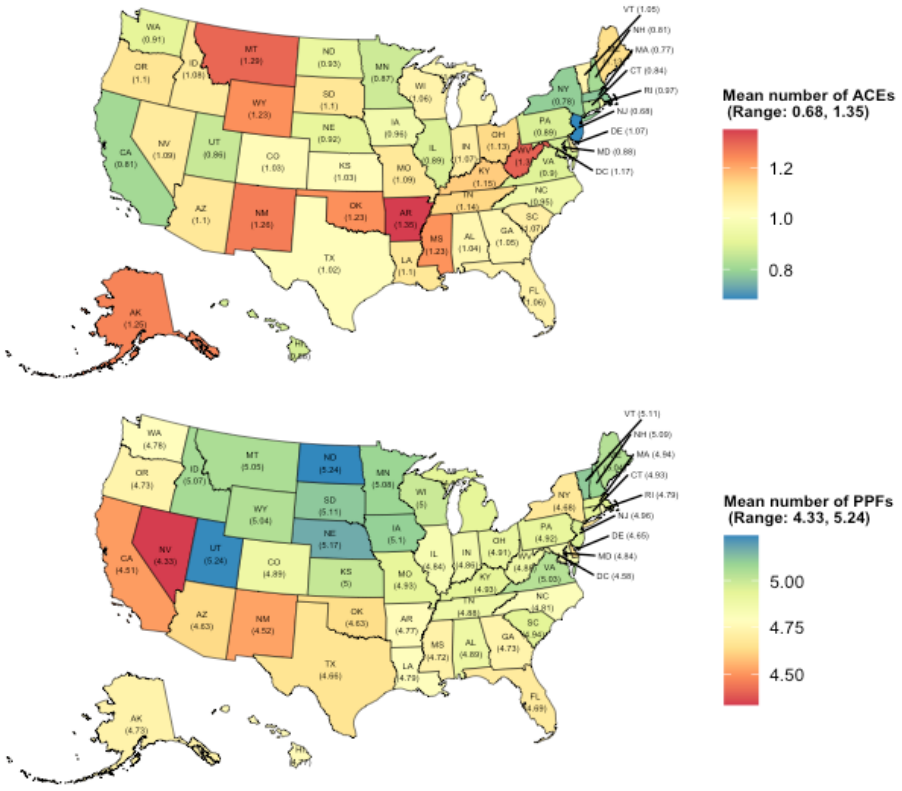

Figure C.2: Across-state distribution of mean number of ACEs and PPFs assessed in the study