Pathways between childhood/adolescent adversity, adolescent socioeconomic status, and long-term cardiovascular disease risk in young adulthood

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ABSTRACT

Objective: The current study investigated mediators between childhood/adolescent adversities (e.g., dating violence, maltreatment, homelessness, and parental death), low socioeconomic status (SES) during adolescence, and cardiovascular disease (CVD) risk in young adulthood. The purpose of these analyses was to understand whether SES during adolescence and childhood/adolescent adversities affect CVD risk through similar pathways, including maternal relationship quality, health behaviors, financial stress, medical/dental care, educational attainment, sleep problems, and depressive symptoms.
Methods: Using the National Longitudinal Study of Adolescent to Adult Health ($N = 14,493$), which has followed US adolescents (Wave 1; $M = 15.9$ years) through early adulthood (Wave 4; $M = 28.9$ years), associations were examined between childhood/adolescent adversity and SES to 30-year CVD risk in young adulthood. The outcome was a Framingham-based prediction model of CVD risk that included age, sex, body mass index, smoking, systolic blood pressure, diabetes, and antihypertensive medication use at Wave 4. Path analysis was used to examine paths through the adolescent maternal relationship to young adult mediators of CVD risk.
Results: Childhood/adolescent adversity significantly predicted greater adult CVD risk through the following pathways: maternal relationship, health behaviors, financial stress, lack of medical/dental care, and educational attainment; but not through depressive symptoms or sleep problems. Lower SES during adolescence significantly predicted greater adult CVD risk through the following pathways: health behaviors, financial stress, lack of medical/dental care, and educational attainment, but not maternal relationship, depressive symptoms, or sleep problems.
Conclusions: Childhood/adolescent adversities and SES affected CVD risk in young adulthood through both similar and unique pathways that may inform interventions.

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1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States; it is responsible for approximately 2200 deaths each day, with 30.8% of deaths in the US in 2013 attributable to CVD

(Hoyert and Xu, 2012; Mozaffarian et al., 2016). By the year 2030, 40.5% of Americans are projected to suffer from some form of CVD (Heidenreich et al., 2011). It is vital to understand the etiology of CVD in order to prevent the disease and to intervene as early as possible. A growing body of research targets childhood as an early period of vulnerability to insults. This period of vulnerability to insults may elevate risks of CVD in adulthood. In addition to factors such as childhood obesity and the establishment of unfavorable health behaviors (e.g., poor diet and inactivity), psychosocial factors have been shown to increase risk for CVD in adulthood (Miller et al., 2011a; Steptoe and Kivimäki, 2013; Taylor, 2010). Specifically, childhood adversities — such as maltreatment, neglect, and family

violence — have been associated with the development of CVD later in life (Miller et al., 2011a). Those who experienced family violence, maltreatment, or other types of adversity during childhood show a greatly increased risk of CVD and early mortality compared to those with no reports of these early adversities (Anda et al., 2009; Felitti et al., 1998). There is evidence which demonstrates that the effects of childhood adversities are additive. For example, the risk of heart disease increased over twofold between those with 3 or more reported adversities and those who reported none, with a dose-response association between the number of childhood adversities and personal heart disease hazard ratio, which estimates the risk of heart disease as a function of childhood adversities (Scott et al., 2011).

A large body of evidence also implicates poverty as a strong predictor of later CVD. Lower childhood SES has consistently been associated with higher CVD risk in both men and women, even after controlling for adult socioeconomic status (SES; Cohen et al., 2010). For example, among a group of affluent physicians who had graduated from medical school at Johns Hopkins, those who experienced low SES in childhood had a 2.4 times higher likelihood of having heart disease at age 50 (Kittleson et al., 2006). Research has shown that the more years spent in a low SES environment during childhood, the greater risk for CVD during adulthood (Pollitt et al., 2005). Additionally, increases across the SES gradient from low-to-high SES have been related to lower physiological risk (Cohen et al., 2010). Unsurprisingly, individuals from low SES backgrounds are more likely to experience childhood adversity (Cohen et al., 2010), which may have both independent and joint impacts on risk for health problems such as CVD. Current hypotheses suggest that both SES and adversities may affect health by increasing life stress. Thus, these results suggest that childhood stress derived from both adversities and low SES may have independent impacts on cardiac health that are not due to the adult environment (Kittleson et al., 2006; Miller et al., 2011a).

There is a great need for research on the mechanisms by which early poverty and adverse experiences affect later CVD risk. Possible mechanisms linking adversities to later CVD include neurobiological, psychological, social, behavioral, and environmental mechanisms. A feature central to many of these pathways may be the adolescent maternal relationship, a potentially important social mediator of both the adversity-CVD risk and SES-CVD risk associations. Greater parental warmth during childhood has been associated with decreased allostatic load (assessed by 18 measures of cardiovascular, anthropometric, hormonal, and inflammatory function) during adulthood (Carroll et al., 2013). In previous analyses with Add Health data, our group has demonstrated a main effect of a more positive adolescent maternal relationship on lower CVD risk in young adulthood (Doom et al., 2016). A supportive parental relationship may influence physiological responses to stressors as well as enhance effective coping strategies, which could ultimately impact cardiovascular health through biological and behavioral pathways. Living in a low SES environment or experiencing multiple adversities may increase the stress faced by families, causing parental warmth to decrease. Factors that contribute to this deteriorating parental relationship may include parental depression and increased parental stress due to adverse circumstances (Conger et al., 1994; McLoyd, 1990; McLoyd et al., 1994). Harsh environments have been shown to affect parenting behaviors, which may consequently influence the health and functioning of children and adolescents (Repetti et al., 2002). Lower SES households have been reported to have more authoritarian parenting behaviors, including more neglectful parenting (Dornbusch et al., 1987; Glasgow et al., 1997). This finding could have been due to poorer and riskier living conditions characteristic of lower SES families or due to adverse events faced by the households investigated.

Adversity experienced by the family during childhood and adolescence could disrupt attachment relationships by altering parenting behaviors and threatening the child's belief that the parent is a source of security and a safe base for exploration (de Wolff and van Ijzendoorn, 1997; Repetti et al., 2002). Likewise, ongoing stress experienced solely by the adolescent, derived from situations like intimate partner violence, has been shown to negatively affect social functioning and trust in others. The persistent stress caused by possible adolescent stressors could also subsequently affect parental relationships (Bonomi et al., 2006).

The current analyses sought to (1) examine SES during adolescence and childhood/adolescent adversity as separate, potentially related risk factors for young adult CVD risk and (2) to test several plausible pathways between SES and adversity and later CVD risk. Specifically, these pathways investigated whether SES during adolescence and childhood/adolescent adversity impacted the maternal relationship during adolescence, and whether the adolescent maternal relationship affected young adult mediators of health behaviors, health care utilization, depressive symptoms, financial stress, sleep problems, and educational attainment. These mediators were tested in the same model in order to examine whether they then increased long-term CVD risk in young adulthood. We hypothesized that both higher levels of adversity and lower SES would be associated with a poorer maternal relationship in adolescence, which would then be associated with poorer health behaviors and sleep, more financial stress, less health care utilization, greater depressive symptoms, and lower educational attainment. We predicted that these mediators would then predict greater CVD risk in young adulthood.

Although associations between childhood/adolescent adversity, SES, and CVD have been established, most prior research has relied on retrospective reports of SES and adversities. In addition, most adversity measures have assessed a narrow range of adverse experiences. The present study examined SES and a broad range of childhood and adolescence adversities in relation to predicted long-term CVD risk in young adulthood, in an effort to reduce bias from retrospective reports. These associations were assessed using the National Longitudinal Study of Adolescent to Adult Health (Add Health), which has followed a large cohort of adolescents through early adulthood. Add Health assessed important childhood/adolescent adversities, including adolescent dating violence, childhood maltreatment, and parental death, SES during adolescence, and CVD risk factors in adulthood, which make these analyses possible. The Add Health study also provided a comprehensive account of adversities and SES and assessed biomarkers and behaviors that permitted calculation of a 30-year CVD risk score using a Framingham-based prediction model. The Framingham-based prediction model was created using data from the Framingham Heart Study to identify risk factors for developing CVD (Pencina et al., 2009). The model uses data from an individual to predict the risk that he or she will have a CVD event in the next 30 years. These long-term prediction models are especially useful for studies of young adults, as they likely will not develop CVD for many years but may have an underlying risk. In this study, the use of a Framingham-based CVD risk score, comprehensive adversity assessment, and examination of a variety of potential young adult mediators provide unique contributions to the literature on psychosocial stress and CVD.

2. Methods

Add Health data was used to conduct the analyses (Harris et al., 2009). Prior to each wave of data collection, participants provided written informed consent in accordance with University of North

Carolina School of Public Health Institutional Review Board guidelines. A stratified random sample was selected out of all United States high schools that had an 11th grade and at least 30 students ($N = 132$ schools) to ensure that the sample was nationally representative based on size, type, region, urbanicity, and ethnicity. There were 4 waves of data collection from adolescence through young adulthood, with response rates of 79%, 89%, 77%, and 80%, respectively. The current analyses included 14,493 participants who had valid sampling weights, did not have a health provider diagnosis of cancer or heart disease, and participated in Wave 4 (W4) during which CVD risk factors were assessed (see Table S1 for demographics). The mean age of participants was 15.9 years at W1 (95% $CI = 15.7$ to 16.1), 21.8 at W3 (95% $CI = 21.5$ to 22.0), and 28.9 (95% $CI = 28.6$ to 29.1) at W4, the CVD risk assessment.

2.1. Exposures: childhood/adolescent adversity index and SES

Childhood/adolescent adversity. An adversity index was created as a sum of 12 items measuring adolescent dating violence, other adolescent interpersonal violence, four forms of child maltreatment, foster care, homelessness, maternal disability, and parental alcoholism, incarceration, and death (see Table S2 for wave of data collection for each variable). Dating violence included any instance of violence in which the participant's partner was the perpetrator. Other adolescent interpersonal violence included either witnessing or being the victim of violence from a non-partner, including physical fights and being jumped, shot, or stabbed. Participants reported on instances of child maltreatment that occurred before the sixth grade. The reportable childhood maltreatment types were 1) being left alone when an adult should be with you, 2) not having caregivers take care of your basic needs, like providing food or clothing, 3) having a caregiver slap, hit, or kick you, or 4) sexual abuse. Homelessness was defined as being homeless for a week or reporting a stay in a homeless shelter at any point at or before W3. Other adversities included parental death, parental incarceration (or incarceration of mother or father figure), parental alcoholism, maternal disability, or being in foster care. Each of the 12 categories was assigned a score of 1 if endorsed, and the sum of the endorsed categories was used as the childhood/adolescent adversity composite. The adversity scores ranged from 0 to 9 ($M = 1.65$, 95% $CI = 1.59$, 1.71).

SES during adolescence. SES during adolescence comprised three variables: total household income, parental education, and neighborhood poverty. The parent reported yearly total household income before taxes. Self-report of parental education was obtained for the parent who completed the baseline demographics questionnaire, which ranged from $0 = $ no formal education to $9 = $ professional training after 4-year college. Neighborhood poverty was defined as the proportion of families with incomes below the poverty line ranging from 0% to 86% in each adolescent's 1989 census block group. Poverty was then reverse scored for the composite variable so that higher numbers corresponded with lower neighborhood poverty. Together these three variables were used for a continuous latent variable of SES during adolescence derived in Mplus (all standardized individual factor loadings ranged from 0.36 to 0.71; all were significant at $p < 0.001$).

2.2. Mediators

Adolescent-mother relationship. A composite of the W1 ($M = 15.9$ years) maternal relationship was created using adolescent and mother report. Adolescents' perceptions of maternal support were assessed using five items from the W1 questionnaire. Two questions probed how close the participant feels to their mother and how much they believe their mother cares about them,

and participants responded on a 5-point Likert scale (not at all $= 1$, very little $= 2$, somewhat $= 3$, quite a bit $= 4$, very much $= 5$). Three items asked about agreement with statements about satisfaction with communication with their mother, lovingness and warmth of their mother, and overall satisfaction with the maternal relationship. Agreement with the statements was reported on a 5-point Likert scale (strongly agree $= 1$, agree $= 2$, neither agree nor disagree $= 3$, disagree $= 4$, strongly disagree $= 5$). The final three statements were reverse-scored so that higher numbers indicated higher maternal support. Previous Add Health research has reported good internal consistency for this scale ($\alpha = 0.84$ for European Americans, $\alpha = 0.83$ for African Americans; Deutsch et al., 2012), and several previous studies with Add Health data have used this scale (Deutsch et al., 2012; Trejos-Castillo and Vazsonyi, 2009; Wolff and Crockett, 2011). Caregivers (preferably resident mothers) also answered a questionnaire about their relationship with their adolescent, and six items were used to create a maternal support composite with the adolescent report. Mothers responded about how often they 1) get along well with their adolescent, 2) make decisions together about the adolescent's life, 3) understand their adolescent, 4) trust their adolescent, and 5) whether the adolescent interferes with the mother's activities. Mothers answered on a 5-point Likert scale: always $= 1$, often $= 2$, sometimes $= 3$, seldom $= 4$, never $= 5$. Mothers were also asked about whether they 6) agreed that they were satisfied with the relationship, and they could respond with strongly agree $= 1$, agree $= 2$, neither agree nor disagree $= 3$, disagree $= 4$, strongly disagree $= 5$. Items 1, 2, 4, and 6 were reverse-scored so that higher scores indicate a better relationship. The five adolescent items and six mother items were averaged to create a composite of the mother-adolescent relationship. The final composite ranged from 1.73 to 5.00 ($M = 4.27$, 95% $CI = 4.25$, 4.29).

Financial stress. Participants reported on their financial stress at W3 ($M = 21.8$ years) by answering if in the past 12 months, they were evicted; did not pay the rent/mortgage; could not afford to see their doctor; could not afford to see their dentist; did not pay the full amount of a gas, electricity, or oil bill because of lack of funds; had service turned off by the gas or electric company; or lacked service for their phone. Individuals positively endorsing any of the items were given a 1 for financial stress while others were given a 0.

Depressive symptoms. The Center for Epidemiologic Studies Depression Scale (CESD) was used to assess depressive symptoms at W3 with nine items to which participants rated the frequency of each symptom in the past week: $0 = $ never/rarely, $1 = $ sometimes, $2 = $ a lot of the time, or $3 = $ most of the time or all of the time. The sum of these responses returned a score from 0 to 27 for each individual ($M = 4.46$, 95% $CI = 4.34$, 4.59).

Health behaviors. Health behaviors at W3 shown to be associated with CVD or CVD risk factors (e.g., obesity) were assessed by frequency of physical activity, including a variety of activities such as aerobics, biking, hiking, playing sports, running, swimming, strength training ($0 = 5$–7 days/week, $1 = 1$–4 days/week, $2 = 0$ days/week); breakfast consumption ($0 = 5$–7 days/week, $1 = 1$–4 days/week, $2 = 0$ days/week); fast food consumption ($0 = $ no days in past week, $1 = 1$–4 days in past week, $2 = 5$–7 days in past week); smoking ($0 = $ never smoked, $1 = $ smoked but not in past 30 days, $2 = $ smoked in past 30 days); and alcohol consumption frequency ($0 = $ did not drink in past year, $1 = $ drank less than weekly in past year, $2 = $ drank at least weekly in past year). Each response was summed for a health behaviors score from 0 to 10 with higher scores indicating poorer health behaviors ($M = 5.39$, 95% $CI = 5.32$, 5.47).

Sleep problems. Sleep problems in the past 7 days were assessed at W3 by asking how often the participant fell asleep when they should have been awake, such as during class or at work.

Reponses ranged from 0 to 3: 0 = did not fall asleep when should have been awake, 1 = fell asleep a few times when should have been awake, 2 = fell asleep almost every day, and 3 = fell asleep every day ($M = 0.20$, 95% $CI = 0.19, 0.22$).

Lack of medical/dental care. Lack of care at W3 was assessed by two questions: 1) whether there was any time in the past 12 months when you thought you should get medical care and did not, and 2) whether you have had a dental exam in the past 12 months. If the participant answered that they had not gotten either medical or dental care, they were given a score of 1, and if they answered that they had received appropriate medical care (if needed) and a dental exam, they were give a score of 0.

Educational attainment. Educational attainment at W3 was assessed by asking the participant if they had less than a high school education = 1, were a high school graduate = 2, had some college = 3, or were a college graduate and above = 4.

Outcome. Data on CVD risk factors were collected at W4 when the participants were 24–34 years old ($M = 28.9$ years). Research assistants collected data at home visits with physical assessments and computer-assisted personal interviews. Height (to the nearest 0.5 cm) and weight (to the nearest 0.1 kg) were assessed using standardized procedures to calculate body mass index (BMI; (Entzel et al., 2009). A Microlife BP3MC1-PC-IB oscillometric blood pressure monitor (MicroLife USA, Inc.; Dunedin, FL) was used to measure systolic blood pressure (SBP) on the exposed right arm, absent contraindications, after participants had been seated for five minutes with legs uncrossed (Entzel et al., 2009). A total of three measurements were taken at 30-s intervals, and the last two measures were averaged to obtain resting SBP. A medication inventory was used to assess antihypertensive medication use in the past four weeks (Tabor and Whitsel, 2010), and cigarette smoking in the past 30 days was self-reported. Seven blood spots were collected on a capillary whole blood collection card via a lancet in order to capture capillary whole blood. Participants who reported eating or drinking anything (other than water) in the past 8 h were considered non-fasting and those who had not were considered fasting. Participants were considered to have diabetes mellitus for the purpose of the assessment if they had a fasting glucose ≥ 126 mg/dl, a non-fasting glucose ≥ 200 mg/dl, an HbA1c $\geq 6.5\%$, a health provider diagnosed them with diabetes outside of pregnancy, or used anti-diabetic medication in the past four weeks (Whitsel et al., 2012).

Young adult CVD risk was calculated using a function that predicted the risk of CVD development within a 30-year time frame (30-year Framingham CVD Risk Score; Pencina et al., 2009). The prediction function considered the co-occurrence of CVD risk factors and the degree of strength in predicting CVD. Thus, the prediction function was able to better predict subsequent risk than each of the risk factors individually. Using young adulthood as a time of predicting risk is an especially useful tool for accurately predicting subsequent CVD risk because risk factors in young adulthood have been demonstrated to be equally good or better predictors of later subclinical disease than assessments later in life (Gidding et al., 2006; Loria et al., 2007). The 30-year FRS is currently the only prediction function for young adults, and this longer timespan has shown better predictive power than shorter-term predictive functions for overt and subclinical CVD (Berry et al., 2009; Laing et al., 2012; Pencina et al., 2009). A strength of this measure is that the predictive model accounted for other causes of death (Pencina et al., 2009), which was important as young adults are much more likely to die from other causes for most of the 30-year prediction window. Ignoring other causes of death would lead to an overestimation of CVD risk (Pencina et al., 2009). The 30-year FRS used age, sex, BMI, use of antihypertensive medications, SBP, smoking and diabetic status (descriptive statistics for the

sample in Table S1) to predict the risk of a composite CVD outcome in the next 30 years, including myocardial infarction, coronary death, angina pectoris, coronary insufficiency, stroke, intermittent claudication, transient ischemic attack, and congestive heart failure (Pencina et al., 2009). CVD risk was assessed as a continuous variable. Mean risk in this sample was 0.132, or 13.2% (95% $CI = 0.128$ to 0.136). Pencina et al. (2009) provides greater detail on the development and validation of the 30-year risk score.

2.3. Covariates

To ensure that any effects of SES or adversity were not due to poorer health in adolescence, self-reported health was assessed at W1. Participants were asked, "In general, how is your health?" They responded with 1 = excellent (27.6%), 2 = very good (39.7%), 3 = good (25.7%), 4 = fair (6.4%), and 5 = poor (0.5%). This continuous variable was used in sensitivity analyses.

2.4. Data analytic plan

Descriptive statistics were calculated using SAS 9.4 (Table S1). Mplus Version 7.4 was used to calculate correlation coefficients among the study variables (Table S3). A single longitudinal mediation model was tested to understand the associations between childhood/adolescent adversity, SES during adolescence, and young adult CVD risk (Fig. 1). The full model entailed using path analysis to simultaneously test the direct and indirect effects of adversity and SES on CVD risk through adolescent maternal relationship and young adult health behaviors, financial stress, educational attainment, lack of medical/dental care, sleep problems, and depressive symptoms using maximum likelihood estimation with robust standard errors. The estimated indirect effects of adversity and SES on CVD risk through the W1 and W3 mediators were computed and standard errors were estimated using the delta method. These tests were adjusted for sex, age, and race/ethnicity (dummy-coded: white, black/African American, Hispanic, other). In a sensitivity analysis, adolescent self-reported health was included as a covariate to ensure that effects were not dependent on baseline health. The covariances of childhood/adolescent adversity and SES during adolescence with age, sex, and race/ethnicity were taken into account in the model as well as the covariances of the W3 variables with each other. All analyses accounted for unequal probability of selection and survey design per Add Health user guidance (Chantala and Tabor, 1999). Missing data was handled in Mplus using maximum likelihood estimation with robust standard errors, which was between 0.0% and 5.0% for all variables except for W1 parent education (14.1%), W1 parent household income (24.3%), W1 maternal relationship (17.0%), W3 financial stress (17.1%), W3 health behaviors (18.1%), W3 education (17.0%), W3 lack of care (16.9%), W3 sleep problems (17.2%), and W3 depressive symptoms (16.9%). Sampling weights provided by the Add Health team were used to compute population estimates. As a result, W4 non-response bias was negligible and the W4 sample adequately represented the W1 sample (Harris, 2013). Alpha was set at 0.05 for determinations of statistical significance.

3. Results

3.1. Analysis of covariates

Model covariates were evaluated first. Greater childhood/adolescent adversity was related to lower SES during adolescence, $\beta = -0.18$, $p < 0.001$. Adult CVD risk was negatively associated with being female, $\beta = -0.45$, $p < 0.001$, but positively associated with age, $\beta = 0.24$, $p < 0.001$. Higher CVD risk was associated with
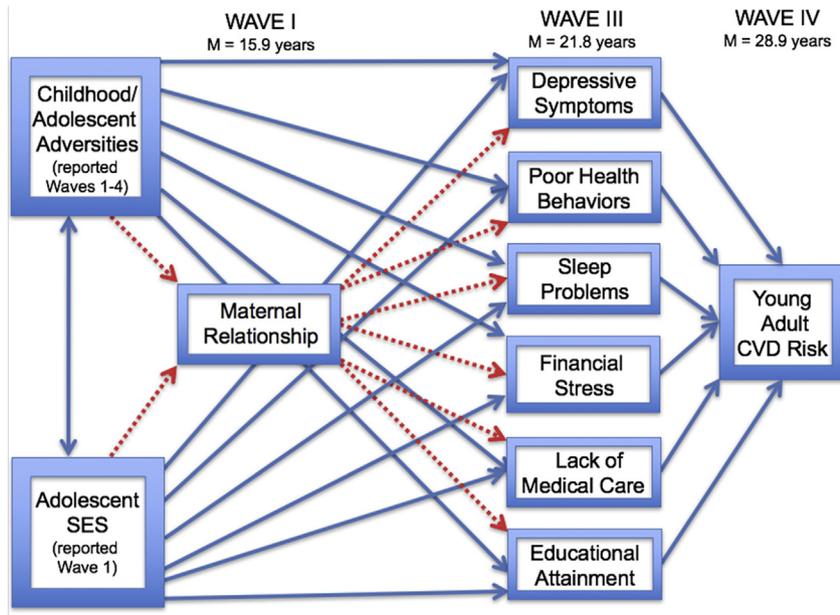
Fig. 1. Model tested in Mplus. Solid blue lines indicate direct paths between W1, W3, and W4 variables. Dashed red lines indicate direct paths that involve the addition of W1 maternal relationship as a mediator between SES → CVD risk and adversity → CVD risk. All direct and indirect paths from SES and adversity to CVD risk were tested. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

African American race, $\beta = 0.04$, $p < 0.001$, but not with being Hispanic or identifying as "other race," $ps > 0.05$. Greater childhood/adolescent adversities were associated with being male, $\beta = -0.09$, $p < 0.001$, African American race, $\beta = 0.09$, $p < 0.001$, Hispanic race, $\beta = 0.06$, $p < 0.001$, and other race, $\beta = 0.03$, $p < 0.01$. Lower SES in adolescence was not associated with sex, $p = 0.84$. SES in adolescence was lower for participants who were African American, $\beta = -0.28$, $p < 0.001$, or Hispanic, $\beta = -0.23$, $p < 0.001$, but not for other race, $p = 0.14$.

3.2. Main effects

All paths with standardized estimates are shown in Fig. 2. Controlling for the above covariates, the model revealed a significant total (direct plus indirect) effect of childhood/adolescent adversity on adult CVD risk, $\beta = 0.06$, 95% $CI = 0.04$, 0.07, $p < 0.001$, such that a one standard deviation increase in adversity during childhood/adolescence was associated with a 6% increased 30-year CVD risk in young adulthood. After controlling for all paths and
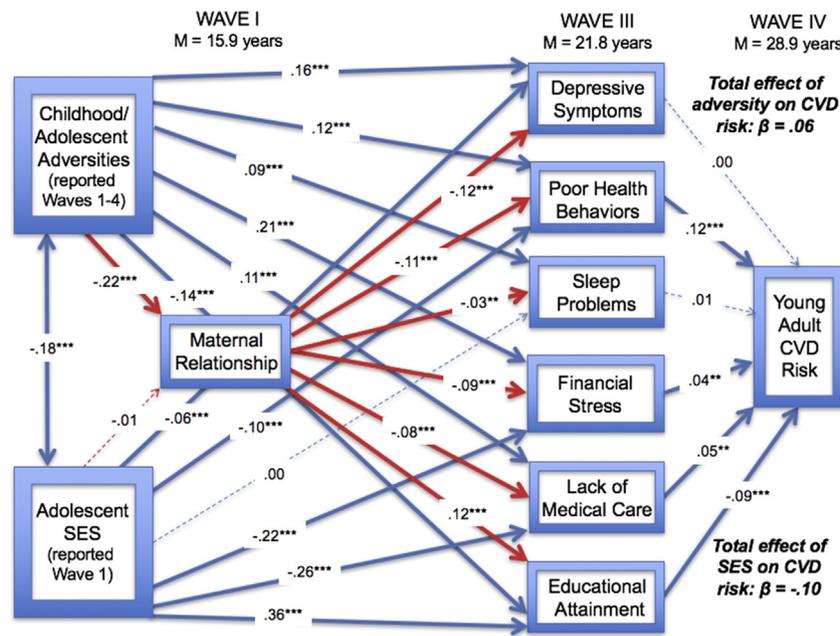
Fig. 2. Variables related to adult CVD risk with associated standardized estimates (β). This standardized estimate was the amount of change in Y per standard deviation unit of X. All paths reported were direct effects, *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. See online supplement for information on waves of data collection for childhood/adolescent adversities.

Table 1
Estimates for model pathways (N = 14,493).

	β	95% CI
Adversity→W1 and W3 Mediators		
Adversity→W1 Maternal Relationship	−0.22***	−0.24, −0.20
Adversity→W3 Poor Health Behaviors	0.12***	0.09, 0.14
Adversity→W3 Financial Stress	0.21***	0.18, 0.23
Adversity→W3 Educational Attainment	−0.14***	−0.16, −0.11
Adversity→W3 Lack of Medical/Dental Care	0.11***	0.07, 0.14
Adversity→W3 Depressive Symptoms	0.16***	0.13, 0.19
Adversity→W3 Sleep Problems	0.09***	0.06, 0.11
SES→W1 and W3 Mediators		
SES→ W1 Maternal Relationship	−0.01	−0.03, 0.02
SES→W3 Poor Health Behaviors	−0.10***	−0.14, −0.06
SES→W3 Financial Stress	−0.22***	−0.26, −0.19
SES→W3 Educational Attainment	0.36***	0.32, 0.39
SES→W3 Lack of Medical/Dental Care	−0.26***	−0.29, −0.22
SES→W3 Depressive Symptoms	−0.06***	−0.10, −0.03
SES→W3 Sleep Problems	0.00	−0.04, 0.04
W1 Maternal Relationship→W3 Mediators		
W1 Maternal Relationship→W3 Poor Health Behaviors	−0.11***	−0.14, −0.08
W1 Maternal Relationship→W3 Financial Stress	−0.09***	−0.13, −0.06
W1 Maternal Relationship→W3 Educational Attainment	0.12***	0.09, 0.14
W1 Maternal Relationship→W3 Lack of Medical/Dental Care	−0.08***	−0.12, −0.05
W1 Maternal Relationship→W3 Depressive Symptoms	−0.12***	−0.15, −0.10
W1 Maternal Relationship→W3 Sleep Problems	−0.03**	−0.06, −0.01
W1 and W3 Mediators→W4 CVD Risk		
W1 Maternal Relationship→W4 CVD Risk	−0.01	−0.03, 0.01
W3 Poor Health Behaviors→W4 CVD Risk	0.12***	0.10, 0.13
W3 Financial Stress→W4 CVD Risk	0.04**	0.01, 0.07
W3 Educational Attainment→W4 CVD Risk	−0.09***	−0.11, −0.06
W3 Lack of Medical/Dental Care→W4 CVD Risk	0.05**	0.02, 0.09
W3 Depressive Symptoms→W4 CVD Risk	0.00	−0.02, 0.02
W3 Sleep Problems→W4 CVD Risk	0.01	−0.01, 0.02

Note. All estimates reported are standardized estimates and 95% confidence intervals (CI) for each of the direct pathways. SES = socioeconomic status. CVD = cardiovascular disease. W1 = wave 1, W3 = wave 3, W4 = wave 4. *p < 0.05. **p < 0.01. ***p < 0.001.

mediators in the model, the direct effect between childhood/adolescent adversities and CVD risk was non-significant, $\beta = 0.01$, 95% CI = −0.01, 0.03, $p = 0.36$. The total effect of SES during adolescence on CVD risk in adulthood was significant, $\beta = -0.10$, 95% CI = −0.13, −0.08, $p < 0.001$, with lower SES related to increased CVD risk. This indicates that for a one standard deviation increase in SES, there was a subsequent 10% lower CVD risk. The direct effect of greater SES on lower CVD risk was significant even after controlling for all paths in the model, $\beta = -0.04$, 95% CI = −0.07, 0.00, $p < 0.01$. Thus, independent of all covariates and mediating pathways, a one standard deviation increase in SES was still associated with a 4% lower CVD risk.

3.3. Mediation

First, the associations between childhood/adolescent adversities and the W1 and W3 mediators were tested (Table 1). Greater childhood/adolescent adversities were related to a poorer maternal relationship in adolescence, poorer health behaviors, higher depressive symptoms, higher financial stress, lower educational attainment, a lack of medical/dental care, and more sleep problems.

Second, the associations between SES during adolescence and the W1 and W3 mediators were examined (Table 1). Lower SES was not related to the maternal relationship in adolescence or sleep problems at W3. Lower SES was related to poorer health behaviors, higher depressive symptoms, higher financial stress, a lack of medical/dental care, and lower educational attainment.

Third, the associations between the maternal relationship and the W3 mediators were tested (Table 1). There were direct associations between a more positive maternal relationship and lower depressive symptoms, poorer health behaviors, greater financial stress, lower educational attainment, a lack of medical/dental care,

and more sleep problems.

Fourth, the associations between the W1 and W3 mediators and adult CVD risk were tested (Table 1). The following were associated with greater adult CVD risk: poorer health behaviors, greater financial stress, lower educational attainment, and a lack of medical/dental care. Depressive symptoms and sleep problems were not associated with CVD risk. After controlling for all other paths, the direct association between adolescent maternal support and adult CVD risk was non-significant.

Finally, each indirect pathway was tested for significant mediation of the childhood/adolescent adversities-CVD risk and adolescent SES-CVD risk associations (see Table 2). The following pathways mediated the adversity-CVD risk association: health behaviors, financial stress, educational attainment, and lack of medical/dental care, but not depressive symptoms or sleep problems. Childhood/adolescent adversity indirectly affected CVD risk by impacting the adolescent maternal relationship, which then significantly affected health behaviors, educational attainment, financial stress, and a lack of medical/dental care ($p < 0.05$).

The following pathways significantly mediated the SES and CVD risk association: health behaviors, financial stress, educational attainment, lack of medical/dental care, but not depressive symptoms, sleep problems, or maternal support (Table 2). No indirect pathways from SES to CVD risk operated through the maternal relationship, as SES and the adolescent maternal relationship were not related.

3.4. Sensitivity analyses

After controlling for the effect of adolescent self-reported health on CVD risk, all paths remained significant, suggesting that these pathways operated above and beyond any effect of baseline self-

Table 2
Estimates for indirect pathways between Adversity→W4 CVD risk and SES→W4 CVD Risk (N = 14,493).

	β	95% CI
Adversity→CVD Risk		
Adversity→W1 Maternal Relationship→CVD Risk	0.002	−0.002, 0.006
Adversity→W3 Health Behaviors→CVD Risk	0.013***	0.009, 0.017
Adversity→W3 Financial Stress→CVD Risk	0.009*	0.002, 0.015
Adversity→W3 Educational Attainment→CVD Risk	0.012***	0.008, 0.015
Adversity→W3 Lack of Medical/Dental Care→CVD Risk	0.005**	0.002, 0.009
Adversity→W3 Depressive Symptoms→CVD Risk	0.000	−0.003, 0.004
Adversity→W3 Sleep Problems→CVD Risk	0.001	−0.001, 0.002
Adversity→W1 Maternal Relationship→W3 Health Behaviors→CVD Risk	0.003***	0.002, 0.004
Adversity→W1 Maternal Relationship→W3 Financial Stress→CVD Risk	0.001*	0.000, 0.002
Adversity→W1 Maternal Relationship→W3 Educational Attainment→CVD Risk	0.002***	0.001, 0.003
Adversity→W1 Maternal Relationship→W3 Lack of Medical/Dental Care→CVD Risk	0.001*	0.000, 0.002
Adversity→W1 Maternal Relationship→W3 Depressive Symptoms→CVD Risk	0.000	−0.001, 0.001
Adversity→W1 Maternal Relationship→W3 Sleep Problems→CVD Risk	0.000	0.000, 0.000
SES→CVD Risk		
SES→ W1 Maternal Relationship→CVD Risk	0.000	0.000, 0.000
SES→W3 Health Behaviors→CVD Risk	−0.012***	−0.017, −0.007
SES→W3 Financial Stress→CVD Risk	−0.009**	−0.016, −0.002
SES→W3 Educational Attainment→CVD Risk	−0.030***	−0.039, −0.022
SES→W3 Lack of Medical/Dental Care→CVD Risk	−0.013**	−0.022, −0.004
SES→W3 Depressive Symptoms→CVD Risk	0.000	−0.001, 0.001
SES→W3 Sleep Problems→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Health Behaviors→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Financial Stress→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Educational Attainment→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Lack of Medical/Dental Care→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Depressive Symptoms→CVD Risk	0.000	0.000, 0.000
SES→W1 Maternal Relationship→W3 Sleep Problems→CVD Risk	0.000	0.000, 0.000

Note. All estimates are standardized estimates and 95% confidence intervals for each of the indirect pathways in the model. SES = socioeconomic status. CVD = cardiovascular disease. W1 = wave 1, W3 = wave 3, W4 = wave 4. *$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

reported health. In order to test whether any effects of SES during adolescence or child/adolescent adversity were attenuated due to the inclusion of both predictors in the model, two models were run separately. The first model was run with only SES pathways to CVD risk (with maternal relationship and W3 mediators) and the second model was run with only adversity pathways to CVD risk (with maternal relationship and W3 mediators). The model with only SES pathways revealed that the direct pathways from SES to W3 mediators remained the same, but pathways from SES→W1 maternal relationship→W3 mediators→CVD risk became significant for the following W3 mediators: financial stress, educational attainment, and health behaviors, $p < 0.05$. The model with only childhood/adolescent adversity pathways did not have any pathways change significance level from the full model.

In order to test whether depressive symptoms and sleep problems were non-significant due to the inclusion of other mediators, we conducted two separate models with the first having depressive symptoms as the only W3 mediator between SES, adversity, maternal relationship, and CVD risk, and a second identical model having sleep problems as the only W3 mediator. In the depressive symptoms model, depressive symptoms significantly mediated the association between childhood/adolescent adversity and CVD risk through the following two pathways: adversity→W3 depressive symptoms→CVD risk, and adversity→W1 maternal relationship→W3 depressive symptoms→CVD risk. Depressive symptoms still did not mediate the SES-CVD risk association in this model. In the sleep problems model, sleep did not mediate the adversity-CVD risk or SES-CVD risk associations.

4. Discussion

The current analyses demonstrate that both SES during adolescence and child/adolescent adversities are significantly associated with young adult CVD risk. Overall, a one standard deviation increase in SES was associated with a 10% decrease in CVD

risk, while a one standard deviation increase in child/adolescent adversity was associated with a 6% increase in risk. The W1 and W3 mediators mediated the association between child/adolescent adversity and CVD risk, with no direct association between adversity and CVD risk. SES had a direct association with CVD risk even after accounting for pathways through W1 and W3 mediators.

These analyses indicated that there were similar pathways from SES during adolescence and child/adolescent adversities to young adult CVD risk. However, the maternal relationship in adolescence only appeared to mediate the adversity-CVD association and not the SES-CVD association. The maternal relationship mediated the adversity-CVD association by altering downstream mediators, including health behaviors, financial stress, medical/dental care, and educational attainment, which then affected CVD risk. As a result, the adolescent maternal relationship may be a target for intervention in adolescents who have experienced early adversity to lower CVD risk by reducing young adult risk factors that are affected by this social relationship in adolescence. However, forms of adversity perpetrated by the mother (e.g., child maltreatment) may be particularly difficult targets for interventions, although there are some evidence-based approaches that have been shown to prevent child maltreatment (Levey et al., 2017; MacMillan et al., 2009). These evidence-based approaches may also have downstream impacts on CVD risk.

The results of this study are consistent with research demonstrating that childhood/adolescent adversities and poverty are independently associated with higher CVD risk over time (Miller et al., 2011a). Our results are unique in that they suggest that the impact of adversities on CVD risk were mediated by the maternal relationship, health behaviors, financial stress, medical/dental care, and educational attainment; SES still showed a direct effect on CVD risk even after accounting for these mediators. Further, these analyses show a negative association between adversity and the perceived adolescent maternal relationship, consistent with the literature on adversity and risky families (Repetti et al., 2002). Our

results were also consistent with evidence that negative relationships and risky environments have downstream impacts on health behaviors, adult functioning, and physical health (Repetti et al., 2002). Thus, our findings on pathways between childhood/adolescent adversities and adult CVD risk were largely consistent with the literature. However, contrary to what we predicted based on previous studies, we found that SES did not impact CVD risk through the adolescent maternal relationship.

As many of the pathways between early SES and adversity and later CVD risk were the same, similar interventions may be applied to individuals who have experienced either or both of these early events. However, it may be beneficial to probe for information about various childhood and adolescent adversities, which may affect the maternal relationship and in turn alter pathways leading to later CVD risk. Interventions may benefit from having a specific arm focused on improving relationships that may have been negatively affected by adverse experiences, as positive relationships may lower the risk usually conferred by environmental factors. For example, a study by Miller and colleagues (Miller et al., 2011b) demonstrated that adults whose parents had low educational attainment had greater metabolic syndrome risk than adults whose parents had higher educational attainment. In addition, adults who experienced high maternal nurturance as a child did not show this childhood SES gradient effect on health (Miller et al., 2011b). Thus, although the maternal relationship did not mediate the SES-CVD association, it may remain an important moderator. Further, adults who experienced abuse in childhood but whose parents showed greater warmth had lower levels of wear and tear on the body, suggesting that the maternal relationship may buffer from negative consequences of early adversity (Carroll et al., 2013). It is certainly plausible that encouraging positive behaviors such as maternal nurturance in families experiencing adversity may mitigate some of these negative effects on physiological systems.

Contrary to the hypotheses, SES was not associated with maternal relationship quality in the full model, which was unexpected considering evidence that lower SES households have higher rates of neglectful parenting, more authoritarian parenting styles, and less parental warmth (Dornbusch et al., 1987; Glasgow et al., 1997; Leventhal and Brooks-Gunn, 2000). Nonetheless, many studies have failed to separate SES from adverse experiences, which often co-occur. It could be that adverse experiences that are more likely to occur in families with low SES contribute more to observed parenting styles than SES, and that adversity is actually more highly associated with parenting quality and the parent-child relationship than SES. Our sensitivity analyses suggested that this might be the case, as there were significant pathways from SES through the adolescent maternal relationship when adversity was removed from the model. Accordingly, mechanisms operating through the adolescent maternal relationship are likely more robust for those who experienced adversity than low SES. Although it is often difficult to tease apart SES and adversities, hypotheses about unique effects of SES versus adversity need to be examined in future research.

This study did not show evidence that the maternal relationship conferred unique effects on CVD risk, but rather that the relationship impacted downstream mediators (e.g., educational attainment, health behaviors) that then went on to affect CVD risk. Thus, mothers, and potentially fathers and other supportive individuals, may serve as protective factors by improving functioning in downstream mediators; but they may also confer additional risk if the relationship is negative by increasing stress and adversely altering these downstream mediators. Interestingly, depressive symptoms and sleep problems did not impact CVD risk as predicted, which could be because other mediators were highly correlated with depressive symptoms and sleep problems and were

more strongly associated with CVD risk, which likely left the associations non-significant after controlling for all paths in the model. This hypothesis is partially supported by the sensitivity analysis demonstrating that depressive symptoms mediated the childhood/adolescent adversity→CVD risk association when depressive symptoms were tested as the only W3 mediator. Still, sleep problems did not mediate associations between adversity, SES, and CVD risk, even when tested as the only W3 mediator. This result was unexpected due to research that daytime sleepiness predicts mortality and CVD morbidity in older adults (Newman et al., 2000); however, this may have been due to the young adult cohort assessed in the current study or the lack of correlation with several factors in the CVD risk index.

Although most of the mediators examined were behavioral, social, or environmental in nature, several potential biological mechanisms may underlie pathways between childhood/adolescent SES and adversity and later CVD risk. For example, repeated responses to stressors in a high adversity or low SES environment may program the hypothalamic-pituitary-adrenal axis or certain inflammatory regulators to respond to future events in ways which may be damaging to long-term health (Doom and Gunnar, 2013; Shonkoff et al., 2012). In addition, stress during sensitive periods of brain development, particularly in childhood and adolescence, may alter neural systems that govern responses to stress, health behaviors, cognitive functioning, emotion, and behavior (Lupien et al., 2009). These alterations in brain development likely have long-term consequences for biology and behavior, including risk for disorders like CVD.

4.1. Limitations

There were limitations to the study that must be discussed. First, this study did not use observed measures of the maternal relationships but rather self-reports by the adolescents and their mothers, so it is unclear whether their perceptions were an accurate reflection of the relationship quality. Second, the maternal relationship was assessed at the same time point as SES and some of the measures of adversity. Although we theoretically framed the analyses as SES and adversity affecting the maternal relationship longitudinally, we cannot rule out bidirectional associations. Some adversities were retrospectively reported; however, they were reported during adolescence or young adulthood, which was much closer in time to the adversity than the typical timing of adversity measures in large epidemiologic studies (frequently in middle to late adulthood). Third, perceptions of paternal relationships were not examined because there were significantly fewer father figures than mothers in this study. Future research should incorporate reports of paternal support.

Fourth, the prediction function of CVD risk was developed on the Framingham Offspring Cohort, initiated in 1971 (Kannel et al., 1979). Some of the underlying disease risk and prevalence of certain risk factors have changed, such as increases in obesity and decreases in smoking rates in the US. However, it is still the only long-term prediction function for use in young adults, and the composite is an improvement over assessing individual risk factors separately. Recent work has documented the utility of the FRS prediction function in the Add Health study (Clark et al., 2014), although ongoing research is needed to validate this function on contemporary cohorts. Finally, the W3 mediators were not all assessed on the same time scale. For instance, financial stress was measured for the past year, while depressive symptoms were measured in the past week and health behaviors from one week to the past year. Therefore, although we presume that depressive symptoms and health behaviors may be somewhat stable over time, it might not be the case. This difference in measurement

timing among the mediators must be considered when interpreting the results. Likewise, there was some overlap in measurement, such that smoking was a negative health behavior at W3 and is part of the CVD risk index at W4. Although it is difficult to untangle risk behaviors from the risk index, this association should be considered during interpretation.

4.2. Conclusion

Overall, these analyses indicated that in a large, nationally representative longitudinal sample of adolescents through young adults, childhood/adolescent SES and adversity each increased risk for CVD risk through the following pathways: financial stress, health behaviors, educational attainment, and lack of medical/dental care, but not through depressive symptoms or sleep problems. Childhood/adolescent adversity negatively impacted the adolescent maternal relationship, which then impacted downstream mediators that affect cardiovascular health. Interestingly, SES during adolescence did not affect the adolescent maternal relationship, indicating that there were some unique pathways by which SES and adversity impacted CVD risk. This knowledge may be applied to interventions by targeting shared and unique risk factors for poor health following early adversity and low SES. Interventions targeting the adolescent maternal relationship following early adversity may be particularly helpful for improving health behaviors, educational attainment, financial stress, and the ability to access medical/dental care, which are important predictors of long-term CVD risk.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2017.06.044.

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