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# Maternal Relationship During Adolescence Predicts Cardiovascular Disease Risk in Adulthood

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***Objective:*** The current study investigated whether greater maternal support during adolescence is associated with lower levels of cardiovascular disease (CVD) risk in adulthood, and whether maternal support serves as a moderator or a mediator of the socioeconomic status (SES) and CVD risk association. In addition, potential moderators and mediators of the association between adult CVD risk and adolescent maternal support and SES were tested. ***Method:*** Using the National Longitudinal Study of Adolescent to Adult Health ($n$ = 11,013), we examined relations between maternal support during adolescence ($M$ = 15.3 years) and CVD risk in young adulthood ($M$ = 28.7 years) via path analysis. Maternal support was assessed by a composite of adolescent and mother report. CVD risk was calculated with a Framingham-based prediction model that uses age, sex, body mass index, smoking, systolic blood pressure, diabetes, and use of antihypertensive medication. ***Results:*** Greater maternal support in adolescence was related to lower CVD risk in young adulthood ($B$ = −0.56, 95% CI: −0.91 to −0.20, $p < .01$). The interaction between adolescent SES and maternal support was not significant, ($p > .05$), but there was an interaction between maternal support and race such that African American adolescents were more sensitive than Whites to the effects of maternal support on CVD risk ($B$ = −0.90, 95% CI: −1.56, −0.25, $p < .01$). In addition, there was no evidence that maternal support mediated the association between SES and CVD risk ($p > .05$), and there was no association between SES and maternal support ($p > .05$), adjusting for confounders. However, the relations of adolescent maternal support and SES to adult CVD risk were mediated by young adult health behaviors and financial stress but not by depressive symptoms. ***Conclusion:*** Greater maternal support during adolescence appears to act independently of SES when impacting CVD risk and may operate through health behaviors and financial stress.

*Keywords:* maternal support, cardiovascular disease, socioeconomic status, Add Health, health disparities



In the United States, cardiovascular disease (CVD) is responsible for over 2,150 deaths each day, making it the leading cause of death for both men and women (Go et al., 2014; Hoyert & Xu, 2012). CVD prevalence is increasing rapidly, with 40.5% of Americans expected to suffer from some form of the disease by 2030 (Heidenreich et al., 2011). In addition, significant disparities in CVD prevalence exist by race and socioeconomic status (SES), indicating that individuals who are black and those in low SES groups are more likely to develop this debilitating disease due to factors often outside of their control (Adler & Newman, 2002). Because of the rapidly increasing prevalence and cost of CVD, as well as significant disparities across race and SES, it is vital to understand risk and protective factors related to CVD to inform prevention and intervention efforts. Research must also focus on factors that may differentially affect groups that might account for the disparities in CVD.

One factor that may contribute to CVD disparities is maternal support during childhood and adolescence. Recent evidence indicates that maternal warmth during childhood is associated with more favorable levels of biomarkers associated with CVD in adulthood, including blood pressure, heart rate, C-reactive protein, cortisol, cholesterol, glucose, and insulin (Carroll et al., 2013). In addition, infants who were securely attached to their caregivers during infancy were less likely to report inflammation-related diseases, such as coronary heart disease, stroke, diabetes, and hypertension, in adulthood than those who were insecurely attached in infancy (Puig, Englund, Simpson, & Collins, 2013). A 35-year follow-up study reported that 91% of men who did not have a warm relationship with their mother during young adulthood had been diagnosed with a chronic disease (e.g., coronary heart disease, hypertension, alcoholism) later in life, while only 45% of those reporting a warm relationship were living with chronic disease years later (Russek & Schwartz, 1997). Thus, there

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appears to be a main effect of maternal support on CVD risk factors in adulthood.

Maternal support during childhood and adolescence may act as a moderator or a mediator of the SES-CVD association. Consistent with a moderation hypothesis, maternal support may be especially protective against poor physical health for individuals from more disadvantaged backgrounds. In these harsher environments, the presence of a maternal figure that provides greater support in the face of adversity may confer greater benefits in terms of CVD risk. For example, adults who were raised in families with lower parental educational attainment had more metabolic syndrome components than children of parents with greater educational attainment (Miller et al., 2011); however, this SES effect was not present for individuals who experienced high maternal nurturance during childhood (Miller et al., 2011). This finding suggests that children from low SES families are more sensitive to variations in maternal support than individuals from high SES families, whose metabolic outcomes remain relatively stable across maternal support groups (Miller et al., 2011). Children from low SES families may be more sensitive to the effects of maternal support as they have fewer institutional and personal resources to promote good health. In addition, a recent study demonstrated both a main effect of parental warmth during childhood and an interaction between warmth and early adversity in predicting adult health (Carroll et al., 2013). The findings indicated that having a warm relationship with a parent during childhood was related to decreased wear and tear on the body during adulthood (Carroll et al., 2013). Those who had the highest multisystem risk were those who were abused during childhood and had the lowest levels of parental warmth (Carroll et al., 2013), suggesting that those in stressful environments may be particularly sensitive to variations in maternal care. Thus, having a supportive relationship with parents may help children and adolescents cope with stressors and buffer themselves from stress-related health problems, which may be the difference between positive adaptation and negative disruptions in the brain and other biological systems.

On the other hand, maternal support may act as a mediator between harsh environments and CVD risk, such that living in low SES environments causes stress for mothers, which decreases maternal warmth and then leads to increased CVD risk. Consistent with a mediation hypothesis, there is evidence that parenting is an important mediator between low childhood SES and adult health outcomes. As differences in parenting have been reported by SES, variations in parenting may be at least partially responsible for disparities in physical health outcomes and behaviors. There is significant evidence that parenting behaviors in low SES families may be affected by harsh circumstances, which then influence offspring's physical and mental health and health behaviors (reviewed in Repetti et al., 2002). Large studies have reported more authoritarian and less authoritative parenting in households with lower SES as well as higher rates of neglectful parenting (e.g., Dornbusch, Ritter, Leiderman, Roberts, & Fraleigh, 1987; Glasgow et al., 1997). Lower maternal warmth and lower quality parenting have also been reported in families living in more impoverished or dangerous neighborhoods than in families living in safer and more advantaged neighborhoods (reviewed in Leventhal & Brooks-Gunn, 2000). It could be that hardships affecting low SES families lead to poorer outcomes in children through higher parental stress and depressive symptoms, which then affect parenting behaviors (Conger, Ge, Elder, Lorenz, & Simons, 1994; McLoyd, 1990; McLoyd, Jayaratne, Ceballo, & Borquez, 1994). The current study aims to understand whether perceived maternal support during adolescence is associated with lower CVD risk in adulthood and whether maternal support acts as a moderator of the effect of SES on CVD risk or a mediator between SES and CVD risk.

Unfortunately, many studies examining the impact of childhood and adolescent risk factors for adult CVD development rely on retrospective reports, where the adult reports on their circumstances oftentimes decades from the exposure. Their adult report could be biased by a number of factors, such as experiences that have occurred since the period of interest. In addition, it is unclear whether maternal support beyond childhood affects CVD risk in adulthood. Most researchers target maternal support during childhood rather than adolescence, which is another sensitive period for development. The present study examines perceptions of maternal support in adolescence in relation to predicted long-term CVD risk calculated with the 30-year Framingham CVD Risk Score using Add Health data. It is hypothesized that the perception that one's mother is supportive and caring during adolescence will be associated with lower risk for CVD in adulthood. Further, it is hypothesized that this relation will be stronger in individuals with higher risk for CVD, specifically those in low SES groups or those who are African American or Hispanic, with the prediction that individuals in these groups will be more sensitive to the effects of maternal support. Finally, mediators will be assessed to understand potential pathways between adolescent maternal support and adult CVD risk. Specifically, health behaviors, financial stress, and depressive symptoms in young adulthood will be assessed with the prediction that each of these will significantly mediate the maternal support-CVD risk association.

## Method

Analyses were conducted using Add Health data (Harris et al., 2009). Written informed consent was obtained from participants at each wave of data collection in accordance with guidelines established by the University of North Carolina, School of Public Health Institutional Review Board. Researchers selected a stratified random sample of all high schools in the United States that had at least 30 students and had an 11th grade. The core sample included 132 schools. These schools were nationally representative based on region, size, type, urbanicity, and ethnicity. There were 4 total waves of data collection from adolescence through young adulthood. Response rates were 79%, 89%, 77% and 80%, respectively. The current study includes 11,013 participants who had a maternal figure in the home at baseline, had a maternal figure reporting on their relationship at baseline, had valid sampling weights, were free of a health provider diagnosis of cancer and heart disease, were White, Black/African American or Hispanic, and participated in Wave 4, the visit in which CVD risk factors needed to calculate the composite CVD risk score were assessed (see Tables 1 and 2 for sample characteristics). Participants who were included did not differ in sex, adolescent SES, or maternal relationship quality from those excluded, but they did differ in age, race, likelihood of living in a single mother household, adolescent health, and CVD risk. Excluded participants were slightly older, were more likely to be categorized as "other race" (not white,

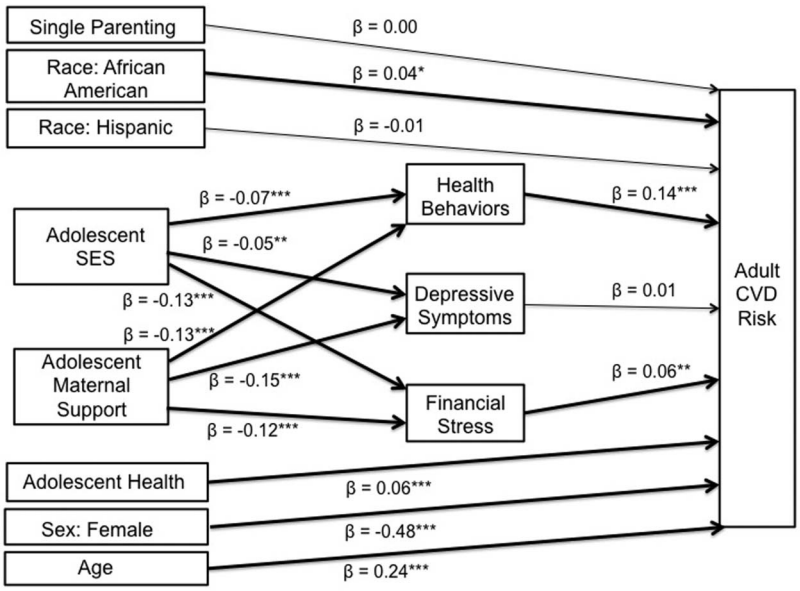


*Figure 1.* Variables affecting adult cardiovascular disease (CVD) risk with associated standardized estimates. Race variables are in comparison with the White race. All paths reported are direct effects. $^{*}$ $p < .05$. $^{**}$ $p < .01$. $^{***}$ $p < .001$.

African American, or Hispanic), live in a single mother family, report being less healthy during adolescence, and have a greater CVD risk score during young adulthood. Participants' mean age was 15.3 years at baseline (95% CI: 15.1 to 15.6) and 28.7 (95% CI: 28.5 to 29.0) at CVD risk assessment.

## Wave 1 Assessment

To assess for the adolescents' perceptions of maternal support, five items from the adolescent questionnaire at Wave 1 were used. The first two questions ask how close the participant feels to their mother and how much they think their mother cares about them. Responses were on a 5-point Likert scale (1 = *not at all*; 2 = *very little*; 3 = *somewhat*; 4 = *quite a bit*; and 5 = *very much*). The final three items are statements about satisfaction with communication with their mother, warmth and lovingness of their mother, and overall maternal relationship satisfaction. Agreement with these statements was scored on a 5-point Likert scale (1 = *strongly agree*; 2 = *agree*; 3 = *neither agree nor disagree*; 4 = *disagree*; 5 = *strongly disagree*). The final three items were reverse-scored so that higher scores indicated higher perceived maternal support. Previous research with Add Health data has reported good internal consistency for the scale ($\alpha$ = .84 for European Americans, $\alpha$ = .83 for African Americans; Deutsch, Crockett, Wolff, & Russell, 2012). This scale has been used in several previous studies examining Add Health data (e.g., Deutsch et al., 2012; Trejos-Castillo & Vazsonyi, 2009; Wolff & Crockett, 2011).

Caregivers (preferably resident mothers) were also given a questionnaire about their relationship with the adolescent, and six items were used for the maternal support composite. Mothers were asked how often they get along well with their adolescent, make decisions about the adolescent's life together, understand their adolescent, trust the adolescent, and whether the adolescent interferes with their activities. Mothers responded on a scale on which 1 = *always*, 2 = *often*, 3 = *sometimes*, 4 = *seldom*, and 5 = *never*. In addition, mothers were asked whether they agreed that they were satisfied with the relationship and could respond 1 = *strongly agree*, 2 = *agree*, 3 = *neither agree nor disagree*, 4 = *disagree*, or 5 = *strongly disagree*. Items 1, 2, 4, and 6 were reverse scored so that higher numbers indicate a better relationship. The five adolescent items and six parent items were averaged to create a two informant composite of the parent−adolescent relationship. The final composite ranged from 1.8 to 5.0.

Single parenting was limited to single mothers, as the maternal relationship was the focus of the study. Adolescents were asked whether their father, mother's husband, or mother's partner lived in their household. If there was no resident father or father figure, adolescents were classified as being in a single mother household. To control for health in adolescence, participants reported their general health at Wave 1 by responding to the following question: "In general, how is your health?" Participants responded with 1 = *excellent*, 2 = *very good*, 3 = *good*, 4 = *fair*, 5 = *poor*, so higher values indicated poorer adolescent health.

The adolescent SES composite was the average of three *z*-scored variables: parental education, total household income, and neighborhood poverty. Parental education was self-reported by the parent who filled out the demographics questionnaire at baseline and ranged from 0 (*no formal education*) to 9 (*professional training beyond 4 years in college*). Total household income before taxes for the year was reported. *Neighborhood poverty* was defined as the proportion of families in the adolescent's census block group with incomes in 1989 that were below the poverty line ranging from 0.0% to 86.0%. These three components were *z*-scored and then averaged for the final adolescent SES composite. If any of the three components were not present, SES was defined as missing.

Table 1
*Descriptive Statistics for Males (N = 5,129)*

| Variable | White nonHispanic ($n$ = 3,221) | | | African American ($n$ = 1,056) | | | Hispanic ($N$ = 852) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Low SES ($n$ = 1,139) | High SES ($n$ = 1,695) | Missing SES ($n$ = 387) | Low SES ($n$ = 542) | High SES ($n$ = 332) | Missing SES ($n$ = 182) | Low SES ($n$ = 509) | High SES ($n$ = 210) | Missing SES ($n$ = 133) |
| Maternal support ($M$ [$SE$]) | 4.28 (.02) | 4.32 (.02) | 4.28 (.03) | 4.30 (.02) | 4.30 (.03) | 4.23 (.07) | 4.35 (.04) | 4.33 (.05) | 4.41 (.03) |
| Single mother household (% [95% CI]) | 22.0 [18.8,25.4] | 10.6 [8.9,12.5] | 15.78 [11.3,21.5] | 54.5 [47.7,61.1] | 28.2 [21.6,35.8] | 55.8 [45.3,65.8] | 25.4 [19.9,31.8] | 19.0 [11.4,30.0] | 32.2 [22.0,44.2] |
| Poverty ($M$ [$SE$]) | .15 (.01) | .04 (.00) | .09 (.01) | .28 (.01) | .07 (.01) | .23 (.03) | .20 (.01) | .05 (.00) | .17 (.02) |
| Household income ($M$ [$SE$]) | 29.02 (.75) | 67.97 (2.45) | 34.2 (4.4) | 20.13 (1.11) | 56.08 (3.13) | 20.2 (3.79) | 23.37 (1.03) | 66.76 (10.0) | 24.82 (4.67) |
| Parent education | | | | | | | | | |
| Less than high school (% [95% CI]) | 20.0 [17.3,23.0] | .1 [.0,.6] | 17.9 [12.0,25.9] | 26.3 [18.7,35.6] | .0 | 23.4 [12.8,38.7] | 60.8 [54.2,67.1] | .0 | 63.1 [46.2,77.4] |
| High school/GED (% [95% CI]) | 56.8 [52.6,60.8] | 16.8 [13.8,20.2] | 38.3 [30.0,] | 36.6 [28.5,45.6] | 11.7 [6.7,19.5] | 36.2 [25.4,48.6] | 24.0 [18.0,31.2] | 20.2 [11.9,32.0] | 10.9 [5.3,21.0] |
| Vocational/some college (% [95% CI]) | 21.0 [17.5,25.0] | 40.8 [37.0,44.6] | 27.2 [20.5,35.2] | 28.2 [24.5,32.2] | 38.8 [30.1,48.4] | 20.9 [13.9,30.3] | 13.3 [8.7,19.7] | 47.44 [37.3,57.9] | 19.1 [10.8,31.4] |
| College graduate (% [95% CI]) | 2.2 [.5,3.6] | 42.3 [37.1,47.8] | 16.6 [11.9,22.7] | 8.9 [4.9,15.8] | 49.5 [39.7,59.4] | 19.5 [11.6,31.0] | 1.9 [.9,4.0] | 32.4 [23.5,42.7] | 6.9 [2.8,15.9] |
| Adolescent health ($M$ [$SE$]) | 2.13 (.04) | 1.93 (.03) | 2.05 (.06) | 2.07 (.08) | 1.92 (.06) | 1.97 (.08) | 2.11 (.07) | 2.03 (.11) | 2.16 (.10) |
| CESD Wave 3 ($M$ [$SE$]) | 3.84 (.14) | 3.63 (.09) | 3.55 (.23) | 4.84 (.33) | 4.22 (.38) | 3.83 (.33) | 4.91 (.03) | 4.07 (.44) | 4.97 (.56) |
| Health behaviors ($M$ [$SE$]) | 5.97 (.09) | 5.56 (.07) | 5.76 (.01) | 5.28 (.16) | 5.62 (.20) | 4.95 (.16) | 5.37 (.15) | 5.52 (.23) | 4.82 (.36) |
| Financial stress ($M$ [$SE$]) | .62 (.04) | .44 (.03) | .38 (.06) | .70 (.08) | .49 (.09) | .60 (.13) | .52 (.06) | .40 (.12) | .62 (.28) |
| CVD risk score ($M$ [$SE$]) | 17.83 (.44) | 15.72 (.29) | 16.58 (.62) | 18.42 (.76) | 19.14 (1.13) | 18.37 (.85) | 17.70 (.68) | 16.15 (.67) | 18.30 (1.42) |
| Current smoker (% [95% CI]) | 50.1 [46.1,54.1] | 38.9 [35.5,42.4] | 45.3 [38.3,52.5] | 45.4 [39.7,51.2] | 35.0 [26.6,44.6] | 42.4 [30.6,55.2] | 36.6 [29.9,43.8] | 39.5 [29.4,50.7] | 41.6 [27.2,57.6] |
| Systolic blood pressure, ($M$ [$SE$]) | 131.25 (.54) | 129.47 (.40) | 128.6 (.77) | 129.74 (.80) | 130.99 (1.28) | 120.59 (1.98) | 130.59 (.96) | 129.26 (1.66) | 127.80 (1.40) |
| Evidence of diabetes (% [95% CI]) | 4.4 [3.1,6.2] | 4.3 [3.2,5.8] | 3.2 [1.5,6.8] | 12.7 [9.7,16.6] | 17.6 [12.2,24.5] | 11.7 [7.8,17.3] | 10.0 [6.4.15.5] | 2.4 [.7,7.7] | 13.9 [7.11,25.5] |
| BMI ($M$ [$SE$]) | 29.60 (.29) | 28.12 (.20) | 28.9 (.56) | 28.86 (.42) | 29.60 (.44) | 29.09 (.78) | 30.90 (.63) | 29.23 (.63) | 30.24 (1.06) |
| Antihypertensive medication (% [95% CI]) | 4.9 [3.3,7.2] | 3.5 [2.5,5.0] | 3.1 [1.5,6.4] | 2.8 [1.7,4.8] | 3.0 [1.4,6.5] | 3.3 [1.3,8.0] | 1.6 [.7,4.0] | 2.3 [.7,7.3] | .1 [.0,.3] |

*Note.* Household income is reported in thousands of dollars. Poverty is the proportion of families in the adolescent's census block group with incomes in 1989 that were below the poverty line. SES = socioeconomic status; GED = general equivalency diploma; CESD = Center for Epidemiologic Studies Depression Scale; CVD = cardiovascular disease; BMI = body mass index.

Table 2
*Descriptive Statistics for Females (N = 5,884)*

| Variable | White nonHispanic ($n$ = 3,505) | | | African American ($n$ = 1,414) | | | Hispanic ($N$ = 965) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Low SES ($n$ = 1,241) | High SES ($n$ = 1,814) | Missing SES ($n$ = 450) | Low SES ($n$ = 752) | High SES ($n$ = 378) | Missing SES ($n$ = 284) | Low SES ($n$ = 568) | High SES ($n$ = 203) | Missing SES ($n$ = 194) |
| Maternal support ($M$ [$SE$]) | 4.23 (.02) | 4.26 (.02) | 4.26 (.03) | 4.21 (.03) | 4.24 (.05) | 4.13 (.05) | 4.22 (.03) | 4.22 (.05) | 4.22 (.06) |
| Single mother household (% [95% CI]) | 24.5 [21.5,27.8] | 13.5 [11.4,16.0] | 15.5 [11.8,20.2] | 55.2 [49.7,60.6] | 33.7 [26.5,41.8] | 55.0 [47.2,62.6] | 25.3 [19.2,32.6] | 14.0 [8.1,23.2] | 15.8 [9.2,25.6] |
| Poverty ($M$ [$SE$]) | .14 (.01) | .04 (.00) | .08 (.01) | .27 (.02) | .07 (.01) | .29 (.02) | .18 (.02) | .04 (.00) | .15 (.02) |
| Household income ($M$ [$SE$]) | 29.60 (.61) | 67.78 (2.73) | 46.86 (9.00) | 21.29 (1.04) | 62.05 (4.49) | 24.91 (4.04) | 23.82 (1.03) | 60.72 (5.11) | 30.37 (6.66) |
| Parent education | | | | | | | | | |
| Less than high school (% [95% CI]) | 24.3 [21.0,28.0] | .2 [.0,1.5] | 16.6 [11.4,23.5] | 25.5 [19.7,32.3] | 1.7 [.4,6.5] | 29.8 [22.6,38.3] | 54.1 [43.9,64.0] | .9 [.2,4.1] | 57.4 [41.9,71.6] |
| High school/GED (% [95% CI]) | 52.5 [47.9,57.1] | 21.2 [17.2,25.8] | 38.4 [31.3,46.0] | 43.7 [37.2,50.5] | 9.3 [4.6,17.9] | 34.8 [27.7,42.5] | 31.6 [23.3,41.4] | 10.8 [6.1,18.5] | 25.6 [15.9,38.5] |
| Vocational/some college (% [95% CI]) | 20.1 [16.8,23.8] | 37.9 [34.4,41.5] | 26.7 [21.4,32.7] | 24.0 [18.5,30.4] | 39.1 [30.7,48.2] | 24.6 [17.2,33.8] | 12.5 [8.3,18.4] | 53.1 [37.5,68.1] | 12.7 [6.9,22.3] |
| College graduate (% [95% CI]) | 3.2 [1.9,5.1] | 40.7 [35.8,45.8] | 18.4 [12.3,26.4] | 6.8 [3.9,11.6] | 50.0 [41.3,58.7] | 10.9 [6.7,17.1] | 1.7 [.9,3.2] | 35.1 [23.0,49.6] | 4.3 [1.1,15.4] |
| Adolescent health ($M$ [$SE$]) | 2.31 (.03) | 2.05 (.03) | 2.15 (.05) | 2.23 (.05) | 2.16 (.10) | 2.06 (.08) | 2.26 (.07) | 2.09 (.11) | 2.46 (.10) |
| CESD Wave 3 ($M$ [$SE$]) | 4.89 (.18) | 4.39 (.10) | 4.52 (.27) | 5.41 (.25) | 4.92 (.39) | 5.03 (.40) | 5.37 (.34) | 5.49 (.51) | 6.17 (.72) |
| Health behaviors Wave 3 ($M$ [$SE$]) | 5.92 (.07) | 5.33 (.07) | 5.58 (.14) | 5.52 (.12) | 5.48 (.24) | 5.29 (.18) | 5.25 (.15) | 4.93 (.28) | 5.26 (.16) |
| Financial stress Wave 3 ($M$ [$SE$]) | .93 (.06) | .44 (.03) | .63 (.07) | .80 (.06) | .53 (.07) | .76 (.09) | .68 (.10) | .45 (.09) | .59 (.11) |
| CVD risk score ($M$ [$SE$]) | 9.74 (.28) | 7.78 (.21) | 8.62 (.43) | 10.45 (.35) | 9.11 (.47) | 10.47 (.69) | 8.83 (.40) | 7.60 (.30) | 7.77 (.62) |
| Current smoker (% [95% CI]) | 45.6 [41.7,49.5] | 32.5 [29.4,35.8] | 36.8 [31.9,42.0] | 22.1 [17.0,28.3] | 19.3 [12.3,29.1] | 25.0 [18.1,33.4] | 24.1 [18.8,30.4] | 20.5 [13.0,30.7] | 19.7 [12.2,30.3] |
| Systolic blood pressure, ($M$ [$SE$]) | 120.88 (.50) | 119.11 (.45) | 118.63 (.54) | 122.98 (.63) | 121.95 (1.33) | 122.54 (1.18) | 120.26 (.93) | 118.42 (1.22) | 118.45 (1.05) |
| Evidence of diabetes % (95% CI) | 5.5 [4.1,7.3] | 2.7 [1.9,3.7] | 4.8 [2.9,7.8] | 13.8 [10.7,17.7] | 8.6 [5.5,13.1] | 14.6 [9.9,20.1] | 7.6 [4.6,12.5] | 4.0 [1.5,10.2] | 6.8 [3.3,13.7] |
| BMI ($M$ [$SE$]) | 29.57 (.34) | 27.47 (.26) | 28.43 (.46) | 33.24 (.58) | 30.69 (.76) | 32.19 (.54) | 30.15 (.45) | 29.24 (.69) | 29.83 (1.06) |
| Antihypertensive medication (% [95% CI]) | 3.9 [2.7,5.5] | 1.6 [1.1,2.4] | 3.2 [1.8,5.7] | 4.3 [2.6,7.0] | 4.0 [1.9,8.0] | 4.1 [2.0,8.3] | .7 [.2,2.9] | 3.5 [1.2,9.7] | 2.8 [.7,10.6] |

*Note.* Household income is reported in thousands of dollars. Poverty is the proportion of families in the adolescent's census block group with incomes in 1989 that were below the poverty line. SES = socioeconomic status; GED = general equivalency diploma; CESD = Center for Epidemiologic Studies Depression Scale; CVD = cardiovascular disease; BMI = body mass index.

## Wave 3 Assessment

Financial stress was assessed by asking participants if in the last 12 months, they were evicted, did not pay the rent/mortgage, did not pay the full amount of bills, had service turned off by the gas or electric company, lacked service for their phone, could not afford to see their doctor, or could not afford to see their dentist. Individuals with a positive endorsement of any of the items were given a score of 1 for financial stress at Wave 3, whereas all others were given a score of 0. This measure was used to test for mediation between adolescent SES and maternal support to adult CVD risk to account for changes in SES over time while keeping the mediators separate from the time of the outcome measurement (Wave 4).

Depressive symptoms were assessed using nine items of the Center for Epidemiologic Studies Depression Scale (CESD), to which respondents answered that they had each symptom (0 = *never/rarely*, 1 = *sometimes*, 2 = *a lot of the time*, or 3 = *most of the time or all of the time*). These responses were summed to create a total CESD score out of 27.

Health behaviors at Wave 3 were measured by fast food consumption (0 = no days in last week, 1 = *1–4 days in last week*, 2 = *5–7 days in last week*), breakfast consumption (0 = *5–7 days in last week*, 1 = *1–4 days in last week*, 2 = *0 days in last week*), frequency of moderate physical activity (0 = *5–7 days in last week*, 1 = *1–4 days in last week*, 2 = *0 days in last week*), alcohol consumption frequency (0 = *did not drink in last year*, 1 = *drank less than weekly in last year*, 2 = *drank at least weekly in last year*), smoking (0 = *never smoked*, 1 = *smoked but not in last 30 days*, 2 = *smoked in last 30 days*), and sleep problems in the last 7 days (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 to every day when should have been awake*). Responses to each were summed for a total health behaviors score, ranging from 0 to 12, with a higher score indicating poorer health behaviors.

## Wave 4 Adult Assessment

CVD risk in young adulthood was assessed using a function that predicts the risk of developing CVD over a 30-year time frame (30-year Framingham CVD Risk Score [FRS]; Pencina et al., 2009). A prediction function takes account of the co-occurrence of CVD risk factors and their differing strength in relation to CVD and is therefore a better predictor of subsequent risk than individual measures alone. In addition, risk factors assessed in young adulthood have been shown to be equally good or better predictors of subsequent subclinical disease (Gidding et al., 2006; Loria et al., 2007) making the use of risk prediction among young adults a powerful tool to accurately predict subsequent risk of CVD. The 30-year FRS is the only prediction function that has been developed for use among young adults and has been shown to better predict overt and subclinical CVD compared to shorter term prediction functions (Berry et al., 2009; Laing et al., 2012; Pencina et al., 2009). The 30-year FRS tends to predict CVD risk more accurately for females than for males (Lloyd-Jones, 2010). The function used in this study is a Cox proportional hazards model that has been modified to account for competing causes of death (Pencina et al., 2009) which is essential for young adults who are much more likely to die from a non-CVD cause for most of the risk prediction window and ignoring competing causes of death results in an overestimation of longer term CVD risk (Pencina et al., 2009). Extensive details on the development and validation of the 30-year FRS is available in Pencina et al. (2009); essentially, the prediction function uses information on age, sex, body mass index (BMI), systolic blood pressure (SBP), use of antihypertensive medications, smoking and diabetic status to predict the risk of occurrence of a composite CVD outcome including coronary death, myocardial infarction, coronary insufficiency, angina pectoris, stroke, transient ischemic attack, intermittent claudication, and congestive heart failure (Pencina et al., 2009). Data elements of the prediction function were collected at wave 4 when the participants were between 24 and 34 years old ($M$ = 28.7 years). At this visit, research assistants collected data during in-home sessions using computer-assisted personal interviews and physical assessments. Standardized approaches to height and weight measurements were used (Entzel et al., 2009). Height was measured to the nearest 0.5 cm against a smooth wall without shoes or hats/hairpieces. Weight was measured to the nearest 0.1 kg using a Health-o-meter 844KL high-capacity digital bathroom scale (Jarden Corporation; Rye, NY) on a hard, flat surface. BMI was calculated according to the following formula: BMI ($kg/m^2$) = weight (kg)/height ($m^2$). SBP was measured using a Microlife BP3MC1-PC-IB oscillometric blood pressure monitor (MicroLife U.S.A., Inc.; Dunedin, FL). Participants were asked to stay seated for 5 min with legs uncrossed before and during the assessment, which was performed on the exposed right arm, absent contraindications (Entzel et al., 2009). Three assessments were taken at 30-s intervals. The latter two measures were averaged as the measure of resting SBP. Use of antihypertensive medication in the prior 4 weeks was assessed through a medication inventory (Tabor & Whitsel, 2010). Cigarette smoking in the 30 days preceding the interview was ascertained by self-report. Capillary whole blood collection was used to collect seven blood spots on a capillary whole blood collection card via a lancet. Participants responded about the last time they ate or drank anything, excluding water. Those who were fasting for 8 or more hours were considered fasting, and those who had eaten or drank in the past 8 hours were considered nonfasting. Diabetes mellitus was considered present if the respondent had: a fasting glucose ≥126 mg/dl, a nonfasting glucose ≥200 mg/dl, an HbA1c ≥ 6.5%, self-reported a health provider diagnosis of diabetes except during pregnancy, or used antidiabetic medication in the prior four weeks (Whitsel et al., 2012). Using this information, the prediction function calculates the risk (%) of developing CVD over the following 30 years. In this sample, mean risk was 13.0% (95% CI: 12.6 to 13.4). Men ($M$ = 17.0%; 95% CI: 16.5, 17.5) demonstrated significantly higher risk scores than females ($M$ = 8.8%; 95% CI: 8.5, 9.2).

## Data Analytic Plan

Using SAS 9.3 and SUDAAN 11.0, descriptive statistics were calculated and restricted cubic spline functions (Desquilbet & Mariotti, 2010) were used to examine the linear association of continuous variables (age, adolescent SES, adolescent maternal support, adolescent self-rated health, depressive symptoms, financial stress, and health behaviors) with CVD risk. All were found to be linear associations except for self-rated health, which closely approximated a linear association. Given the large sample size, the

variable was not transformed. Descriptive statistics of study variables were generated by sex, race/ethnicity, and socioeconomic status, dichotomized as high (at or above median) or low (below median). Using Mplus (Version 7), correlations among the study variables were examined—Pearson, biserial, and tetrachoric—depending on the nature of the variables (see Table 3). Three main models were tested to understand the relationship between maternal support, SES, and CVD risk: main effects, moderation, and mediation. The first model entailed using path analysis to simultaneously test the direct effects of maternal support and adolescent SES on CVD risk and adolescent SES on maternal support using maximum likelihood estimation with robust standard errors. These tests were adjusted for sex, age, race/ethnicity (dummy-coded: White, Black/African American, Hispanic), adolescent health status, and single mother in adolescence. The covariances of SES with age, race/ethnicity, single parenthood and adolescent health were modeled, and these covariances are reported in the Main Effects section and in Table 4.

In a second set of models, effect modification was examined to ascertain if the maternal support−CVD risk relationship differed by adolescent SES and race. This was done by incorporating a Maternal Support × Adolescent SES interaction term in the path model described in preceding text. Another model incorporated a Support × African American race term and a Support × Hispanic race term. Effect Modification × Sex and Modification × Single Motherhood in Adolescence was similarly examined.

A third model examined mediators of the adolescent maternal support−CVD risk and adolescent SES−CVD risk relationships. This test was conducted through the use of a path model, in which the direct effects of maternal support and adolescent SES on CVD risk were tested simultaneously with the relationship between adolescent SES and maternal support, adjusting for the covariates listed in preceding text. The estimated indirect effect of adolescent SES on CVD risk relationship through adolescent maternal support was examined using delta method standard errors to compute the indirect effect. In the same model, tests for mediation were conducted using the same method to examine whether the relations between adolescent maternal support (Wave 1) and CVD risk (Wave 4) and between SES and CVD risk were mediated by depressive symptoms, health behaviors, and financial stress at Wave 3. This final model was conducted to test whether maternal support mediated the SES-CVD risk association and to examine potential mediators between SES and maternal support at Wave 1 and CVD risk at Wave 4. All analyses incorporated survey design and unequal probability of selection per Add Health user guidance (Chantala & Tabor, 2010). Maximum likelihood with robust standard errors was used to deal with missing data, which was between 0.0% and 5.0% for all variables except for adolescent SES, Wave 3 CESD, Wave 3 financial stress, and Wave 3 health behaviors, which had 14.8%, 16.6%, 16.7%, and 16.9% missing, respectively. Alpha was set at 0.05 for determinations of statistical significance.

## Results

### Main Effects Model

**Analysis of covariates.** First, potential covariates in the model were evaluated. Maternal support and adolescent SES were not related ($B = 0.00$, 95% CI: −0.02, 0.03, $p = .75$). CVD risk was positively associated with age ($B = 1.06$, 95% CI: 0.95, 1.16, $p < .001$) and adolescent health problems ($B = 1.19$, 95% CI: 0.96, 1.42, $p < .001$) but was negatively associated with female gender ($B = -8.27$, 95% CI: −8.70, −7.84, $p < .001$). Higher CVD risk was associated with African American race ($B = 0.75$, 95% CI: 0.19, 1.31, $p < .01$) and lower CVD risk with Hispanic race ($B = -0.62$, 95% CI: −1.23, −0.0, $p = .05$). CVD risk was not related to single motherhood ($B = 0.0$, 95% CI: −0.46, 0.46, $p = .99$).

Maternal support was associated with age ($B = -0.03$, 95% CI: −0.04, −0.02, $p < .001$), gender ($B = -0.06$, 95% CI: −0.09, −0.03, $p < .001$), single parenting status ($B = -0.07$, 95% CI: −0.10, −0.03, $p < .001$), and perceived health problems in adolescence ($B = -0.07$, 95% CI: −0.09, −0.06, $p < .001$) but was not related to African American race ($B = 0.02$, 95% CI: −0.02, 0.05, $p = .36$) or Hispanic race ($B = 0.03$, 95% CI: −0.01, 0.07, $p = .16$). Age, female gender, parenting by a single mother, and perceived health problems were all negatively associated with perceived maternal support.

Table 3
*Correlation Matrix Computed by Mplus Version 7*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Age | — | | | | | | | | | | | | | |
| 2. Adolescent SES | −.02 | — | | | | | | | | | | | | |
| 3. African American | −.03 | −.30 | — | | | | | | | | | | | |
| 4. Hispanic | .12 | −.35 | −.85 | — | | | | | | | | | | |
| 5. Wave 4 CVD risk score | .24 | −.11 | .04 | .02 | — | | | | | | | | | |
| 6. Wave 1 single mother | −.01 | −.33 | .47 | −.01 | .03 | — | | | | | | | | |
| 7. Wave 1 poor adolescent health | .00 | −.11 | −.02 | .07 | .09 | .06 | — | | | | | | | |
| 8. Wave 1 maternal support | −.10 | .03 | −.01 | .02 | −.04 | −.08 | −.16 | — | | | | | | |
| 9. Wave 3 depressive symptoms | −.02 | −.09 | .08 | .06 | .02 | .09 | .14 | −.16 | — | | | | | |
| 10. Wave 3 poor health behaviors | −.01 | −.06 | −.04 | −.09 | .18 | .04 | .11 | −.13 | .15 | — | | | | |
| 11. Wave 3 financial stress | .05 | −.15 | .07 | −.05 | .08 | .12 | .11 | −.16 | .23 | .20 | — | | | |
| 12. Wave 1 parent education | −.08 | .72 | .06 | −.41 | −.09 | −.08 | −.11 | .04 | −.09 | −.05 | −.09 | — | | |
| 13. Wave 1 household income | .01 | .72 | −.31 | −.27 | −.07 | −.71 | −.06 | .02 | −.06 | −.06 | −.12 | .29 | — | |
| 14. Wave 1 poverty | −.02 | −.70 | .42 | .10 | .07 | .27 | .05 | .00 | .05 | .02 | .10 | −.23 | −.24 | — |
| 15. Female | −.07 | −.02 | .07 | −.01 | −.63 | .06 | .12 | −.08 | .13 | −.01 | .08 | −.03 | .00 | .04 |

*Note.* SES = socioeconomic status; CVD = cardiovascular disease.

Table 4
*Weighted Linear Model Results for Independent Variables on CVD Risk (N = 11,013)*

| Variable | *B* | SE | β | *p* |
|---|---|---|---|---|
| CVD risk | | | | |
| Adolescent maternal Support | −.56 | .18 | −.03 | <.01 |
| Adolescent SES | −1.33 | .17 | −.10 | <.01 |
| Age | 1.06 | .05 | .22 | <.01 |
| Female | −8.27 | .22 | −.48 | <.01 |
| Single mother | .00 | .23 | .00 | .99 |
| African American | .75 | .29 | .03 | <.01 |
| Hispanic | −.62 | .31 | −.02 | .05 |
| Adolescent health | 1.19 | .12 | .12 | <.01 |
| Maternal support | | | | |
| Adolescent SES | .00 | .01 | .01 | .75 |
| Age | −.03 | .00 | −.12 | <.01 |
| Female | −.06 | .01 | −.06 | <.01 |
| Single mother | −.07 | .02 | −.06 | <.01 |
| African American | .02 | .02 | .01 | .36 |
| Hispanic | .03 | .02 | .02 | .16 |
| Adolescent health | −.07 | .01 | −.13 | <.01 |
| Adolescent SES | | | | |
| Age | .02 | .04 | .02 | .58 |
| Female | .01 | .01 | .02 | .15 |
| Single mother | −.05 | .01 | −.17 | <.01 |
| African American | −.07 | .01 | −.28 | <.01 |
| Hispanic | −.06 | .01 | −.25 | <.01 |
| Adolescent health | −.07 | .01 | −.11 | <.01 |

*Note.* Race variables are dummy variables comparing the group of interest with White participants. CVD = cardiovascular disease; SES = socioeconomic status.

Lower SES in adolescence was associated with poorer perceived health in adolescence ($B = -0.07$, 95% CI: −0.08, −0.05, $p < .001$) and with parenting by a single mother ($B = -0.05$, 95% CI: −0.06, −0.04, $p < .001$). However, SES in adolescence was not associated with age ($B = 0.02$, 95% CI: −0.05, 0.10, $p = .58$) or sex ($B = 0.01$, 95% CI: −0.00, 0.02, $p = .15$). SES in adolescence was negatively related to both African American race ($B = -0.07$, 95% CI: −0.09, −0.04, $p < .001$) and Hispanic race ($B = -0.06$, 95% CI: −0.08, −0.03, $p < .001$).

**Main effects.** Controlling for the covariates above, the main effects model revealed a significant effect of maternal support on adult CVD risk ($B = -0.56$, 95% CI: −0.91, −0.20, $p < .01$; see Table 4), such that greater maternal support during adolescence is associated with decreased CVD risk in young adulthood. In addition, lower SES during adolescence was related to higher CVD risk in adulthood ($B = -1.33$, 95% CI: −1.66, −0.99, $p < .001$).

## Moderation Models

There was no interaction between adolescent maternal support and SES in predicting adult CVD risk ($B = 0.18$, 95% CI: −0.48, 0.85, $p = .59$), suggesting that the effect of maternal support is similar at all levels of SES. However, there was an interaction with African American race, such that African American adolescents were more sensitive to the effect of maternal support on CVD risk ($B = -0.90$, 95% CI: −1.56, −0.25, $p < .01$), but there was no interaction of maternal support with Hispanic race ($B = 0.23$, 95% CI: −0.79, 1.24, $p = .66$). The test for Moderation × Sex was not significant ($B = 0.80$, 95% CI: −0.05, 1.66, $p = .07$), and the test for Moderation × Single Mother was similarly nonsignificant ($B = -0.38$, 95% CI: −1.17, 0.41, $p = .35$).

## Mediation Models

After adding depressive symptoms, financial stress, and health behaviors at Wave 3 as mediators to the full model with covariates, the direct association between maternal support and CVD risk is nonsignificant ($B = -0.19$, 95% CI: −0.55, 0.17, $p = .30$). First, the associations between adolescent maternal support and the mediators were tested. Lower adolescent maternal support was related to poorer health behaviors at Wave 3 ($B = -0.51$, CI: −0.63, −0.39, $p < .001$), higher Wave 3 depressive symptoms ($B = -1.23$, CI: −1.42, −1.03, $p < .001$), and higher Wave 3 financial stress ($B = -0.27$, CI: −0.35, −0.19, $p < .001$). Second, the relationships between the mediators and the outcome, adult CVD risk, were tested. There were direct associations between poorer health behaviors at Wave 3 and higher adult CVD risk ($B = 0.62$, CI: 0.49, 0.75, $p < .001$), and between greater financial stress and higher adult CVD risk ($B = 0.49$, CI: 0.18, 0.80, $p < .01$) but not between depressive symptoms and CVD risk ($B = 0.03$, 95% CI: −0.03, 0.09, $p = .35$). Overall, there was a significant total effect of lower maternal support on greater CVD risk ($B = -0.67$, CI: −1.03, −0.32, $p < .001$). Finally, each of the specific pathways was tested to examine whether they significantly mediated the maternal support-CVD risk association. There were specific indirect effects between maternal support and CVD through Wave 3 health behaviors ($B = -0.32$, CI: −0.43, −0.21, $p < .001$) and Wave 3 financial stress ($B = -0.13$, CI: −0.23, −0.03, $p < .01$) but not depressive symptoms ($B = -0.04$, 95% CI: −0.11, 0.04, $p = .35$).

There was a significant total effect of adolescent SES on CVD risk through the mediators of health behaviors, financial stress, and depressive symptoms ($B = -1.26$, CI: −1.64, −0.89, $p < .001$). Higher SES was associated with better health behaviors at Wave 3 ($B = -0.21$, CI: −0.32, −0.09, $p < .001$), lower depressive symptoms ($B = -0.28$, CI: −0.47, −0.09, $p < .01$), and lower financial stress ($B = -0.20$, CI: −0.26, −0.14, $p < .001$). Associations between mediators and CVD risk are described above. Similar to the maternal support-CVD risk analyses, there was significant mediation between SES and CVD risk through health behaviors ($B = -0.13$, CI: −0.20, −0.05, $p = .001$) and financial stress ($B = -0.10$, CI: −0.16, −0.04, $p < .01$) but not through depressive symptoms ($B = -0.01$, 95% CI: −0.03, 0.01, $p = .37$). However, examining maternal support as a mediator between adolescent SES and adult CVD risk yielded no significant indirect effect of SES on CVD risk through maternal support ($B = 0.00$, 95% CI: −0.00, 0.01, $p = .66$).

## Discussion

Our results indicated a significant main effect of adolescent maternal support on CVD risk, with greater maternal support linked to lower CVD risk scores in young adulthood. However, neither the moderation nor mediation models of maternal support and SES were shown to be predictive of adult CVD risk. Contrary to hypotheses, the interaction between maternal support and ado-

lescent SES was not significant. As a result, greater maternal support may reduce risk for CVD for both lower and higher levels of SES. However, there was a significant interaction between African American race and maternal sensitivity such that African American adolescents were more sensitive to the effects of maternal sensitivity on CVD risk. This may be because of differences in the interpretation of maternal support across cultures. For example, African American adolescents may place greater meaning on the quality of their relationship with their mothers, which may make them more sensitive to variations in maternal support. Likewise, having a more positive relationship with the mother may be protective against stressors associated with minority status, while a more negative relationship may confer additional risk.

There was a main effect of adolescent maternal support on adult CVD risk such that lower perceived support was related to greater CVD risk. However, this effect became nonsignificant when mediators at Wave 3, including health behaviors and financial stress, were added. The data suggest that lower adolescent maternal support is associated with poorer health behaviors and greater financial stress over time, which is then related to higher CVD risk in young adulthood. A less supportive maternal relationship may lead to other psychological outcomes such as lower self-esteem and greater helplessness, which may translate into a poorer diet and less exercise as well as financial difficulties. Research is needed to clarify what psychological factors may mediate the association between maternal support and poorer health behaviors and greater financial stress. Further, depressive symptoms, though significantly related to maternal support, were not a mediator of the maternal support-CVD risk pathway. It appears that health behaviors and financial stress may be stronger mediators of the association at this age. It could be that depressive symptoms are more transient in young adulthood while health behaviors and SES may be more stable. Depressive symptoms and clinical depression may become more strongly associated with CVD and cardiovascular risk factors in middle and late adulthood when individuals are more likely to develop CVD. Similarly, the SES-CVD risk association was also mediated through health behaviors and financial distress, but not depressive symptoms at Wave 3. SES may be associated with continuing financial difficulties in young adulthood as well as the emergence of poorer health behaviors that increase CVD risk.

There was no evidence of mediation of SES effects on CVD risk through maternal support as there is no association between SES and maternal support during adolescence in the main effects model, adjusting for confounders. It may be that although differences in parenting have been reported across SES groups, the perception of maternal support by the adolescent and parent is not a function of SES. In addition, this is a measure of maternal support, which may differ from overall parenting measures that have been examined in the past, and associations between SES and maternal support may be different in childhood versus adolescence, when this sample was assessed. Further research is needed to understand associations between SES, perceived maternal support, and actual parenting behaviors during adolescence.

Low SES in adolescence may decrease opportunities for medical care and health education and increase the likelihood for negative health behaviors such as poor eating and lack of exercise (Adler & Newman, 2002). Thus, there are many pathways between low SES and the development of CVD risk factors that do not involve maternal support. These findings add to the existing literature by testing possible pathways through which variations in maternal support during adolescence may contribute to long-term CVD risk, which revealed evidence for a main effect of lower maternal support on higher CVD risk that operates through poorer health behaviors and greater financial stress.

These findings differ from those of other studies that report an interaction between childhood SES and maternal support in predicting physical health outcomes, which demonstrate the largest effect of maternal warmth and nurturance for the group with the lowest SES (e.g., Miller et al., 2011). This may be due to differences in the timing of SES measurement (childhood vs. adolescence), timing of outcome (young vs. middle adulthood), and the type of outcome (CVD risk vs. metabolic syndrome). It could be that adolescence is a particularly salient time for social relationships across SES groups, which impacts individuals into adulthood. The finding may also be specific to CVD risk, such that other outcomes (e.g., metabolic syndrome or psychopathology) demonstrate an interaction although CVD risk does not. The relationship between maternal support and CVD risk may change over time, with all groups equally affected in early adulthood but lower SES groups showing larger effects of maternal support over time.

Contrary to hypotheses, interventions that aim to improve parenting or adolescents' perceptions of parenting may not have stronger effects if they target individuals from more disadvantaged backgrounds. Instead, these types of interventions may be disseminated widely as they may result in lower risk of CVD at all SES levels. The current findings corroborate evidence of CVD risk disparities by race and SES even in young adulthood using a large nationally representative sample. Additionally, our results suggest that there may be differences in sensitivity to maternal support by race as African American adolescents showed the greatest variations in CVD risk by maternal support. This finding could indicate that interventions that improve maternal support may have the largest impact for African American adolescents, which is especially important considering this group is at greater risk for developing CVD. Future research should seek to understand relations between maternal support and CVD risk within and across cultures as differences in parenting have been reported across ethnic groups (Brooks-Gunn & Markman, 2005). It is important to establish which cardioprotective factors are particularly salient in the parent–child relationship within various groups to increase intervention efficacy for all ethnic and socioeconomic groups. Although the effect size of maternal support is small compared with other factors such as gender, age, and race, it is still clinically meaningful when considering the growing prevalence and cost of CVD.

This study is not without limitations. First, there were no objective measures of maternal support obtained, so it is unclear whether adolescents' perceptions of maternal support were an accurate reflection of the relationship. Second, perceptions of maternal support earlier in childhood were not obtained, and as a result, it is difficult to know whether perceptions during adolescence are specifically associated with CVD risk. It could be that adolescent perceptions are an artifact of childhood perceptions and that childhood is the most important period for parental buffering against adult CVD risk. However, the present assessment period in adolescence is an improvement over studies relying on retrospective reports in adulthood. In addition, adolescent perceptions of the

maternal relationship are likely highly related to adult perceptions, so it could be that adult perceptions are equally or more important in predicting CVD risk. However, maternal support is very different during adolescence than it is during the late 20s because of increased independence and decreased reliance on the mother, so adult perceptions of maternal support were not included at the time of the outcome measurement. Future studies must try to disentangle effects across childhood, adolescence, and adulthood to understand what periods are most highly related to CVD risk. Third, perceptions of paternal support were not examined as there were significantly fewer father figures than mothers in this study, but future research should incorporate reports of paternal support. Fourth, the risk prediction function used in this study was originally developed on the Framingham Offspring Cohort, which was initiated in 1971 (Kannel et al., 1979). Since that time, the underlying risk of disease and the prevalence of some risk factors have changed. Validation of this function in more contemporary cohorts has not yet been done. The use of this prediction function, however, remains an improvement over assessment of individual factors and further research is needed to fine-tune the functions for contemporary young adults. Finally, as these participants were in their late 20s and early 30s during assessment, they were examined for CVD risk rather than actual disease. However, young adulthood is an important time for setting up lifelong health and behavior, and risk factors assessed in this time frame have been shown to predict subsequent subclinical disease equally or better than assessments later in adulthood (Gidding et al., 2006; Loria et al., 2007) and risk prediction was used to improve the assessment of CVD risk. Therefore, the associations examined in this manuscript were examined at valid and important time frames and demonstrate the importance of early life parenting to downstream risk of CVD.

The current study provides evidence that both the adolescent's socioeconomic and psychosocial environments affect CVD risk. Maternal support during adolescence serves as a risk factor for future CVD if negative and a protective factor if positive, regardless of SES. Increased attention should be paid to psychosocial factors such as parenting, especially during sensitive periods for later development such as adolescence as this study provides evidence of a protective effect of positive perceptions of the maternal relationship. These results suggest that interventions that improve perceptions of maternal support may also be cardioprotective.



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