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CHILDHOOD ADVERSITY AND CARDIOVASCULAR DISEASE RISK: AN APPRAISAL OF RECALL METHODS WITH A FOCUS ON STRESS-BUFFERING PROCESSES IN CHILDHOOD AND ADULTHOOD

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1. Introduction

Numerous social science studies have indicated that exposure to childhood adversity is a significant risk factor for developing chronic illnesses, including cardiovascular diseases (CVD) in adulthood (e.g., Doom, Mason, Suglia, & Clark, 2017; Jimenez et al., 2019; Lei, Beach, & Simons, 2018). Conditions of financial hardship, food insecurity, harsh parenting, neighborhood violence, and racial discrimination are more frequently encountered by African Americans than whites and other racial and ethnic groups (Lei et al., 2019; Simons et al., 2019). These conditions combine to form a context of adversity that connotes danger, uncertainty, and threat, which triggers physiological stress responses in multiple bodily systems (Alessie, Angelini, van den Berg, Mierau, & Viluma, 2019; Johnson & Acabchuk, 2018). Research increasingly finds that, among African Americans, childhood adversity

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Authors' Contributions

MKL led design and analysis, participated in the construction of measures, and drafted the manuscript; MTB participated in the design of the study and drafting of the manuscript; RLS, LGS, and SRHB conceived of the study and made substantive contributions to the manuscript regarding interpretation of findings.

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predicts the onset and progression of CVD, metabolic syndrome, premature mortality, and type II diabetes, even after adjusting for adult lifestyle factors.

The vast majority of these investigations are based on retrospective measures obtained from adults concurrently with reports of their current health burden (Suglia et al., 2018). Retrospective measures of childhood adversities are vulnerable to the limitations of memory and selective recall biases, which likely undermine their accuracy and validity (Hardt & Rutter, 2004). Further, perhaps even more problematic, retrospective reports over long periods of time are vulnerable to possibility that one's current health distorts their recollections of early childhood. How an adult perceives or reports adverse childhood is thought to be interpreted through the perspective of their current health burden. Of course, prospective measures, collected closer to or at the time that the events occurred, also suffer certain limitations of their own. Still, some social scientists have privileged prospective measurement while casting strong doubts on the utility of retrospective measures to study long-term health outcomes (Scott & Alwin, 1998).

Given the limitations of each measurement strategy, there is need for research that examines the correlation between the two measures and the extent to which they separately predict CVD assessed in adulthood. Baldwin, Reuben, Newbury, and Danese's (2019) recent meta-analysis reported generally low agreement between retrospective and prospective measures of childhood maltreatment. They noted, however, that some of weak agreement might result from systematic differences in the information obtained from each measure. Specifically, most comparison studies investigate retrospective and prospective measures obtained from different study informants, whether caseworkers, respondents, or caretakers (see Baldwin et al., 2019; e.g., Reuben et al., 2016). Further, most studies compare prospective and retrospective measures that include related but non-overlapping forms of adversities (see Hardt & Rutter, 2004, p. 269). Thus, the measurement tools often capture content that varies to some extent, thereby making clear comparisons difficult. To better understand the scope and implications of the apparent divergence between the two measures it is critically important to compare overlapping adversities obtained from the same respondent but measured at different developmental periods. Such comparisons with a corresponding focus on objective health biomarkers are exceedingly rare in the health sciences literature (e.g., Offer, Kaiz, Howard, & Bennett, 2000) and, to the best of our knowledge, very rarely are they conducted with data from a prospective longitudinal community-based sample (Goltermann, Opel, & Dannlowski, 2019).

Indeed, given that limitations of these two approaches, it may be that each measure of childhood adversities makes an independent contribution to the prediction of CVD or that one approach is predictive of CVD whereas the other is not. Our study's first purpose is to (a) compare overlapping retrospective and prospective measures of childhood adversities reported by the same respondent, and (b) to examine their relative power to predict the Framingham CVD 30-year risk score.

Importantly, past research has reported that not all individuals who experience adversities in childhood will subsequently experience health problems in adulthood (Brody et al., 2014). The stress-buffering hypothesis asserts that social support reduces the negative effects of

toxic stress exposures on adult health outcomes (Cohen, 1988). In particular, research has consistently reported that parental emotional support has protective effects against poor physical health (Chen, Brody, & Miller, 2017). Yet, it is not clear whether theoretically predicted patterns of stress moderation will be different when using prospective versus retrospective measures of childhood adversity to test models of stress-buffering effects. The second purpose of our study is to examine the buffering effects of parental emotional support measured currently in childhood and in adulthood on CVD risk using both prospective and retrospective measures of childhood adversity. This second question allows us to determine if the hypothesized stress-buffering effects of parental support on CVD vary by retrospective and prospective measures of adversities and to test the stress-buffering process at different developmental periods.

1.1. The Impact of Retrospective and Prospective Measures of Childhood Adversity on Cardiovascular Risk

Scientific evidence of a linkage between adverse childhood experiences and reduced cardiovascular health is growing. Yet, the American Heart Association (AHA) recently released a scientific statement expressing caution about the conclusions of this research because most of the work is cross-sectional and based on retrospective reports of childhood life conditions (Suglia et al., 2018: e20). Given that retrospective measures ask participants to recall experiences that in many instances occurred years earlier, the AHA emphasized the possibility that retrospective tools may be contaminated by memory biases (Hardt & Rutter, 2004). If such contamination is evident, the positive association between childhood adversities and CVD would be artificially overstated.

Both prospective and retrospective assessment methods elicit information from respondents about events that have already occurred and thus they both involve some gradient of recall about past events (Scott & Alwin, 1998). Of course, prospective measures involve a shorter window of recall, often significantly shorter in the case of research on the long-term effects of childhood adversities, and they often ask about events or experiences currently or in the recent past. Because prospective measures typically assess events occurring in the recent past, they are presumably less subject to memory bias, and less likely to be contaminated by later adult outcomes. That is, they should be less susceptible to forgotten memories and recall biases, including the possibility that current adult health colors how the person perceives their childhoods (Hardt & Rutter, 2004) and mood congruency bias (Sheikh, 2018). Still, prospective measures are not without their own limitations. Specifically, children might not accurately interpret the intent of survey questions addressing traumatic or other contextual stressors. Further, children are often unaware of certain events (e.g., financial stress) that nonetheless affect them, and they may be reluctant to report some events, such as family violence, owing to fears about discovery or shame (Brewin, Andrews, & Gotlib 1993). In addition, children reporting on relatively recent events may be less likely to identify important patterns and may be influenced by current emotional context.

A small number of studies provide mixed conclusions about the reliability and validity of each data collection method. Most studies point to weak agreement among the measures and suggest retrospective and prospective measures cannot be used interchangeably. For

example, using court records, Widom and colleagues (1999) found that retrospectively recalled, but not prospectively monitored, childhood victimization was significantly associated with adult drug abuse pathology. A cohort study found that prospective maltreatment measures from official records predicted psychopathology but only if the maltreatment was recalled retrospectively by respondents (Newbury et al., 2018).

Some scholars have recently suggested that retrospective and prospective measures provide somewhat different but complementary information (Sheikh, 2018), although this work is based on informant reports of prospective items (e.g., Newbury et al., 2018). Baldwin et al. (2019) suggest the poor-to-fair agreement between retrospective and prospective measures of child maltreatment might result from systematic differences in the sensitivity of the measures to different types of maltreatment. As the authors note, a large proportion of studies that investigate the discordance between them evaluate prospective measures gathered entirely or partially from official reports (see Scott, McLaughlin, & Ellis, 2012). The different sources of information conceivably result in statistical discrepancies among the measures, which in some cases are significant. For instance, maltreatment reports from official records might capture more severe types of abuse, whereas retrospective reports "might detect more true cases" (Baldwin et al., 2019: 591). Furthermore, because the measures rely on separate informants with differing levels of exposure (e.g., caseworker, official records, and respondents) to the maltreatment in question this systematic design difference can result in low agreement between retrospective and prospective measures. As Widom (2019) notes, the measures may be discordant because "child abuse and neglect are socially constructed, poorly defined constructs" (p. 567). A study by Offer et al. (2000) compared the reports of 73 men, at age 14 and then again at age 48, to the same questions about aspects of their early-life upbringing (e.g., discipline, peer relations). The results indicated that their recollections changed substantially across measurement periods. The Offer et al. (2000) study, while based on a small sample, provides some evidence that reliance on the same measurement instrument does not substantially improve concordance between retrospective and prospective measures.

It is worth emphasizing the distinction between prospective and retrospective measurement in the context of longitudinal cohort studies of individuals. Prospective measures in longitudinal designs collect information on factors that may be relevant to the introduction of a treatment or the development of certain outcomes, say adult disease burden, often years before the outcome or disease manifests; whereas retrospective designs ask respondents to simultaneously recall information on events often occurring years before and to also report their current disease burden (Scott & Alwin, 1998). The windows of recall differ between the two survey designs in that retrospective measurement is based on reports of past events from the vantage point of present circumstances. Prospective measurement is common to prospective longitudinal designs, although retrospective measurement can also be incorporated in the same designs. Among the most important advantages of prospective measurement is the dual ability to specify properly temporal ordering and to control for the possibility of shared methods variance between the outcome and the independent variable.

Upon close inspection of the literature, however, only rarely do studies compare self-report items on overlapping childhood adversities gathered from the *same* respondents at different

time points in the lifespan, and even rarer still is research that compares their capacity to predict health outcomes. If part or all of the discordance arises from systemic measurement differences, then comparative studies would benefit from research using single informants who report on overlapping content. Thus, it is important to compare self-reported measures of childhood adversities, obtained from the same respondent but measured at different developmental periods to examine the agreement between prospective and retrospective reports and the extent to which each measure makes an independent contribution to the prediction of CVD. Just as Goltermann et al. (2019) noted, "further longitudinal studies applying identical instruments based on the identical source of information over differing times … are required to clearly delineate the extent of temporal stability and situational consistency of the assessed extent of maltreatment experiences" (p. el).

Unfortunately, the few data sets that include both prospective and retrospective measures of child adversity tend to use different time frames for the two reports. In the National Longitudinal Study of Adolescent to Adult Health (Add Health) study, for example, respondents between ages 24 and 32 were asked whether they had experienced physical abuse and neglect before the age of 18, whereas the prospective data capture reports of abuse and neglect collected between ages 15 and 16 (see Doom, Mason, Suglia, & Clark, 2017). The present study uses a data set that also suffers from this limitation but to a lesser extent. Specifically, we compare reports obtained at age 29 regarding child adversity prior to age 10, with age 10 reports of adversity occurring during the previous year. Thus, the time frames used for the two measurement approaches are similar but not identical. Nevertheless, it remains unclear whether both assessments from the same respondents and instrumentation method (i.e., self-reported) will influence objective health outcomes.

Research suggests that despite the disadvantages of retrospective measurement, there may be clinical and scientific value in both approaches to assessment and each may provide different windows into the burden of childhood adversity (See Baldwin et al., 2019; Widom 2019), often but certainly not always leading to similar conclusions. Other research has provided a more nuanced interpretation, which views the two measures as somewhat complimentary indicators of early life traumatic experiences (see Brewin et al., 1993; Hardt & Rutter, 2004; Reuben et al., 2016). Of interest here is whether the two approaches will lead to similar conclusions regarding stress-buffering interactions with supportive parenting during childhood.

Further, most previous studies of the impact of adversity on CVD utilize self-reported measures of cardiovascular disease as opposed to objective biomarkers. Indeed, a recent systematic review by Caceres et al. (2017: el 8) indicated that "a total of 23 of 26 studies that assessed CVD included self-report measures only." The associations reported in such research may be inflated due to *shared methods variance*, which arises from using self-reports to assess both the predictors and health outcomes (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003).

A way to overcome these methodological challenges is to test predictions using both prospective and retrospective measures of childhood adversity. Another strategy is to utilize scores from the Framingham algorithm to estimate an objective measure of adult CVD risk

(Pencina, D'Agostino, Larson, Massaro, & Vasan, 2009). This algorithm is based on seven metabolic syndrome and cardiovascular risk metrics that estimate the probability of a coronary heart disease event over the next 30 years. It has strong empirical validity and is used by clinicians to predict long-term risk of CVD outcomes in otherwise healthy populations, including young adults (Doom et al., 2017). Accordingly, this allows for tests of the hypothesis that long-term cardiovascular risk, calculated with the Framingham algorithm, similarly predicted by both prospective and retrospective measures of childhood adversity.

1.2. Childhood Adversity, Parental Emotional Support, and Cardiovascular Risk

Different measures of adversity might also produce different conclusions about how developmental processes interact to affect CVD. For instance, a wealth of research indicates that a significant number of individuals who experience adversity do not become ill or develop physiological impairment (Chen et al., 2017). In response to this finding, there has been increasing interest in identifying factors that protects and buffers against stress and adversities.

Several coping resources, defined as factors that ameliorate the impact of adverse conditions on health, have been suggested in the literature. One of the most frequently cited is parental emotional support. Such support is seen as ameliorating the deleterious impact of unpredictable, threatening, and frightening social experiences (Chen et al., 2017). Studies with African American youth have revealed, for example, that children who have higher levels of perceived parental emotional support tend to report less distress and health problems relative to those who receive lower levels of support (e.g., Brody et al., 2014). Consistent with this perspective, parental support has been shown to reduce the impact of child and adolescent stressors on inflammatory markers, and thereby reducing the probability of developing negative health outcomes following exposure to acute adversities (Carroll et al., 2013).

Furthermore, research suggests that parental emotional support received during adolescence has a more pronounced effect than parental support received during adulthood (Brody et al., 2014) and that receipt of such support can buffer against the effects of childhood maltreatment on health outcomes (Carroll et al., 2013; Chen et al., 2017). Yet, no published studies, to the best of our knowledge, have compared the buffering effects of parental emotional support in adolescence and adulthood across retrospective and prospective measures of childhood adversities. Thus, there is a need for studies that examine the timing of parental emotional support with regard to its buffering effect using both prospective and retrospective measures of childhood adversity.

Despite developing research in this area, there continues to be limited longitudinal research on childhood adversity, stress-buffering, and CVD risk among African Americans—a group that disproportionately experiences traumatic childhood experiences and elevated rates of CVD (Lei et al., 2018). Accordingly, a better understanding of childhood adversity as a possible determinant of CVD risk among African Americans, and the processes that may possibly buffer their deleterious physiological impact, could be of value to prevention programming.

The present study examines the extent to which parental support received in either adolescence or adulthood serves to buffer the effect of childhood adversity on CVD. Based upon arguments and findings from past research, we expect that adolescents will have the strongest effect. Specifically, we hypothesize that childhood adversity will have an increasingly stronger association with CVD risk for adults with low levels of adolescent parental emotional support, and, conversely, that strong parental support in adolescence will buffer the positive association between childhood adversity and elevated CVD risk. Our findings regarding this buffering effect will be especially compelling if this buffering effect is found across both prospective and retrospect reports of adversity.

2. Method

2.1. Participants

We tested hypotheses using data from the Family and Community Health Study (FACHS) (see Simons et al., 2019). The protocol and all study procedures were approved by the University of Georgia Institutional Review Board. At the first wave (1997–1998), the FACHS sample consists of 889 African American fifth-grade children (467 from Iowa and 422 from Georgia). The mean ages were 10.56 years (*SD* = .631; range 9–13). The sample had an average family per capita income of $6,956. Thirty-six percent of the families were below the poverty line, and 51% of the respondents identified as single parents. The second through sixth waves were collected between 1999 and 2012 to capture information when the target children were ages 12 to 13, 14 to 15, 17 to 18, 20 to 21, and 23 to 24, respectively.

Between 2014 and 2015, a Wave 7 of data collection was completed, one included blood draws. The mean age was 29 years. Given the logistics of scheduling home visits by phlebotomists, only members of the sample residing in Georgia, Iowa, or a contiguous state were identified as eligible. After also excluding persons who were deceased, incarcerated, or otherwise unreachable, we were left with a pool of 545 individuals, 470 (86%) of whom agreed to be interviewed and to provide blood. The outliers were defined by the 1.5 × interquartile range and were removed from the final analysis. After eliminating outliers, complete data were available for 454 respondents (173 men and 281 women). Rates of missing data ranged from 0.7% for married/cohabited to 5.7% for binge drinking. Analyses indicated that those individuals who did not participate in Wave 7 did not differ significantly from those who participated with regard to Wave 1 scores on sociodemographic and health-related covariates. Given this result, we assume that the data were missing at random. Therefore, missing values were handled by multiple imputation using the “MI” function of the STATA 15 software.

2.2. Measures

Cardiovascular risk.—Cardiovascular disease (CVD) risk was calculated following the gender-specific Framingham algorithm developed by Pencina and colleagues (2009). To estimate 30-year risk of CVD (coronary death, myocardial infarction, fatal or non-fatal stroke), the Framingham algorithm uses systolic blood pressure (SBP), body mass index (BMI), and diabetes, plus it adjusts for an individual’s age and gender, and whether the person currently smokes or take antihypertensive medication. Resting SBP was monitored

with Dinamap Pro 100 (Critikon; Tampa, FL) while the participants sat reading quietly. Three readings were taken every two minutes, and the average of the last two readings was used as the resting index. Mean SBP was 122.476 (*SD* = 16.291). Mean BMI (kg/m^2) was 31.564 (*SD* = 8.522). Finally, diabetes was defined as participants who used anti-diabetic medication or HbA1c > 6%. About 4% were classified as diabetic according to this metric. Using the Framingham algorithm, the predicted 30-year risk for CVD represents the probability of developing CVD within the next 30 years. At the time of the blood draw (age 29), the mean CVD risk of participants was 6.2%.

Childhood adversity.—Childhood adversity was measured by the 10-item short form of the Childhood Adversity Questionnaire. A *retrospective measure of childhood adversity* administered at Wave 7 (age 29) asks respondents to recall (1 = *yes*, 0 = *no*) whether they experienced each of 10 items of childhood adversities before the age of 10 years (e.g., prior to age 10, would you say … I don't have enough to eat at home). Then, to measure *childhood adversity prospectively*, we selected a set of 10 items that mostly closely resembled the retrospective measures. At Wave 1 (age 10), respondents were asked to report whether or not (1 = *yes*, 0 = *no*) they experienced a variety of negative events during the past year (e.g., my family did not have enough money to afford the kind of food we need; my family did not have enough money to afford the kind of clothing we need; my parents hit me with a belt, a paddle, or something else; there was a lot of murder and violence in my neighborhood). The precise wording of each question was shown in Table 1. Each item was summed to a total childhood adversity score ranging from 0 to 10. Cronbach's α was .633.

Parental emotional support.—Parental emotional support was measured prospectively and referred to the emotional support situation at the time of the interview. At Waves 1 and 7, respondents answered four questions regarding how often (1 = *never*, 4 = *always*) during the preceding year the primary caregiver engaged in various supportive and warm behaviors. Items include, "act supportive and understanding toward you", "let you know they really cares about you", "listen carefully to your point of view", and "tell you they loves you." Cronbach's αs = .782 and .830 at Waves 1 and 7.

Control variables.—To account for variables that could provide plausible rival explanations, we controlled for *gender* and health-related covariates at the last wave (age 29). Education was measured in years of education completed. Income was assessed by asking participants to report their income in the past year. This measure was log-transformed to reduce skew. Respondents reported their current relationship status with a romantic partner (1 = *married or cohabiting*, 0 = *others*), and the item asks respondents about their *health insurance* status during the previous year. *Sleep quality* was measured using the subjective item (1= *very bad*, 4 = *very good):* "During the past month, how would you rate your sleep quality overall?" *Healthy* diet was assessed using two items: (a) During the past seven days, how many times did you eat a whole piece of fruit (for example, an apple, orange, or banana) or drink a glass of 100% fruit juice (do not count punch, Kool-Aid, or sports drinks)? (b) During the past seven days, how many times did you eat vegetables like green salad, carrots, or potatoes (do not count French fries, fried potatoes, or potato chips)? The response categories ranged from 1 (*none*) to 6 (*more than once every day).* The

relationship the two items was significant ($r = .412$, $p < .001$). Scores were averaged to form the healthy diet variable. *Exercise* was measured with two items: (a) On how many of the past seven days did you exercise or participate in physical activity for at least 30 minutes that made you breathe hard such as running or riding a bicycle hard? and (b) On how many of the past seven days did you exercise or participate in physical activity for at least 30 minutes that did not make you breathe hard but was still exercise such as fast walking, slow bicycling, skating, pushing a lawn mower, or doing active household chores? The response categories ranged from 1 (*0 days*) to 5 (*all 7days*). These two items were correlated ($r = .580$, $p < .001$). Scores on the two items were averaged to form the exercise measure. *Depression* was assessed with a revised version of the University of Michigan Composite International Diagnostic Interview (Kessler & Mroczek, 1994). Respondents were asked to report (1 = yes, 0 = no) whether they experienced several symptoms of depression (e.g., "felt sad, empty, or depressed most of the day") for at least a two-week period in the past year. Cronbach's α was .850. *Binge drinking* was measured by asking the respondent (0 = *never*, 5 = *every day*) how often they had consumed more than three drinks of alcohol. Although current smoking is a part of the Framingham CVD risk, recent studies have shown that health behaviors (e.g., smoking, physical activity, diet, and alcohol consumption) are strongly associated with the Framingham CVD risk (Doom et al., 2017). Thus, all analyses controlled for previous smoking experience (former smokers and lifetime never smokers) to avoid over-interpreting the data.

2.3. Analytic Strategy

In all analyses, we utilized beta regression models (using the Stata 15) because our dependent variable, Framingham CVD risk, is a proportion ranging from zero to one. Beta regression models were introduced by Ferrari and Cribari-Neto (2004) and are useful when the dependent variable is bounded, or when it has a ceiling or floor effect, introducing a non-normal distribution for the dependent variable. Given that a beta regression model is a generalized linear model with a beta distribution and a logit link function, the estimated coefficients have a similar interpretation as in logistic regression. Thus, all results are presented as odds ratios, which represent the increase or decrease in the odds of CVD associated with a unit change of the independent variables.

We first used the intra-class correlation coefficient (ICC) to examine agreement between prospective and retrospective measures of childhood adversity. Unlike Pearson's correlation coefficient, the ICC reflects the degree of similarity and agreement between measurements. The values can range from zero to one, where one represents a complete agreement. A value of over .30 indicates fair agreement (Landis & Koch, 1977). Then, the analyses were performed separately for prospective and retrospective measures of childhood adversity. Variables were entered into the beta regression model in the following steps: (a) the main effect model, which was used to estimate the effects of childhood adversity, using either prospective or retrospective measures, on long-term CVD risk; (b) the main effect model with control variables, which tested the effects while controlling for health-related covariates; (c) the main effect model with a parental emotional support, which was used to test the effect of parental support on the dependent variable; (d) the stress-buffering model, which tested the interaction effects by adding the interaction between parental emotional

support and childhood adversity. To examine the time effect of parental emotional support, we included two sets of models. In the first set of models, parental emotional support at age 10 was used as the moderating variable and in the second set of models, parental emotional support at age 29 was used as the moderating variable.

To make coefficients easier to interpret, the parental emotional support was standardized (mean of 0 and *SD* of 1) before the interaction term was calculated. When interaction effects were present, we examined simple slope test and graphed the interaction with 95% error bars by plotting regression lines for childhood adversity at one standard deviation above and below the mean value of parental emotional support.

3. Results

3.1. Initial findings

Associations among study variables, with means and standard deviations, appear in Table 2. As expected, prospective and retrospective measures of childhood adversity were significantly associated with each other ($r = .243$, $p < .001$), and both were significantly associated with CVD risk ($r = .099$, $p = .034$; and $r = .170$, $p = .001$, respectively). Receipt of parental emotional support at age 10 was related to CVD risk ($r = -.121$, $p = .010$). Among the control variables, CVD risk was associated with most of the variables, including males, education, healthy diet, binge drinking, and cigarette use. Further, the intraclass correlation coefficient (ICC) was .351. According to the guidelines proposed by Landis and Koch (1977), there was fair agreement between prospective and retrospective measures.

3.2. Childhood adversity and cardiovascular risk

Table 3 shows the results of using beta regression analyses with a logit link function to examine the effect of childhood adversity on CVD risk. Model 1A shows that the effects of prospective measures of childhood adversity at age 10 was significantly associated with CVD risk ($e^b = 1.037$, $p = .008$). Model 2A added control variables. As hypothesized, the result remained significant and in the expected direction ($e^b = 1.048$, $p < .001$), suggesting that a unit increase in childhood adversity was related to a 4.8% increase in the odds of long-term CVD risk. Turning to the models using retrospective reports of childhood adversity prior to age 10, the pattern of results was parallel to the findings using prospective measures. Model 1B shows that childhood adversity was associated with CVD risk ($e^b = 1.050$, $p = .002$), and is also the case in Model 2B, even after adjusting for relevant control variables ($e^b = 1.059$, $p < .001$), suggesting that a unit increase in childhood adversity from 0 to 10 years was related to a 5.9% increase in the odds of long-term CVD risk.

3.3. Childhood adversity, parental emotional support at age 10, and cardiovascular risk

Table 4 presents the results from a series of beta regression models used to determine the effect of parental emotional support at age 10 on CVD risk. Beginning with the prospective measures of childhood adversity at age 10, Model 1A shows that the effect of childhood adversity was statistically significant ($e^b = 1.038$, $p = .013$), suggesting that a unit increase in childhood adversity was associated with a 3.8% increase in the odds of long-term CVD risk.

But, parental emotional support at age 10 was marginally significant and negative (e^b = .957, p = .059).

To address the hypothesized buffering effects of parental emotional support at age 10, Model 2A added the multiplicative interaction term by multiplying the prospective measure of childhood adversity by parental support as a predictor of long-term CVD risk. The analysis reveals the hypothesized interaction (e^b = .964, p = .005), which Figure 1 illustrates. High and low parental emotional support are defined as one standard deviation (SD) above the sample mean and one *SD* below the sample mean. About 34% of respondents in our sample scored above one *SD* on emotional support, and 13% of respondents scored below one *SD* from the mean on this moderator. As Figure 1 indicates, for those with high parental emotional support, the slope for the prospective effect of childhood adversity on long-term CVD risk is essentially zero (b = −.0001, p = .930), whereas for those reporting low emotional support, the slope is significant (b = .0043, p < .001). These findings suggest that childhood adversity is associated with higher long-term CVD risk for those with low levels of emotional support at age 10.

Turning to the retrospective measures of childhood adversity from 0 to 10 years, Models 1B and 2B in Table 4 show a pattern of results very similar to those just described for prospective measures. Model 1B shows that childhood adversity and parental emotional support have effects on long-term CVD risk (e^b = 1.056, p < .001; and e^b = .963, p = .094, respectively). Model 2B enters the interaction of childhood adversity (retrospective measure) × parental emotional support at age 10. The results reveal that there was a significant interaction of childhood adversity and parental emotional support in predicting long-term CVD risk (e^b = .970, p = .002), indicative of a stress-buffering effect. As shown in Figure 2, those with greater parental support showed no impact of childhood adversity on CVD risk (b = .0009, p = .425), whereas those with less parental emotional support at age 10 showed a significant, positive impact of childhood adversity on CVD risk (b = .0046, p < .001).

3.4. Childhood adversity, parental emotional support at age 29, and cardiovascular risk

Shifting the focus to parental emotional support in adulthood, we repeated the analyses using parental emotional support measured at age 29 (Wave 7). As can be seen in the Online Supplement Table 1, CVD risk is predicted by both prospective and retrospective measures of childhood adversity but not by parental emotional support at age 29. Further, Models 2A and 2B add the multiplicative interaction term formed by multiplying childhood adversity by parental emotional support at age 29. Yet, these interactions are not significant, suggesting that the effects of childhood adversity on long-term CVD risk are not buffered by parental emotional support during adulthood.

3.5. Sensitivity analysis

We performed a series of sensitivity analyses to examine the robustness of our results. First, given that current smoking is a part of CVD risk index, we removed smoking-related variables and re-estimated the models. The results are same as those shown in Table 3 (see Table S2). Second, it is possible that the childhood adversity and stress-buffering effects of parental emotional support on CVD are conditional on gender. We used the interaction of

gender with the independent variables to examine potential gender effects. These interaction effects were found to be non-significant, suggesting no gender difference in the effects of childhood adversity and/or parental emotional support on adult CVD risk (see Tables S3 and S4). Third, to ensure robustness of results (Sidi & Harel, 2018), we repeated all analyses using listwise deletion ($N = 404$). The results showed no change in overall effects (see Table S5). Finally, given that the distribution of parental emotion support is skewed, we re-estimated the models using a natural log transformation of those variables. The results are similar to those found in Table 4 (see Table S6).

4. Discussion

The AHA recently released a scientific statement indicating that there is compelling evidence that child and adolescent adversity are linked to CVD in adulthood (Suglia et al., 2018). Moreover, studies have found that this relationship may be particularly relevant for African Americans who are at increased risk for childhood/adolescent maltreatment and CVD (Lei et al., 2018; Simons et al., 2019). Consistent with this idea, the notion of biological embedding suggests early exposure to adverse experiences may "get under the skin," to increase physiological impairment in adulthood (Johnson & Acabchuk, 2018), especially African Americans (Brody et al., 2014). The findings of this study provide further evidence that, after considering measurement issues, childhood adversity might systematically alter health states across the lifespan.

Although the association between childhood adversity and CVD has been documented, studies cast some doubt on its validity and suggest prior findings may be tainted by biases arising from retrospective measures (Suglia et al., 2018). Toward this end, we examined both retrospective and prospective reports of childhood adversity and their associations with CVD in a longitudinal cohort study. For this study, the prospective measures were collected at age 10 and referred to events that occurred in the previous year, whereas the retrospective measures, collected at age 29, asked about experiences from 0 to 10 years. We observed statistically significant correlations between the retrospective and prospective measures of childhood adversities, with some types of adversities exhibiting stronger associations than others. Still, there was poor-to-fair agreement between items obtained from the two assessments. These findings are consistent with recent meta-analyses (Baldwin et al., 2019) reporting poor agreement between prospective and retrospective measures of childhood maltreatment and with past longitudinal cohort research (Henry, Moffitt, Caspi, Langley, & Silva, 1994); together, this work suggests measures from the different assessment tools may not be used interchangeably.

The current findings are important in part because the two assessments are collected from the same respondents and based on the same method (i.e., self-reports) and focused on closely overlapping adversities items. The results suggest that even when holding constant the informant, measurement method, and, to a great extent, the item content, the two measures demonstrate sizeable discordance. This pattern suggests the discordance observed in prior comparative studies is not necessarily a function of instrumentation and design differences between prospective and retrospective assessments (see also, Baldwin et al., 2019). Some of the low agreement we observed may occur because the two assessments

specify different recall windows. Still, the findings comport with past work on different informants and methods, which find that retrospective and prospective reports of childhood adversities do not closely converge (e.g., Widom 2019). Indeed, Colman et al. (2016) observed that a large fraction of respondents (39%) in a two-wave 12-year study gave discordant reports of exposures to childhood stressors.

Despite the low agreement between prospective and retrospective measures, as hypothesized, children who were reared in neglectful, turbulent, and hostile environments were at increased risk for CVD. Indeed, the retrospective and prospective measures were predictive of CVD risk net of controls for relevant lifestyle and dietary measures. This pattern of results suggests that although these two measures have their limitations and demonstrate low agreement, they may be complementary predictors of CVD in adulthood (Newbury et al., 2018). The predictive similarity of the two measures suggests the retrospective measures may have meaning as clinical indicators of CVD risk. As Widom (2019: 568) stresses, such findings do not dismiss the value of "listening to what a patient says but they suggest cautious should be used" in drawing connections between retrospective measures and current health conditions. Accordingly, more broadly, our findings provide evidence supporting a link between childhood adversity and adult health. Future research should consider the biopsychosocial mechanisms, especially inflammation and epigenetic aging, underlying this association.

In addition, we also examined the stress-buffering effects of parental emotional support on the predicted CVD risk in adulthood using both measures of childhood adversities. Chen et al. (2017) proposed that warm, supportive parenting provides the child a feeling of being loved and cared for within the context of a caregiver relationship, and thereby it might attenuate the potentially detrimental consequences of psychosocial stress. Consistent with the stress-buffering model (Cohen, 1988), we sought to determine whether emotionally supportive relationships with parents served to buffer the association between childhood adversity and later CVD. As hypothesized, we found results supporting the hypothesis where high levels of parental emotional support received during adolescence moderated the effect of childhood adversity on CVD risk. Further, it is noteworthy that positivity in the parent-child relationship in adolescence, but not in adulthood, was consequential for the adult cardiovascular response to traumatic stressors experienced years earlier. This finding may be because young adults have less contact with their parents than adolescents. Just as Simons et al. (2007) noted, "As children grow older, they dramatically decrease the amount of time that they spend with parents while increasing the time spent away from home with peers" (p. 482). It is also consistent with recent intervention research showing that supportive family environments during adolescence can buffer the effects of childhood adversity and maltreatment on health outcomes (Brody et al., 2014). Indeed, childhood adversity only had a significant positive association with adult CVD risk in the context of weak parental emotional support during adolescence. Moreover, these findings were evident across each measure of childhood adversities.

Altogether, the findings provide evidence that retrospective and prospective measures provide similar information about the stress-buffering process. The estimates derived from each method point to a similar set of conclusions regarding risk for CVD, which highlights

the apparent validity of the retrospective and prospective measures in the current data. Note that the current study is among the first to compare self-reported measures of childhood adversities from different methods that were gathered years apart and reported by the *same* respondent.

Limitations

Although we believe our findings advance the literature on childhood adversity and adult CVD, our study is not without shortcomings. First, our prospective measures were collected at age 10 (Wave 1) and asked about adversity experiences during the preceding year. Our retrospective measure, on the other hand, was collected at age 29 (Wave 7) and referred to adverse experiences prior to age 10. These measurement strategies reflect the two most common approaches for research on childhood adversity and health. Concerns about the differences between the lacks of complete overlap in the time periods assessed are mollified to *some degree* by the fact that childhood adversity tends to show continuity across childhood into adolescence (e.g., Hazel et al., 2008), which suggests that the age-10 reports of adversity might be considered to be rough indicators of adversity prior to age 10. Consonant with this idea, there was fair agreement between the prospective and retrospective measures of childhood adversity. Still, it is important that our results be replicated with samples that include prospective and retrospective measures based on the same time interval and is an important step for future work. Second, our data collection began when the respondents were approximately age 10. For prospective measures, this might not capture children who experience adversity only at earlier ages. In addition, past studies have shown strong effects of early socio-environmental adversity from infancy to around age five on adult physical health (Chen, Martin, & Matthews, 2007). Yet, methodologically it is difficult to collect reliable data from younger children because they may misunderstand and misinterpret the questions. Further, there is strong continuity in experiences with adversity across childhood (e.g., Duncan & Rodgers, 1988; Schoon et al., 2002), meaning that children who reported experiencing adversities around age 10 (Wave 1) were more than likely to have experienced them during earlier years relative to children who did not report adversities at age 10.

6. Conclusions

Taken together, our findings add to the literature by documenting the poor-to-fair agreement between prospective and retrospective measures, and by suggesting that children who are reared in adverse social environments have elevated risk of long-term cardiovascular disease. Moreover, the study also identified parental emotional support in adolescence, but not in adulthood, as a significant stress-buffering process. From an intervention standpoint, identifying such protective factors may help inform future interventions designed to reduce the deleterious impact of childhood adversity for CVD health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

Alessie RJ, Angelini V, van den Berg GJ, Mierau JO, & Viluma L. (2019). Economic conditions at birth and cardiovascular disease risk in adulthood: Evidence from post-1950 cohorts. Social Science & Medicine, 224, 77–84. [PubMed: 30769195]

Baldwin JR, Reuben A, Newbury JB, & Danese A. (2019). Agreement between prospective and retrospective measures of childhood maltreatment: a systematic review and meta-analysis. JAMA Psychiatry, 76, 584–593. [PubMed: 30892562]

Brewin CR, Andrews B, & Gotlib IH (1993). Psychopathology and early experience: a reappraisal of retrospective reports. Psychological Bulletin, 113, 82–98. [PubMed: 8426875]

Brody GH, Lei MK, Chae DH, Yu T, Kogan SM, & Beach SR (2014). Perceived discrimination among African American adolescents and allostatic load: A longitudinal analysis with buffering effects. Child Development, 85, 989–1002. [PubMed: 24673162]

Caceres BA, Brody A, Luscombe RE, Primiano JE, Marusca P, Sitts EM, & Chyun D. (2017). A systematic review of cardiovascular disease in sexual minorities. American Journal of Public Health, 107, e13–e21.

Carroll JE, Gruenewald TL, Taylor SE, Janicki-Deverts D, Matthews KA, & Seeman TE (2013). Childhood abuse, parental warmth, and adult multisystem biological risk in the Coronary Artery Risk Development in Young Adults study. Proceedings of the National Academy of Sciences, 110, 17149–17153.

Chen E, Martin AD, & Matthews KA (2007). Trajectories of socioeconomic status across children's lifetime predict health. Pediatrics, 120, e297–e303. [PubMed: 17606533]

Chen E, Brody GH, & Miller GE (2017). Childhood close family relationships and health. American Psychologist, 72, 555–566. [PubMed: 28880102]

Cohen S. (1988). Psychosocial models of the role of social support in the etiology of physical disease. Health Psychology, 7, 269–297. [PubMed: 3289916]

Colman I, Kingsbury M, Garad Y, Zeng Y, Naicker K, Patten S, … & Thompson AH (2016). Consistency in adult reporting of adverse childhood experiences. Psychological Medicine, 46, 543–549. [PubMed: 26511669]

Doom JR, Mason SM, Suglia SF, & Clark CJ (2017). Pathways between childhood/adolescent adversity, adolescent socioeconomic status, and long-term cardiovascular disease risk in young adulthood. Social Science & Medicine, 188, 166–175. [PubMed: 28747248]

Duncan GJ, & Rodgers WL (1988). Longitudinal aspects of childhood poverty. Journal of Marriage and the Family, 50, 1007–1021.

Ferrari S, & Cribari-Neto F. (2004). Beta regression for modelling rates and proportion. Journal of Applied Statistics, 31, 799–815.

Goltermann J, Opel N, & Dannlowski U. (2019). Considering the source of information in the evaluation of maltreatment experiences. JAMA Psychiatry, 76, 985–986.

Hardt J, & Rutter M. (2004). Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. Journal of Child Psychology and Psychiatry, 45, 260–273. [PubMed: 14982240]

Hazel NA, Hammen C, Brennan PA, & Najman J. (2008). Early childhood adversity and adolescent depression: the mediating role of continued stress. Psychological Medicine, 38, 581–589. [PubMed: 18261247]

Henry B, Moffitt TE, Caspi A, Langley J, & Silva PA (1994). On the" remembrance of things past": a longitudinal evaluation of the retrospective method. Psychological Assessment, 6, 92–101.

Johnson BT, & Acabchuk RL (2018). What are the keys to a longer, happier life? Answers from five decades of health psychology research. Social Science & Medicine, 196, 218–226. [PubMed: 29153315]

Kessler RC, & Mroczek D. (1994). Scoring of the UM-CIDIshort forms. Ann Arbor (MI): The University of Michigan Institute for Social Research/Survey Research Center.

Jimenez MP, Wellenius GA, Subramanian SV, Buka S, Eaton C, Gilman SE, & Loucks EB (2019). Longitudinal associations of neighborhood socioeconomic status with cardiovascular risk factors: A 46-year follow-up study. Social Science & Medicine, 241, 112574.

Landis JR, & Koch GG (1977). The measurement of observer agreement for categorical data. Biometrics, 33, 159–174. [PubMed: 843571]

Lei M-K, Beach SRH, & Simons RL (2018). Childhood trauma, pubertal timing, and cardiovascular risk in adulthood. Health Psychology, 37, 613–617. [PubMed: 29672100]

Lei MK, Simons RL, Beach SR, & Philibert RA (2019). Neighborhood disadvantage and biological aging: Using marginal structural models to assess the link between neighborhood census variables and epigenetic aging. The Journals of Gerontology: Series B, 74, e50–e59.

Newbury JB, Arseneault L, Moffitt TE, Caspi A, Danese A, Baldwin JR, & Fisher HL (2018). Measuring childhood maltreatment to predict early-adult psychopathology: Comparison of prospective informant-reports and retrospective self-reports. Journal of Psychiatric Research, 96, 57–64. [PubMed: 28965006]

Offer D, Kaiz M, Howard KI, & Bennett ES (2000). The altering of reported experiences. Journal of the American Academy of Child & Adolescent Psychiatry, 39, 735–742. [PubMed: 10846308]

Pencina MJ, D'Agostino RB, Larson MG, Massaro JM, & Vasan RS (2009). Predicting the 30-year risk of cardiovascular disease: the Framingham Heart Study. Circulation, 119, 3078–3084. [PubMed: 19506114]

Podsakoff PM, MacKenzie SB, Lee JY, & Podsakoff NP (2003). Common method biases in behavioral research: A critical review of the literature and recommended remedies. Journal of Applied Psychology, 88, 879–903. [PubMed: 14516251]

Reuben A, Moffitt TE, Caspi A, Belsky DW, Harrington H, Schroeder F, … & Danese A. (2016). Lest we forget: comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. Journal of Child Psychology and Psychiatry, 57, 1103–1112. [PubMed: 27647050]

Schoon I, Bynner J, Joshi H, Parsons S, Wiggins RD, & Sacker A. (2002). The influence of context, timing, and duration of risk experiences for the passage from childhood to midadulthood. Child Development, 73, 1486–1504. [PubMed: 12361314]

Scott KM, McLaughlin KA, Smith DA, & Ellis PM (2012). Childhood maltreatment and DSM-IV adult mental disorders: comparison of prospective and retrospective findings. The British Journal of Psychiatry, 200, 469–475. [PubMed: 22661679]

Scott J, & Alwin D. (1998). Retrospective versus prospective measurement of life histories in longitudinal research In Giele JZ & Elder GH (Eds.), Methods of life course research (pp. 98–127). Thousand Oaks, CA: Sage.

Sheikh MA (2018). Coloring of the past via respondent's current psychological state, mediation, and the association between childhood disadvantage and morbidity in adulthood. Journal of Psychiatric Research, 103, 173–180. [PubMed: 29883925]

Sidi Y, & Harel O. (2018). The treatment of incomplete data: Reporting, analysis, reproducibility, and replicability. Social Science & Medicine, 209, 169–173. [PubMed: 29807627]

Simons RL, Simons LG, Chen YF, Brody GH, & Lin KH (2007). Identifying the psychological factors that mediate the association between parenting practices and delinquency. Criminology, 45, 481–517.

Simons RL, Lei MK, Beach SR, Simons LG, Barr AB, Gibbons FX, & Philibert RA (2019). Testing Life Course Models Whereby Juvenile and Adult Adversity Combine to Influence Speed of Biological Aging. Journal of Health and Social Behavior, 60, 291–308. [PubMed: 31409156]

Suglia SF, Koenen KC, Boynton-Jarrett R, Chan PS, Clark CJ, Danese A, … & Pratt CA (2018). Childhood and adolescent adversity and cardiometabolic outcomes: a scientific statement from the American Heart Association. Circulation, 137, e15–e28. [PubMed: 29254928]

Widom CS (2019). Are retrospective self-reports accurate representations or existential recollections? JAMA Psychiatry, 76, 567–568. [PubMed: 30892563]

Widom CS, Weiler BL, & Cottler LB (1999). Childhood victimization and drug abuse: a comparison of prospective and retrospective findings. Journal of Consulting and Clinical Psychology, 67, 867–880. [PubMed: 10596509]

Research Highlights

- Cardiovascular risk was predicted by prospective measures of childhood adversity.
- Retrospective measures also reliably predicted cardiovascular risk.
- Both relationships were buffered by parental emotional support at age 10.
- Identifying buffering processes will guide intervention efforts.

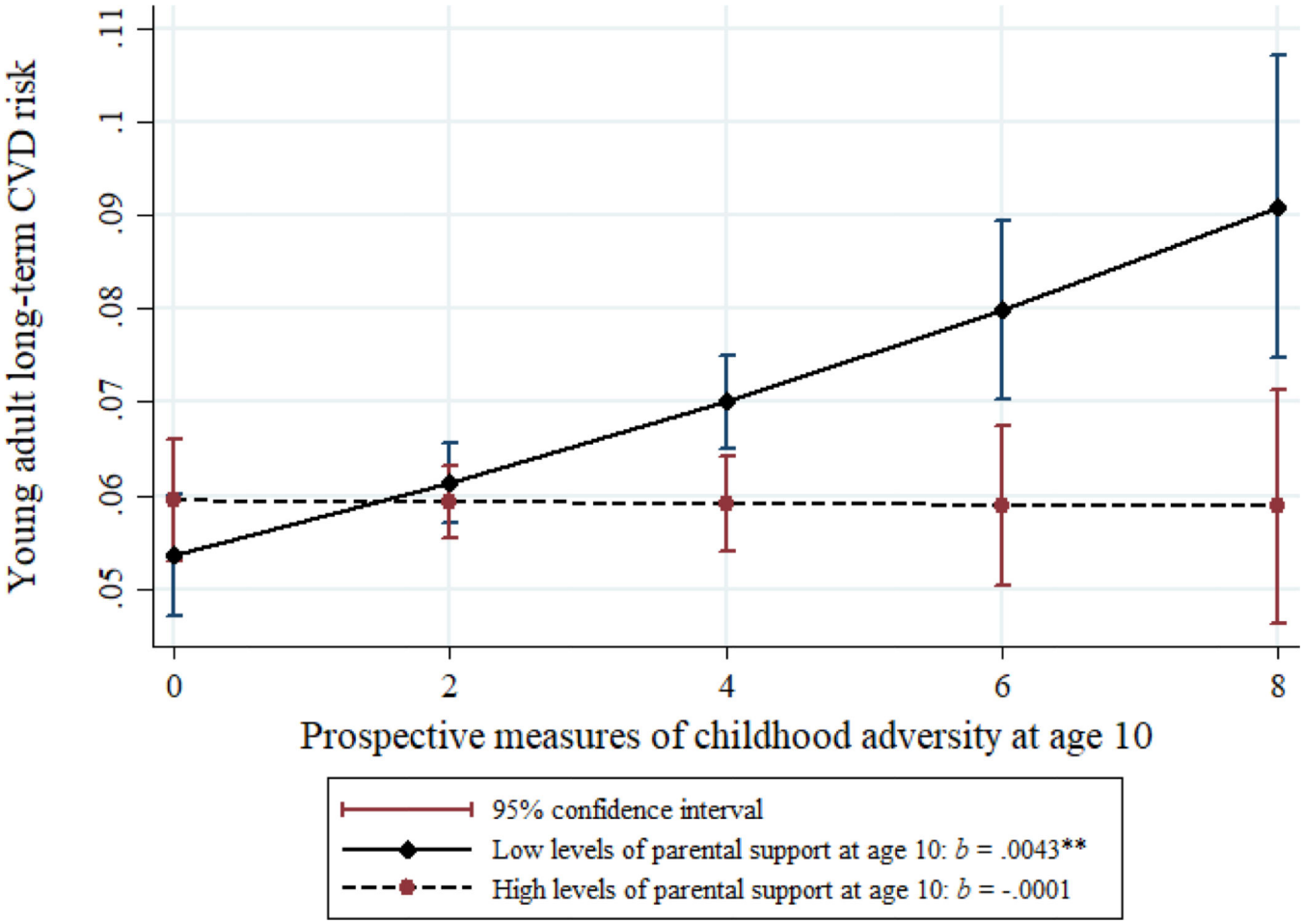

Fig. 1.

Prospective measures of childhood adversity on cardiovascular disease risk by levels of parental emotional support. The lines represent the regression lines for different levels of parental emotional support (low: one *SD* below the mean; high: one *SD* above the mean). Numbers in parentheses refer to simple slopes—significant slope only for those with low parental support.

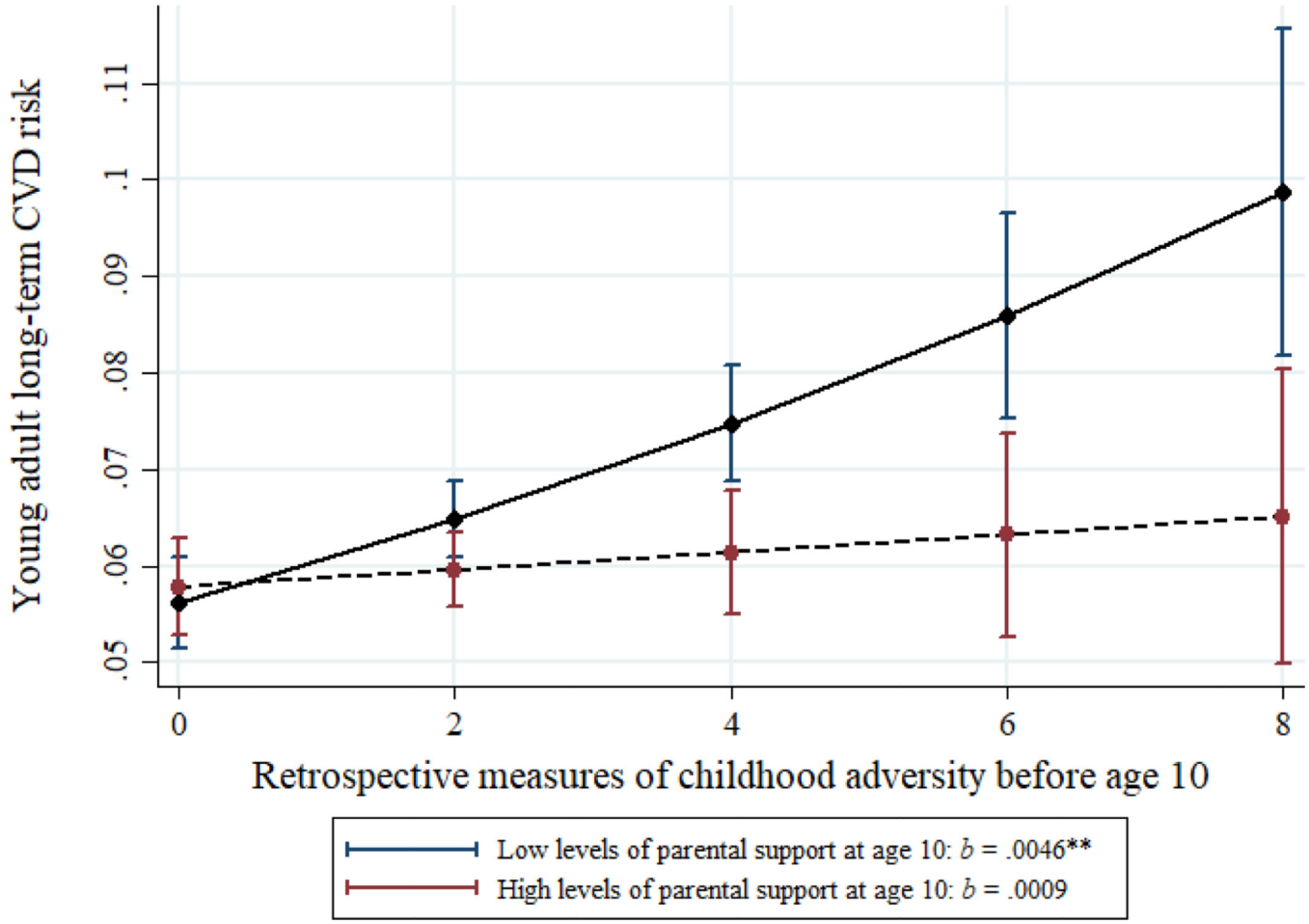

Fig. 2.

Retrospective measures of childhood adversity on cardiovascular disease risk by levels of parental emotional support. The lines represent the regression lines for different levels of parental emotional support (low: one *SD* below the mean; high: one *SD* above the mean). Numbers in parentheses refer to simple slopes—significant slope only for those with low parental support.

Table 1

Measures of childhood adversity across retrospective and prospective reports (N=454)

Items	Retrospective measures before age 10	Prospective measures at age 10	ICC
1	I didn't have enough to eat at home (*freq.* = 45, 9.9%)	My family did not have enough money to afford the kind of food we need (*freq.* = 18, 4.0%)	.249**
2	I had to wear old or dirty clothes or clothes that did not fit (*freq.* = 9, 2.0%)	My family did not have enough money to afford the kind of clothing we need (*freq.* = 37, 8.1%)	.185*
3	People in my family hit me so hard that it left me with bruises or marks (*freq.* = 18, 4.0%)	My parents slap or hit me (*freq.* = 94, 20.8%)	.151*
4	I was punished with a belt, a board, a cord, or some other hard object (*freq.* = 152, 33.4%)	My parents hit me with a belt, a paddle, or something else (*freq.* = 284, 62.4%)	.166*
5	There was a lot of violence in my neighborhood (*freq.* = 54, 11.9%)	There was a lot of murder and violence in my neighborhood (*freq.* = 89, 19.8%)	.241**
6	There was a lot of graffiti and run-down buildings in my neighborhood (*freq.* = 79, 17.4%)	In my neighborhood, there was graffiti on buildings and walls. (*freq.* = 124, 27.3%)	.176*
7	A family member was the victim of a crime (*freq.* = 37, 8.1%)	A close family member was a victim of a violent crime (*freq.* = 92, 20.4%)	.150*
8	Did your parents separate or divorce? (*freq.* = 174, 38.2%)	Did your parents separate or divorce? (*freq.* = 99, 22%)	.227**
9	Someone said something insulting to you just because of your race or ethnic background† (*freq.* = 157, 34.5%)	Someone said something insulting to you just because of your race or ethnic background† (*freq.* = 267, 62.2%)	.272**
10	Members of your family or close friends were treated unfairly because of their race or ethnic background † (*freq.* = 145, 31.9%)	Members of your family or close friends were treated unfairly because of their race or ethnic background† (*freq.* = 184, 44.3%)	.198*
Total			.351**

Note.

†Given that all participants were African Americans, we designed an item to focus on racism experiences.

Table 2

Correlations, Means, and Standard Deviations among the Study Variables (N = 454)

Variable or statistic	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Cardiovascular risk (Age 29)	—																
2. Childhood adversity (prospective)	.099*	—															
3. Childhood adversity (retrospective)	.170**	.243**	—														
4. Parental emotional support (Age 10)	−.121**	−.156**	−.087†	—													
5. Parental emotional support (Age 29)	−.047	−.066	−.105*	.070	—												
6. Males	.403**	−.008	−.016	−.032	.026	—											
7. Education (Age 29)	−.150**	−.075	.126**	.022	−.038	−.070	—										
8. Income (Age 29)	−.030	−.070	.152**	.038	−.033	.095*	.314**	—									
9. Married or cohabiting (Age 29)	.044	.006	.039	−.024	.066	.030	.078†	.117*	—								
10. Health insurance (Age 29)	−.059	−.108*	.059	−.001	−.051	−.134**	.227**	.161**	.050	—							
11. Sleep quality (Age 29)	.066	−.187**	−.271**	.055	.132**	.155**	−.100*	−.093*	.005	−.005	—						
12. Healthy diet (Age 29)	−.129**	.026	.064	.048	.014	−.170**	.168**	.005	−.015	.137**-	.087†	—					
13. Exercise (Age 29)	−.031	.056	.121*	−.059	.061	.189**	.127**	174**	.057	.052	.078	.149**	—				
14. Depression (Age 29)	.070	.079†	.398**	−.010	−.079†	−.153**	−.009	−.037	−.081†	.085†-	.313**	.042	−.024	—			
15. Binge drinking (Age 29)	.106*	.110*	.272**	−.055	−.036	.115*	.140**	175**	−.043	.043	118*	.016	.099*	.192**	—		
16. Former smokers	−.106*	.118*	.041	−.062	−.046	.101*	−.039	−.041	.016	−.026	.028	.098*	.121**	−.008	.066	—	
17. Lifetime never smokers	−.222**	−.146**	−.119*	.014	.029	−.087†	.214**	.120*	−.021	.035	052	.007	−.119*	−.128**	−173**	553**	—
Mean	.062	2.833	1.916	3.475	3.189	.381	13.068	21126.1	.268	.824	3.074	6.641	5.055	1.768	.840	.203	.546
SD	.048	1.651	1.836	.568	.969	.486	1.737	16528.8	.443	.381	.871	2.429	2.310	2.259	1.174	.402	.498

† $p \leq .10$,

* $p \leq .05$,

** $p \leq .01$ (two-tailed tests).

Table 3

Beta regression models of the results of childhood adversity using estimated 30-year cardiovascular risk as the outcome

	Prospective measure				Retrospective measure			
	Model 1A		Model 2A		Model 1B		Model 2B	
	b	e^b	*b*	e^b	*b*	e^b	*b*	e^b
Childhood adversity (prospective)	.036*	1.037	.040**	1.040				
Childhood adversity (retrospective)					.049**	1.050	.057**	1.059
Control variables								
Males			.728**	2.071			.685**	1.983
Education			−.011	.989			−.016	.984
Log income			−.020**	.980			−.021**	.979
Married or cohabiting			.048	1.049			.040	1.041
Health insurance			.086	1.090			.083	1.087
Sleep quality			.018	1.018			.044	1.045
Healthy diet			.006	1.006			.006	1.006
Exercise			−.037**	.964			−.038**	.963
Depression			.029*	1.030			.017	1.017
Binge drinking			−.003	.997			−.009	.991
Former smokers			−.606**	.545			−.592**	.553
Lifetime never smokers			−.595**	.551			.−.573**	.564
Constant	−2.810**		−2.478**		−2.802**		−2.442**	

Note. Log odds (*b*) and odds ratio (e^b) are presented in the table; income is log-transformed; $N = 454$.

† $p \leq .10$;

* $p \leq .05$;

** $p \leq .01$ (two-tailed tests).

Table 4

Beta regression models explicating the impact of childhood adversity, parental emotional support at age 10, and their interaction on cardiovascular risk, controlling for sociodemographic and health-related variables

	Prospective measure				Retrospective measure			
	Model 1A		Model 2A		Model 1B		Model 2B	
	b	e^b	*b*	e^b	*b*	e^b	*b*	e^b
Childhood adversity (prospective)	.037*	1.038	.035*	1.036				
Childhood adversity (retrospective)					.055**	1.056	.047**	1.048
Parental support (Age 10)	−.044†	.957	.055	1.057	−.038†	.963	.016	1.016
Childhood adversity (prospective) × Parental support (Age 10)			−.037**	.963				
Childhood adversity (retrospective) × Parental support (Age 10)							−.031**	.970
Control variables								
Males	.727**	2.069	.734**	2.084	.686**	1.986	.693**	2.001
Education	−.011	.989	−.014	.986	−.016	.984	−.018	.982
Log income	−.019**	.981	−.016*	.984	−.020**	.980	−.020**	.980
Married or cohabiting	.043	1.044	.043	1.044	.037	1.037	.036	1.037
Health insurance	.081	1.084	.084	1.088	.081	1.084	.065	1.067
Sleep quality	.019	1.019	.015	1.015	.044	1.045	.038	1.038
Healthy diet	.007	1.007	.005	1.005	.006	1.006	.006	1.006
Exercise	−.039**	.962	−.040**	.961	−.040**	.961	−.040*	.960
Depression	.030*	1.030	.030**	1.031	.018	1.018	.018	1.019
Binge drinking	−.005	.995	−.008	.992	−.011	.989	−.012	.988
Former smokers	−.606**	.545	−.608**	.544	−.594**	.552	−.593**	.553
Lifetime never smokers	−.599**	.550	−.595**	.551	−.580**	.560	−.560**	.571
Constant	−2.476**		−2.447**		−2.445**		−2.382**	

Note. Log odds (*b*) and odds ratio (e^b) are presented in the table; parental emotional support is standardized by *z*- transformation; income is log-transformed; $N = 454$.

† $p \leq .10$;

* $p \leq .05$;

** $p \leq .01$ (two-tailed tests).