



# A prospective study of positive early life psychosocial factors and favorable cardiovascular risk in adulthood

## Citation

Appleton, Allison A., Stephen L. Buka, Eric B. Loucks, Eric B. Rimm, Laurie T. Martin, and Laura D. Kubzansky. 2013. "A Prospective Study of Positive Early-Life Psychosocial Factors and Favorable Cardiovascular Risk in Adulthood." *Circulation* 127 (8): 905–12. <https://doi.org/10.1161/circulationaha.112.115782>.

## Permanent link

<http://nrs.harvard.edu/urn-3:HUL.InstRepos:41245542>

## Terms of Use

This article was downloaded from Harvard University's DASH repository, and is made available under the terms and conditions applicable to Open Access Policy Articles, as set forth at <http://nrs.harvard.edu/urn-3:HUL.InstRepos:dash.current.terms-of-use#OAP>

## Share Your Story

The Harvard community has made this article openly available. Please share how this access benefits you. [Submit a story](#).

[Accessibility](#)



Published in final edited form as:

*Circulation*. 2013 February 26; 127(8): 905–912. doi:10.1161/CIRCULATIONAHA.112.115782.

## A prospective study of positive early life psychosocial factors and favorable cardiovascular risk in adulthood

Allison A. Appleton, ScD, MPH<sup>1</sup>, Stephen L. Buka, ScD<sup>2</sup>, Eric B. Loucks, PhD<sup>2</sup>, Eric Rimm, ScD<sup>1,3</sup>, Laurie T. Martin, ScD<sup>4</sup>, and Laura D. Kubzansky, PhD, MPH<sup>5</sup>

<sup>1</sup>Geisel School of Medicine at Dartmouth, Department of Community and Family Medicine

<sup>2</sup>Brown University, Department of Epidemiology

<sup>3</sup>Harvard School of Public Health, Department of Nutrition

<sup>4</sup>Rand Corporation

<sup>5</sup>Harvard School of Public Health, Department of Society, Human Development and Health

### Abstract

**Background**—The American Heart Association’s national goals for cardiovascular health promotion emphasize that cardiovascular risk originates early in life, but little is known about child factors that may increase the likelihood of having favorable cardiovascular risk (FCR) in adulthood. We examined the prospective association between positive child factors and likelihood of midlife FCR. We also considered pathways through which child factors may influence FCR.

**Methods and Results**—We studied 415 adults (mean age=42.2 years) of the Collaborative Perinatal Project, a national cohort initiated in 1959–1966. We examined three positive child factors assessed at age 7 years: attention regulation (ability to stay focused), cognitive ability and positive home environment. 10.6% had FCR in midlife. Adjusting for demographics and child cardiovascular health, a one unit increase in child attention regulation, cognitive ability and positive home environment was associated with 2.4 (95%CI: 1.1 to 4.7), 1.8 (95%CI: 1.1 to 2.9), and 1.3 (95%CI: 1.1 to 1.6) higher respective odds of having midlife FCR. The association with child attention regulation was maintained when accounting for adult factors; education and diet partly explained the associations with child cognitive ability and home environment. The effect of each attribute was additive as those with high levels of each child factor had 4.3 higher odds (95%CI: 1.01 to 18.2) of midlife FCR compared to those low in all factors.

**Conclusions**—Positive child psychosocial factors may promote healthy adult cardiovascular functioning. Primordial prevention efforts aimed at preventing the development of cardiovascular risk should consider building on child psychosocial resources.

### Keywords

Favorable cardiovascular risk; psychosocial factors; life course

---

Children are typically born with many of the requisite components of ideal cardiovascular health. They generally have healthy blood pressure, lipid and glucose levels, they do not

---

Correspondence and reprint requests to: Allison A. Appleton, ScD, Geisel School of Medicine at Dartmouth, Department of Community and Family Medicine, HB7927, Room 519; Hanover, NH, 03755; phone: 617-645-9842; allison.a.appleton@dartmouth.edu.

### DISCLOSURES

The authors report no conflicts.

smoke and have the potential for developing an ideal body weight, healthy dietary and physical activity practices. That said, numerous studies have indicated that loss of cardiovascular health often begins in childhood and progresses over the life course.<sup>1</sup> Those who reach midlife with a favorable constellation of cardiovascular risk factors (e.g., systolic blood pressure <120 mmHg, diastolic blood pressure <80 mmHg, total cholesterol <200 mg/dL, body mass index <25 kg/m<sup>2</sup>, non-smoker, no diabetes) experience significantly lower lifetime remaining risk for developing cardiovascular diseases (CVD) and have increased longevity relative to individuals with unfavorable levels of such factors.<sup>2-6</sup> However, the vast majority of middle aged U.S. adults have poor cardiovascular health<sup>5</sup> and once risk factors are elevated, they are difficult to ameliorate.<sup>4</sup> As such, primordial prevention of CVD, or preventing the development of CVD risk factors in the first place, has increasingly become of interest to cardiovascular epidemiologists and clinicians in order to more effectively reduce the burden of CVD in the population.<sup>4</sup> As many CVD risk factors such as obesity and atherosclerosis develop early in life<sup>1</sup>, childhood is a life stage particularly amenable to primordial prevention efforts as cardiovascular risk is not yet well established. Moreover, childhood may serve as a sensitive period of development whereby exposures occurring early in life, including health promoting experiences, may impact physical health over the life course.<sup>7, 8</sup> Therefore, the identification of child factors associated with adult favorable cardiovascular risk (FCR) may be of great utility in furthering primordial prevention work.

Significant public health efforts have focused on the reduction and prevention of CVD risk factors during childhood (e.g., reduction in sedentary behavior). While important and necessary, this approach often overlooks the role of positive factors in childhood that may actively promote health. Positive factors do not simply reflect the absence of risk factors, but instead are independent attributes or assets that enhance health and resilience over time.<sup>9</sup> Recent work has identified several early life psychosocial factors that may influence cardiovascular health over the life course, including self-regulation (the ability to manage behavior, emotion, attention and social interactions), cognitive ability, and aspects of the home environment.<sup>10, 11</sup> Cognitive ability and the capacity to self-regulate are attributes that emerge in childhood and remain stable over the life course.<sup>10</sup> They are hypothesized to influence life-long health by facilitating education attainment, effective problem solving, communication, memory, sense of control and ability to cope with stressful situations.<sup>10, 12, 13</sup> While less frequently studied than childhood adversity, positive features of the child home environment are hypothesized to promote resiliency over the life course through a variety of pathways including the promotion of more resilient biology (e.g., increased expression of oxytocin in response to warm parenting) and the development of effective coping and emotion regulation strategies.<sup>7, 11, 13, 14</sup> Following a life course accumulation model<sup>8</sup>, the benefits of positive child attributes may cumulatively build over time, thereby safeguarding and promoting cardiovascular health in adulthood, although this hypothesis has not yet been tested. Thus, this study was designed to test the hypothesis that positive child psychosocial factors would be associated with more FCR in adulthood.

The greatest insight into potential targets for primordial prevention may be gained by rigorous control for potential confounding, including accounting for child cardiovascular health (e.g., blood pressure, diabetes, body mass index), and considering likely pathways through which positive early life psychosocial factors may contribute to adult cardiovascular health. Therefore, the aim of the current study is to examine the prospective association of three positive child psychosocial factors self-regulation, cognitive ability and positive home environment with the likelihood of having FCR 35 years later, while rigorously controlling for child cardiovascular health and other potential confounders. A secondary aim is to examine potential pathways (e.g., diet, physical activity) from adulthood that might help explain the associations. While there has been much recent interest in defining healthy

cardiovascular functioning and identifying the life course predictors of FCR in adulthood<sup>4, 15</sup>, the role of psychosocial factors in the development of healthy cardiovascular functioning has not been evaluated. To our knowledge, this is the first study to examine prospectively the association between multiple positive child psychosocial factors with FCR in adulthood.

## METHODS

### Sample

Figure 1 displays a flow chart describing how the study sample was selected. The sample comes from offspring of participants of the Collaborative Perinatal Project (CPP). Pregnant women enrolled in the CPP between 1959–1966, and their offspring were regularly assessed from birth to 7 years<sup>16, 17</sup>. The New England Family Study (NEFS) is a set of follow-up studies of the now adult offspring from the New England sites. Details on selection of the current sample is described elsewhere<sup>18</sup>. Briefly, our sample includes NEFS participants involved in two adult follow-up studies: first in the Brown-Harvard Transdisciplinary Tobacco Use Research Center (TTURC) and subsequently in the EdHealth study which included 618 TTURC participants, selected with preference for racial/ethnic minorities and those with low and high levels of education as required by the aims of the project. Of the 618 individuals interviewed (69% response rate), 42 who were not interviewed in-person (and did not complete physiological assessments) were excluded, resulting in 576 eligible participants. Of these, 430 (75%) participated in the clinical assessment, where a blood sample and anthropomorphic measurements were obtained by trained study personnel. Of these, 415 had complete outcome data and were included in analysis. Missing covariate information was imputed for 27 of these participants (6.5%). As results were highly similar when using imputed and non-imputed datasets, we present the imputed results here. Human subjects committees at Brown University and Harvard School of Public Health approved the study protocol. All participants provided informed consent.

### Measures

**Favorable cardiovascular risk**—Consistent with other work in this area<sup>2, 3, 6</sup>, FCR was defined as meeting the following criteria in midlife: systolic blood pressure (SBP)  $\leq 120$  mmHg, diastolic blood pressure (DBP)  $\leq 80$  mmHg, not taking antihypertensive medication, total cholesterol  $< 200$  mg/dL, not taking cholesterol lowering medication, body mass index  $< 25$  kg/m<sup>2</sup>, not having diabetes, and non-smoker. FCR was dichotomized according to whether or not participants met all criteria (yes/no).

Total cholesterol was measured in non-fasting plasma samples at CERLab (Harvard Medical School, Boston, MA) using a Hitachi 911 analyzer, and participating in the CDC/NHLBI Lipid Standardization Program. Total cholesterol (CV=1.7%) was measured enzymatically as described elsewhere.<sup>19, 20</sup> Five blood pressure readings were obtained over one-minute intervals after 5 minutes rest, in the right arm at heart level, using automated blood pressure monitors (VSMedTechBpTru, Coquitlam, BC, Canada), which have good validity and reliability compared to auscultation.<sup>21</sup> Systolic and diastolic blood pressure values were calculated as the mean of the lowest three readings, excluding the first. Body mass index (BMI) was calculated as the ratio of weight in kilograms to the square of height in meters (kg/m<sup>2</sup>) using height and weight measurements obtained by study personnel. Current smoker, antihypertensive medication use, cholesterol lowering medication use and presence of diabetes (as told by doctor/health professional; excluding gestational diabetes) were each self-reported (yes/no).

**Positive child psychosocial factors**—Attention regulation capacity, cognitive ability and positive home environment were each assessed during childhood.

Attention regulation capacity is a specific form of self-regulation that reflects the child's ability to stay focused on a task, persevere, and persist in problem solving.<sup>22</sup> At the age 7 year study visit, trained psychologists administered a two-hour battery of cognitive, sensory and motor tests to the child without the mother present and rated the child on 15 behaviors observed during those tests. A reliable measure of attention regulation capacity was derived from these behaviors ( $\alpha=0.82$ ).<sup>22</sup> Past work has documented the validity of this scale against a contemporary gold standard for assessing child behavior (Achenbach Child Behavior Checklist; CBCL).<sup>23</sup> Attention regulation capacity is negatively correlated with internalizing, externalizing and attention problems as assessed with the CBCL, and has been found to predict emotional functioning in adulthood.<sup>22</sup> Scores were standardized to have a mean of 0 and standard deviation of 1. Scores above the mean indicate better attention regulation capacity.

Child cognitive ability reflects the ability to reason, think abstractly, comprehend ideas and learn from experience.<sup>24</sup> Cognitive ability was assessed during the age 7 study visit using the Wechsler Intelligence Scale for Children<sup>25</sup>, from which an estimate of IQ was derived. In the general population, the mean IQ is 100 with a standard deviation of 15. Scores above the mean indicate greater cognitive ability. For ease of interpretation, logistic regression coefficients for child IQ were multiplied by 15 and then exponentiated to obtain odds ratios that correspond to a 15-point (or 1 standard deviation in the general population) advantage in childhood cognitive performance.

Child home environment was a composite measure that assessed positive emotional, social, and physical aspects of the home from birth to age 7 years. Home environment was assessed as a composite, as emotional, social and physical aspects of home life are not experienced in isolation from one another and may cumulatively affect FCR. Using composite variable construction methods common in child adversity research<sup>26</sup>, the positive home environment score was a sum of the number of positive emotional, social and physical aspects of the home environment. Because no component was hypothesized to be more important than another, parts of the composite were not additionally weighted, resulting in a simple and conservative summary of the cumulative experience of positive features of the home environment. The emotional domain was assessed via maternal parenting behaviors. Mother and child interactions were observed when the child was 8 months old by study psychologists during standardized cognitive and developmental testing of the infant. Several maternal parenting behaviors were observed and scored as present or not (1/0) including expression of affection, acceptance of child, positive physical handling, guidance of child, and responsiveness to child's needs. Individual parenting behaviors were not necessarily positive experiences themselves, but together represent a parenting style that may have implications for FCR later in life. Therefore, a summary maternal parenting score was subsequently derived, with higher scores indicating greater tendency toward warm and responsive parenting behaviors (range 0–5;  $\alpha=0.68$ ). Experiencing warm and responsive parenting in infancy is associated with better psychological functioning in adulthood, including greater optimism ( $t(340)=2.27, p=0.02$ ), marginally fewer depressive symptoms ( $t(344)=1.89, p=0.08$ ), greater agreeableness ( $t(343)=2.45, p=0.01$ ) and marginally greater emotional stability ( $t(343)=1.63, p=0.10$ ) in this sample.

The overall emotional domain score was then dichotomized as high (5) and low (<5) based on the available distribution for inclusion in the home environment composite (1=high/0=low; range 0–1). The social environment domain was assessed with a summary score (range 0 – 4) based upon the presence or absence (1/0) of the following characteristics: two

parent household at age 7, never living poverty, high parental education (more than high school; according to the highest level of education in the household as reported by either parent), and father consistently employed during the child's first 7 years of life. The physical environment domain was assessed according to a sum score (range 0–2) across presence or absence (1/0) of residential stability (no moves over 7 years) and not living in a crowded dwelling at age 7 (<1.5 people per room). To obtain the summary positive home environment variable, one point was given to each emotional, social and physical environmental domain item, and overall sum scores were derived (range 0–7). Higher scores indicate a more positive child home environment.

Additionally, we examined the potential additive effects of having high levels of one, two and all three positive childhood factors (attention regulation capacity, cognitive ability, positive home environment). We assessed this by creating a dichotomous variable (high/low) for each factor followed by a composite four category variable: high for all three factors, high for two factors, high for one factor, high for zero factors. Following prior work with child cognitive ability scores<sup>27</sup>, high cognitive ability was defined as those with IQ scores of 115 or higher (i.e., at least 1 standard deviation above the mean). High attention regulation and high positive home environment reflect those in the top tertile of the distributions. These cut points are based on the available distribution of scores within this sample, and represent those with a high degree of child psychosocial assets hypothesized to be relevant for FCR.

## Covariates

Demographic, child and adult covariates were selected based on their potential for either confounding<sup>28</sup> or mediating<sup>29</sup> the associations between childhood psychosocial assets and FCR in adulthood. Self-reported demographics that could potentially confound the associations included age at the midlife assessment, race (white/not white), and gender. A site variable (Boston, Massachusetts/Providence, Rhode Island) was included in all models to adjust for potential differences between study locations.

Several potential confounding variables from childhood were examined including socioeconomic status (SES; index reflecting the education, occupation and income of the head of household when the child was 7 years, scores range from 0/low-100/high)<sup>30</sup>, small for gestational age (SGA; birthweight was <10<sup>th</sup> percentile for gestational age at delivery), presence of a chronic physical health condition during childhood, and child cardiovascular health. Childhood chronic physical health conditions were derived from physical examinations by CPP pediatricians at ages 1 and 7 years, obtained via mothers' reports at each visit, and extracted from medical records at ages 1 and 7. We used a summary score of the number of chronic physical health conditions (including abnormalities of the liver, cardiovascular conditions, hematologic conditions (e.g. anemia), lower respiratory tract abnormality (e.g. asthma), neoplastic disease, neurologic abnormality, and prolonged/recurrent hospitalization), coded as 0 or 1 conditions. Child cardiovascular health was assessed according to BMI, blood pressure, and presence of diabetes at age 7 years. BMI was calculated as kg/m<sup>2</sup> using height and weight obtained by trained study personnel using standard anthropometry equipment for the time. Child systolic and diastolic blood pressure (mmHg) was measured by a study pediatrician. Presence of diabetes was assessed by a study pediatrician. However, as only one child was a suspected case and no children received a diagnosis of diabetes, we did not include this variable in the analysis.

Adulthood potential pathway variables were self-reported. Depressive symptoms were assessed via the validated Center for Epidemiologic Studies of Depression scale<sup>31</sup> ( $\alpha=0.88$ ), with higher scores indicating more depressive symptoms. Education attainment was the total years of education completed. Physical activity was assessed with a single item that asked

whether or not participants engaged in vigorous physical activity in a typical week. Western and prudent dietary patterns were assessed with a 25-item Food Frequency Questionnaire.<sup>32, 33</sup> Western diets were characterized by higher consumption of red meats, refined grains, high-fat dairy products and sugar sweetened beverages. Prudent diets were characterized by higher consumption of fruits, vegetables, legumes, whole grains, fish and poultry.

## Analysis

We used multiple imputation procedures (PROC MI, PROC MIANALYZE; SAS Institute Inc) to impute missing values on covariates and pool estimates from five imputed datasets.<sup>34, 35</sup> Those excluded from the analytic sample due to missing outcome data (e.g., did not complete the clinical assessment; n=203) were compared to those analyzed (n=415) in terms of childhood psychosocial factors, child health (SGA, chronic condition, blood pressure, age 7 BMI), age, race, gender, adult BMI and education attainment via  $\chi^2$  and independent t-tests. Bivariate associations for participant characteristics and positive childhood factors were assessed with Pearson's correlations. Bivariate associations between FCR and study variables were assessed with independent t and  $\chi^2$  tests. Multiple logistic regression models assessed the associations of each positive childhood psychosocial factor and FCR in adulthood as well as the additive effect of having high levels of one, two or three positive childhood factors. Demographic and child covariates were considered as potential confounders, while covariates from adulthood were considered as possible pathways through which positive child psychosocial factors might influence the development of FCR. Home environment models do not adjust for child SES as socioeconomic factors in part comprise the home environment variable. Evidence that adulthood factors may serve as pathways was assessed first by including all adulthood factors in the models as a single block and examining whether the coefficient for the childhood psychosocial factor was attenuated.<sup>36</sup> When the child coefficients were attenuated, Sobel tests adapted for logistic regression<sup>37</sup> were conducted for each adulthood factor to determine if the mediated effect attributable to that factor was significantly different from zero. Finally, to evaluate which components of FCR profile may be most strongly related to the child psychosocial factors, logistic regression models were fit for each child factor with components of the FCR profile as separate outcomes. Statistical significance was determined by 95% confidence intervals and p-values less than 0.05.

## RESULTS

### Descriptive statistics

There were no significant differences in positive child psychosocial factors, child health, adult BMI, education attainment or gender among those who were excluded (n=203) and included in the study (n=415) (all  $p>0.05$ ), although the excluded were older by 0.85 years ( $t(616)=-5.5$ ,  $p<0.001$ ) and more likely to be white ( $\chi^2=4.4$ ,  $p<0.05$ ). Table 1 lists sample characteristics and bivariate associations of these characteristics with the positive childhood psychosocial factors. Participants were on average 42.2 years old at follow-up, 80.5% white, and 59% female. The prevalence of adult FCR was 10.6%, which is within the range of what other studies have observed.<sup>2, 3</sup> Positive childhood psychosocial factors were positively correlated with higher child SES (all  $p<0.001$ ), higher educational attainment (all  $p<0.10$ ) and negatively correlated with adult depressive symptoms (all  $p<0.05$ ). Child psychosocial factors were correlated with one another (all  $p<0.05$ ) but the magnitude of the associations were small to moderate ( $r$  ranged from 0.11–0.41) indicating limited overlap in constructs. Compared to those who did not have FCR in adulthood, those with FCR were significantly younger at follow-up ( $t(413)=2.6$ ,  $p=0.01$ ), more likely to be female ( $\chi^2=15.2$ ,  $p<0.001$ ), had higher SES as children ( $t(398)=-2.1$ ,  $p=0.04$ ), and had higher education attainment

( $t(410)=-2.4, p=0.02$ ), fewer depressive symptoms ( $t(413)=-3.5, p=0.008$ ), and greater adherence to a prudent diet ( $t(402)=2.9, p=0.004$ ) versus a western diet ( $t(402)=3.3, p=0.007$ ) as adults.

### Positive child psychosocial factors and favorable cardiovascular risk in adulthood

Positive child psychosocial factors were significantly associated with FCR in adulthood, controlling for demographic and childhood covariates (Table 2). The odds ratios (ORs) for adult FCR associated with a one standard deviation increase in child attention regulation capacity, a 15 point increase in child IQ score, and a one unit increase in positive home environment score were 2.4 (95% CI: 1.1 to 4.7), 1.8 (95% CI: 1.1 to 2.9) and 1.3 (95% CI: 1.1 to 1.6) respectively.

Five percent of participants had high levels of all three positive childhood psychosocial factors. Relative to individuals with low levels of all three factors, those who were high in all three assets had significantly higher odds (OR=4.3; 95% CI: 1.01, 18.2) of FCR in adulthood, and a non-significant trend was evident among those high in two childhood factors (OR=2.5, 95% CI: 0.94, 6.7;  $p<0.10$ ), controlling for demographic and childhood covariates (Table 2).

Child attention regulation capacity remained independently associated with FCR when adjusting for adulthood pathways factors (Table 2). In contrast, controlling for adulthood factors attenuated associations for FCR with cognitive ability and home environment, suggesting one or more adulthood factors may be on the pathway (Table 2). Results from the Sobel tests indicated that no single adult factor mediated the association between cognitive ability and FCR, although trends were evident for fewer depressive symptoms ( $p=0.07$ ), higher education attainment ( $p=0.15$ ) and greater adherence to a prudent diet ( $p=0.12$ ). For home environment, the observed mediated effect was significantly different from zero for higher education attainment ( $p=0.02$ ) and fewer depressive symptoms ( $p=0.05$ ), and a trend was evident for greater adherence to a prudent diet ( $p=0.13$ ).

Associations of positive child factors with component parts of the FCR profile are displayed in Table 3. Associations of attention regulation, cognitive ability and home environment with each FCR component were typically in the expected directions, although the confidence intervals overlapped the referent category for most factors. Attention regulation was significantly associated with not using antihypertensive medication (OR=1.4; 95% CI: 1.0 to 2.0); home environment was associated with low DBP (OR=1.2; 95% CI: 1.0 to 1.4), not having diabetes (OR=1.5; 95% CI: 1.0 to 2.1) and not smoking (OR=1.3, 95% CI: 1.1 to 1.5). Overall, findings suggest that attention regulation, cognitive ability and home environment are modestly associated with each FCR component. Thus, a modest effect on each factor likely led to significant associations of the positive child factors with the overall FCR score, which combines all factors into a single score.

## DISCUSSION

Results from this study indicate that positive child psychosocial factors may promote cardiovascular health in midlife. Specifically, higher levels of childhood attention regulation capacity, cognitive ability and positive home environment were associated with greater likelihood of FCR in adulthood, as characterized by healthy levels of blood pressure, cholesterol, and BMI as well as cardiovascular related medication use, smoking and diabetes status. The cardiovascular benefit of each factor appears to be additive as the overall effect of having high levels of each factor is large: those with high levels of all assets had over four fold increased odds of FCR in adulthood. We illustrate that while possessing any of the positive factors in childhood may benefit cardiovascular health, the effect of having multiple

psychosocial assets may cumulatively build to protect cardiovascular health. These findings are novel as this study is the first to examine positive early life psychosocial determinants of a cardiovascular health profile indicative of low remaining lifetime risk of developing CVD and increased longevity.<sup>2, 3, 6</sup>

Prior work has suggested that these childhood factors are associated with reduced risk of CVD in adulthood. For example, one previous report from our team among 569 men and women found child attention regulation capacity measured at age 7 was associated with fewer adult physical health conditions, including heart disease, diabetes and stroke, 30 years later.<sup>38</sup> In another study among 1122 adult men (mean age=60.3 years), high self-regulation was associated with 20% reduced risk of incident coronary heart disease over 13 years of follow-up.<sup>13</sup> Similarly, several studies suggest that low child cognitive ability (indexed by IQ score) is associated with increased CVD risk in adulthood.<sup>27, 39</sup> For example, in a study of 938 Scottish men and women, a one standard deviation lower IQ score at age 10 was associated with a 19% increase in CVD related hospital admissions and deaths over 25 years of follow-up.<sup>39</sup> Also, while few studies have considered the association between a positive child home environment and cardiovascular health, much work has considered adversities in the home environment and specific parenting behaviors in association with CVD risk.<sup>7, 11, 18, 40</sup> For example, among 1205 participants of the Midlife in the United States Study (MIDUS), retrospective accounts of high levels of maternal nurturance buffered the deleterious effects of growing up in poverty on metabolic syndrome risk in midlife.<sup>11</sup> Similarly, among 3554 participants of the Coronary Artery Risk Development in Young Adults (CARDIA) study, a retrospective account of adversities in the childhood family psychosocial environment (e.g., child was physically abused, lacking affection, lived with a substance abuser) was associated with higher 10-year coronary heart disease risk in adulthood as estimated by the Framingham algorithm.<sup>40</sup> Taken together, these studies suggest that child attention regulation, cognitive ability and aspects of the home environment may contribute to adult CVD risk. Our study extends this work to look at protective childhood factors for maintaining and promoting cardiovascular health over the life course.

Results from the pathways models suggest that the associations between child cognitive ability and positive home environment with FCR may be working through adult social and behavioral factors, including fewer depressive symptoms, higher education attainment and healthier dietary patterns. As such, consistent with a life course accumulation model<sup>8</sup>, the benefits of higher child cognitive ability and positive home environment may cumulatively protect cardiovascular health by way of reducing or preventing social and behavioral risk factors for CVD. This finding is also consistent with recent prospective work among 3154 CARDIA participants whereby healthy lifestyle factors (e.g., healthy diet, physical activity, no smoking) originating in childhood and continuing through early adulthood contributed to the development of FCR in midlife.<sup>15</sup> Early life cognitive ability and positive home environments may help to set up such healthy lifestyle trajectories which cumulatively promote cardiovascular health in adulthood.

We also found child attention regulation to be independently associated with adult FCR. Attention regulation capacity is a higher order feature of self-regulation and executive functioning<sup>10</sup> that pervades many aspects of functioning relevant for cardiovascular health (e.g., maintaining healthy diets and exercise regimens, attending to health messages, navigation of the health care system). Moreover, effective self-regulation may reduce the intensity and duration of sympathetic nervous system and hypothalamic-pituitary-adrenal activation attributable to stress and negative emotions that could otherwise have deleterious effects on cardiovascular functioning over time.<sup>41</sup> Effective self-regulation may therefore help protect cardiovascular health by way of reducing or preventing such physiologic wear-

and-tear over the life course. Future work is advised to map such potential linkages explicitly.

This study has a number of strengths. First, the measures of child psychosocial factors were assessed 35 years prior to adult assessments, and were largely based on psychologist observations of maternal and child behaviors and standardized testing. Also, the outcome measure incorporated objectively measured biomarkers of CVD. Moreover, a broad range of covariates from across the life course were assessed, including multiple measures of childhood cardiovascular health, thereby addressing concerns of prior confounding by early life CVD risk. This study also has some limitations. First, unmeasured confounding may remain (e.g., genetics) and should be assessed in future work. Also, when CPP went into the field in the 1950s, there were no population-based research tools to assess parenting behavior or home environment. Though our measures perform moderately well, contemporary measures may provide more accurate assessments. Additionally, generalizability may be somewhat limited as participants were selected from a NEFS subsample based on certain demographic characteristics per the aims of the project. However, the prevalence of FCR we observed is comparable to rates found in similar population based studies<sup>2, 3</sup>, which helps mitigate some concerns about generalizability. Also, although loss to follow-up due to death was minimal in this study, exclusion of those who died could yield a healthier sample, which could lead to overestimated associations. Finally, outcome and pathway variables were assessed concurrently making tests of mediation less robust. These limitations notwithstanding, we note that this study is among the first to examine the associations of multiple positive child psychosocial factors with FCR in adulthood. As such, we demonstrate that the accumulation of psychosocial assets beginning early in life may help protect cardiovascular health over time.

This work has important applications for clinicians as the capacity to attain, maintain or regain FCR depends in part on life experience. Physician counseling in making lifestyle and behavior changes is a mainstay of clinical practice. This study suggests that it may not be enough to counsel individuals absent some understanding of their life history; the effectiveness of counseling may depend in part on psychosocial resources within the patient, including capacity for attention and problem solving. These factors may influence a patient's ability to engage in behavior change or understand the medical advice being given. Thus, taking a medical history that includes information regarding the social environment and availability of psychosocial resources can provide clinicians important contextual information to use in tailoring medical advice. Simple paper-and-pencil attention deficit self-report symptom scales for adults are available for use in clinical practice and can be informative in identifying patients who may need additional supports to change behavior. While cognitive ability assessments may not be practically conducted in a clinical setting, physicians can ask patients about their educational history as a proxy (e.g., Did you graduate high school? Did you receive any educational services as a child? Have you been diagnosed with cognitive or developmental disability?). Such information can be useful in identifying the need to tailor a behavior change plan to be understandable, and potentially more efficacious, for the patient.

From a primordial prevention perspective, this study also suggests that many of the assessments pediatricians already perform could be useful in promoting cardiovascular health. Child attention capacity can be assessed in a clinical setting with short, validated paper-and-pencil measures. Aspects of the home environment and can be assessed via taking medical histories to identify instability or social stressors in the home. These assessments provide information on the child's psychosocial assets and needs while also affording an opportunity to intervene (e.g., developmental testing, connections to social services). While assessing cognitive ability generally requires standardized testing outside the clinician's

office, pediatricians can refer children at high-risk of developing cognitive problems (e.g., premature infants) to programs that build such capacities. These efforts may enhance the child's immediate psychosocial environment as well as support the development of healthy cardiovascular functioning.

Evidence is accumulating that CVD may have developmental origins in childhood and that psychosocial factors may influence risk and resilience trajectories earlier than was previously considered.<sup>7, 40</sup> This study further demonstrates that positive early life psychosocial factors may promote healthy cardiovascular functioning later in life. As such, increasing the prevalence of FCR in the population may hinge in part on enhancing and protecting early life psychosocial assets. Doing so may lay the foundation for a lifetime of positive cardiovascular health.

## Acknowledgments

### FUNDING SOURCES

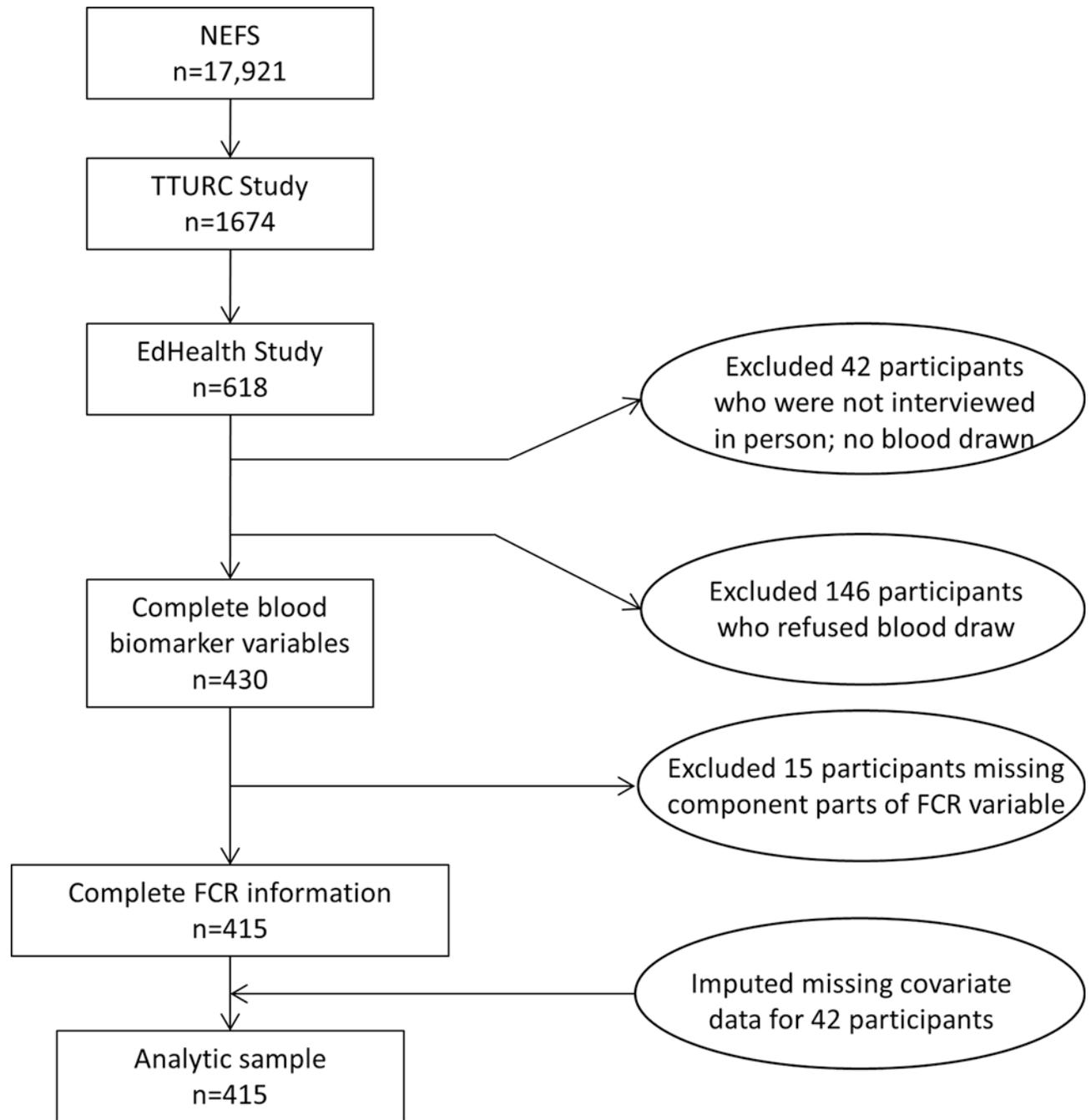
This work was supported by National Institutes of Health (NIH) Transdisciplinary Tobacco Use Research Center (TTURC) Award (P50 CA084719) by the National Cancer Institute, the National Institute on Drug Abuse, the National Institute of Aging grant AG023397, and the Robert Wood Johnson Foundation's Pioneer Portfolio, through a grant, "Exploring Concepts of Positive Health." Dr. Appleton was supported by the National Heart Lung and Blood Institute Training Grant at the Harvard School of Public Health (T32 HL098048), and the Quantitative Biomedical Sciences training program at Geisel School of Medicine at Dartmouth (R25 CA134286).

## REFERENCES

- Berenson GS, Srinivasan SR. Cardiovascular risk factors in youth with implications for aging: The bogalusa heart study. *Neurobiol Aging*. 2005; 26:303–307. [PubMed: 15639307]
- Lloyd-Jones DM, Leip EP, Larson MG, D'Agostino RB, Beiser A, Wilson PWF, Wolf PA, Levy D. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation*. 2006; 113:791–798. [PubMed: 16461820]
- Lloyd-Jones DM, Dyer AR, Wang R, Davi GL, Greenland P. Risk factor burden in middle age and lifetime risks for cardiovascular and non-cardiovascular death (chicago heart association detection project in industry). *Am J Cardiol*. 2007; 99:535–540. [PubMed: 17293199]
- Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenland K, Daniels S, Nichol G, Tomaselli GF, Arnett DK, Fonarow GC, Ho PM, Lauer MS, Masoudi FA, Robertson RM, Roger V, Schwamm LH, Sorlie P, Yancy CW, Rosamond W. Defining and setting national goals for cardiovascular health promotion and disease reduction: The american heart association's strategic impact goal through 2020 and beyond. *Circulation*. 2010; 121:586–613. [PubMed: 20089546]
- Folsom AR, Yatsuya H, Nettleton JA, Lutsey PL, Cushman M, Rosamond W. Community prevalence of ideal cardiovascular health, by the american heart association definition, and relationship with cardiovascular disease incidence. *J Am Coll Cardiol*. 2011; 57:1690–1696. [PubMed: 21492767]
- Davi GL, Stamler J, Pirzada A, Yan LL, Garside DB, Liu K, Wang R, Dyer AR, Lloyd-Jones DM, Greenland P. Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality. *JAMA*. 2004; 292:1588–1592. [PubMed: 15467061]
- Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities. *JAMA*. 2009; 301:2252–2259. [PubMed: 19491187]
- Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. *Int J Epidemiol*. 2002; 31:285–293. [PubMed: 11980781]
- Kobau R, Seligman MEP, Peterson C, Diener E, Zack M, Chapman D, Thompson W. Mental health promotion in public health: Perspectives and strategies from positive psychology. *Am J Public Health*. 2011; 101:e1–e9. [PubMed: 21680918]

10. Shonkoff, JP.; Phillips, DA. From neurons to neighborhoods: The science of early childhood development. Washington DC: National Academy Press; 2000.
11. Miller GE, Lachman ME, Chen E, Gruenewald TL, Karlamangla AS, Seeman T. Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. *Psychol Sci.* 2011; 22:1591–1599. [PubMed: 22123777]
12. Martin LT, Kubzansky LD, LeWinn KZ, Lipsitt LP, Satz P, Buka SL. Childhood cognitive performance and risk of generalized anxiety disorder. *Int J Epidemiol.* 2007; 36:769–775. [PubMed: 17470490]
13. Kubzansky LD, Park N, Peterson C, Vokonas P, Sparrow D. Healthy psychological functioning and incident coronary heart disease: The importance of self-regulation. *Archi Gen Psychiatry.* 2011; 68:400–408.
14. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of the offspring. *Psychol Bull.* 2002; 128:330–336. [PubMed: 11931522]
15. Liu K, Daviglius ML, Loria CM, Colangelo LA, Spring B, Moller AC, Lloyd-Jones DM. Healthy lifestyle through young adulthood and the presence of low cardiovascular disease risk profile in middle age: The coronary artery risk development in (young) adults (cardia) study. *Circulation.* 2012; 125:996–1004. [PubMed: 22291127]
16. Broman, SH.; Nichols, PI.; Kennedy, WA. Preschool IQ: Prenatal and early developmental correlates. New York: Hallstead Press; 1975.
17. Niswander, KR.; Gordon, M. The women and their pregnancies. Washington, DC: US Government Printing Office; 1972.
18. Almeida ND, Loucks EB, Kubzansky LD, Pruessner J, Maselko J, Meaney MJ, Buka SL. Quality of parental emotional care and calculated risk for coronary heart disease. *Psychosom Med.* 2010; 72:148–155. [PubMed: 20064905]
19. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. *Clin Chem.* 1974; 20:470–475. [PubMed: 4818200]
20. Rifai N, Cole TG, Iannotti E, Law T, Macke M, Miller R, Dowd D, Wiebe DA. Assessment of interlaboratory performance in external proficiency testing programs with a direct hdl-cholesterol assay. *Clin Chem.* 1998; 44:1452–1458. [PubMed: 9665423]
21. Mattu GS, Heran BS, Wright JM. Overall accuracy of the bptru--an automated electronic blood pressure device. *Blood Press Monit.* 2004; 9:47–52. [PubMed: 15021078]
22. Kubzansky LD, Martin LT, Buka SL. Early manifestations of personality and adult emotional functioning. *Emotion.* 2004; 4:364–377. [PubMed: 15571435]
23. Achenbach, TM. Manual for the child behavior checklist/4–18 and 1991 profile. Burlington: Department of Psychiatry, University of Vermont; 1991.
24. Nisbett RE, Aronson J, Blair C, Dickens W, Flynn J, Halpern DF, Turkheimer E. Intelligence: New findings and theoretical developments. *Am Psychol.* 2012; 67:130–159. [PubMed: 22233090]
25. Wechsler, D. Wechsler intelligence scale for children. New York: The Psychological Corporation; 1949.
26. Evans GW, Fuller-Rowell TE, Doan SN. Cumulative childhood risk and obesity: The mediating role of self-regulatory ability. *Pediatrics.* 2012; 129:68–73.
27. Jokela M, Batty D, Gale CR, Kivimaki M. Low child iq and early adult mortality: The role of explanatory factors in the 1958 british birth cohort. *Pediatrics.* 2009; 124:e380–e388. [PubMed: 19706576]
28. Rothman, KJ.; Greenland, S. Modern epidemiology. Philadelphia: Lippincott Williams & Wilkins; 1998.
29. MacKinnon DP, Fairchild AJ, Fritz MS. Mediation analysis. *Annu Rev Psychol.* 2007; 58:593–614. [PubMed: 16968208]
30. Myrionthopoulos N, French K. An application of the u.S. Bureau of the census socioeconomic index to a large, diversified patient population. *Soc Sci Med.* 1968; 2:283–299. [PubMed: 5760819]
31. Radloff LS. Ces-d scale: A self-report depression scale for research in the general population. *Appl Psychol Measurement.* 1977; 1:385–401.

32. Willett, WC. Nutritional epidemiology. New York: Oxford University Press; 1998.
33. Michaud DS, Skinner HG, Wu K, Hu F, Giovannucci E, Willett WC, Colditz GA, Fuchs CS. Dietary patterns and pancreatic cancer risk in men and women. *J Natl Cancer Inst.* 2005; 97:518–524. [PubMed: 15812077]
34. SAS Institute Inc. *Sas/stat 9.2 users guide*. Cary, NC: SAS Institute; 2008. The mianalyze procedure.
35. Graham JW. Missing data analysis: Making it work in the real world. *Annu Rev Psychol.* 2009; 60:549–576. [PubMed: 18652544]
36. Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D. How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *Am J Psychiatry.* 2001; 158:848–856. [PubMed: 11384888]
37. MacKinnon DP, Dwyer JH. Estimating mediated effects in prevention studies. *Eval Rev.* 1993; 17:144–158.
38. Kubzansky LD, Martin LT, Buka SL. Early manifestations of personality and adult health: A life course perspective. *Health Psychol.* 2009; 28:364–372. [PubMed: 19450043]
39. Hart CL, Taylor MD, Davey Smith G, Whalley LJ, Starr JM, Hole DJ, Wilson V, Deary IJ. Childhood iq and cardiovascular disease in adulthood: Prospective observational study linking the scottish mental survey 1932 and the midspan studies. *Soc Sci Med.* 2004; 59:2131–2138. [PubMed: 15351478]
40. Loucks EB, Almeida ND, Taylor SE, Matthews KA. Childhood family psychosocial environment and coronary heart disease risk. *Psychosom Med.* 2011; 73:563–571. [PubMed: 21810898]
41. Rozanski A, Kubzansky LD. Psychologic functioning and physical health: A paradigm of flexibility. *Psychosom Med.* 2005; 67:S47–S53. [PubMed: 15953801]



**Figure 1. Flow chart of participant selection into the study**  
 NEFS=New England Family Study; TTURC=Transdisciplinary Tobacco Use Research Center; FCR=Favorable Cardiovascular Risk

Table 1

Characteristics of Study Participants and Correlations with Positive Child Psychosocial Factors

Characteristic	M(SD) or % n=415	Attention	Cognitive	Positive Home
		Regulation	Ability	Environment
r				
Demographics				
Age (years)	42.2 (1.8)	0.02	-0.02	0.11*
Race (not white)	19.5	-0.10 <sup>+</sup>	-0.25***	-0.002
Gender (male)	41.0	-0.03	0.13**	-0.04
Childhood covariates				
Socioeconomic status index	54.0 (23.0)	0.14**	0.46***	0.59***
Born small for gestational age	10.2	-0.01	-0.10*	-0.11*
Body mass index (kg/m <sup>2</sup> )	16.1 (1.6)	-0.01	0.09 <sup>+</sup>	-0.02
Chronic condition	18.3	0.03	-0.12*	-0.02
Systolic blood pressure (mmHg)	104.4 (10.9)	-0.04	0.01	0.10*
Diastolic blood pressure (mmHg)	60.4 (10.4)	0.02	0.13*	0.14**
Adulthood covariates				
Education attainment (years)	13.6 (2.6)	0.09 <sup>+</sup>	0.36***	0.33***
Depressive symptoms	1.59 (0.56)	-0.11*	-0.16**	-0.16**
Vigorous physical activity	73.2	0.06	0.13*	0.06
Western diet score (z)	0.03 (1.1)	-0.01	0.04	0.0004
Prudent diet score (z)	-0.01 (0.69)	0.07	0.14**	0.17**

<sup>+</sup>  
p<0.10,\*  
p<0.05,\*\*  
p<0.01,\*\*\*  
p<0.001

**Table 2**

Odds Ratios\* (95% Confidence Intervals) for the Associations of Positive Child Psychosocial Factors with Favorable Cardiovascular Risk in Adulthood

Child factor	Unadjusted	+ Demographic <sup>†</sup>	+ Childhood <sup>‡</sup>	+ Pathways <sup>§</sup>
Attention regulation	<b>2.3 (1.2, 4.6)</b>	<b>2.4 (1.2, 4.9)</b>	<b>2.4 (1.1, 4.7)</b>	<b>2.0 (1.0, 3.9)</b>
Cognitive ability	<b>1.6 (1.1, 2.3)</b>	<b>1.7 (1.1, 2.7)</b>	<b>1.8 (1.1, 2.9)</b>	1.5 (0.90, 2.6)
Positive home environment	<b>1.3 (1.1, 1.6)</b>	<b>1.3 (1.1, 1.6)</b>	<b>1.3 (1.1, 1.6)</b>	1.2 (0.90, 1.5)
Additive effect of positive factors				
High in all factors (n = 19)	<b>2.6 (1.5, 4.5)</b>	<b>4.3 (1.1, 17.4)</b>	<b>4.3 (1.0, 18.2)</b>	1.9 (0.42, 9.1)
High in two factors (n = 63)	<b>1.6 (1.1, 2.4)</b>	2.4 (0.93, 6.4)	2.5 (0.94, 6.7)	2.0 (0.68, 6.0)
High in one factor (n = 206)	0.97 (0.69, 1.4)	1.0 (0.49, 2.1)	0.98 (0.46, 2.1)	0.81 (0.38, 1.8)
High in zero factors (n = 127)	--	--	--	--

\* Odds ratios are interpreted as the odds of having favorable cardiovascular risk in adulthood per unit change in childhood psychosocial factors. Significant odds ratios (p<0.05) are in bold.

<sup>†</sup>Demographic model adjusts for site, age and race.

<sup>‡</sup>Childhood model adjusts for demographic and child factors (born small for gestational age, chronic conditions, blood pressure, body mass index, socioeconomic status). Home environment models do not adjust for socioeconomic status as socioeconomic factors in part comprise the variable.

<sup>§</sup>Pathways model adjusts for demographic, child and adult factors (education attainment, depressive symptoms, physical activity, diet)

**Table 3**

Odds ratios (95% confidence intervals)\* for the Associations Between Positive Child Psychosocial Factors and Component Parts of the Favorable Cardiovascular Risk Profile

Favorable cardiovascular risk component	%	Attention Regulation	Cognitive Ability	Home Environment
Systolic blood pressure <140 mmHg	69.6	1.0 (0.77, 1.3)	0.89 (0.67, 1.2)	1.1 (0.93, 1.3)
Diastolic blood pressure <80 mmHg	68.0	1.1 (0.86, 1.4)	0.87 (0.66, 1.1)	<b>1.2 (1.0, 1.4)</b>
Not using antihypertensive medication	89.4	<b>1.4 (1.0, 2.0)</b>	1.2 (0.78, 1.8)	1.2 (0.92, 1.4)
Total cholesterol <200 mg/dL	56.8	1.2 (0.93, 1.5)	1.1 (0.88, 1.5)	0.98 (0.85, 1.1)
Not using cholesterol lowering medication	92.5	1.2 (0.74, 1.8)	1.2 (0.78, 2.0)	0.97 (0.74, 1.3)
Body mass index < 25 kg/m <sup>2</sup>	29.4	1.2 (0.90, 1.6)	1.1 (0.86, 1.5)	1.0 (0.89, 1.2)
Non-Diabetic	96.1	1.4 (0.85, 2.3)	1.3 (0.68, 2.4)	<b>1.5 (1.0, 2.1)</b>
Non-Smoker	72.8	1.1 (0.67, 1.1)	1.2 (0.87, 1.6)	<b>1.3 (1.1, 1.5)</b>

\* Odds ratios are interpreted as the odds of having the favorable cardiovascular risk component in adulthood per unit change in child psychosocial factors, adjusted for demographic (age, race, gender, site) and child (born small for gestational age, chronic conditions, blood pressure, body mass index, socioeconomic status) covariates. Home environment models do not adjust for socioeconomic status as socioeconomic factors in part comprise the variable covariates. Significant odds ratios ( $p < 0.05$ ) are in bold.