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Childhood Family Psychosocial Environment and Coronary Heart Disease Risk

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Abstract

Objective—Little is known about whether the childhood family psychosocial environment affects coronary heart disease (CHD). Study objectives were to evaluate associations of childhood family psychosocial environment (termed “risky families”; characterized by cold, unaffectionate interactions, conflict, aggression, neglect, and/or low nurturance) with calculated risk for CHD.

Methods—Study participants included 3554 participants of the Coronary Artery Risk Development in Young Adults Study, aged 33 to 45 years. Childhood family psychosocial environment was measured using a risky family questionnaire via self-report. Ten-year CHD risk was calculated using the validated Framingham risk algorithm.

Results—In a multivariable-adjusted regression analysis adjusted for age, race/ethnicity, and childhood socioeconomic position, a 1-unit (range, 0–21) increase in risky family score was associated with 1.0% (95% confidence interval = 0.4%–1.7%) and 1.0% (95% confidence interval = 0.2%–1.8%) higher CHD risk in women and men, respectively. Multiple mediation analyses suggested significant indirect effects of education, income, depressive symptomatology, and anger-out expression in women and education in men, indicating that these may be mediating mechanisms between childhood psychosocial environment and CHD risk. Of the modifiable Framingham algorithm components, smoking (in women and men) and high-density lipoprotein (in women) were the factors most strongly associated with risky family score.

Conclusions—Childhood family psychosocial environment was positively associated with the calculated 10-year CHD risk. Mechanisms may include the potential negative impact of childhood family psychosocial environment on later-life socioeconomic position (e.g., education in men and women) and/or psychosocial functioning (e.g., depression and anger-out expression in women), which may in turn lead to higher CHD risk, particularly through smoking (in men and women) and low level of high-density lipoprotein cholesterol (in women).

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Keywords

family; risky family; childhood; life course; coronary heart disease; epidemiology

INTRODUCTION

Coronary heart disease (CHD) remains a major cause of mortality in developed nations and increasingly in developing countries (1). There is substantial interest in early-life determinants of CHD, spurred on in part by findings of early atherosclerotic lesions in adolescents and young adults (2); development of CHD risk factors such as obesity, elevated blood pressure, and high cholesterol level in infants and children (3); and suggestions that early-life markers such as birth weight and parental socioeconomic position (SEP) may be risk markers for CHD (3). A lesser-studied early-life potential determinant of CHD is the childhood family psychosocial environment. “Risky families” is a term proposed and developed by Taylor et al. (4), which is defined as a childhood family environment composed of cold, unaffectionate interactions; conflict; aggression; neglect; and/or low nurturance. Preliminary evidence suggests that risky families, or other measures of the childhood family psychosocial environment, may be associated with CHD risk (5,6). Plausible mediating mechanisms include observed associations of the childhood family psychosocial environment (such as childhood abuse, neglect, and household dysfunction) with increased risk for CHD risk markers such as obesity (5,7–9), smoking (5,10), psychosocial variables such as depression (5,11–13), and low educational attainment (14). Few studies have investigated associations of the childhood family psychosocial environment with overall risk for CHD or with individual CHD risk factors such as cholesterol level, blood pressure, and diabetes. Overall, evidence on associations of childhood family psychosocial environment with risk factors for CHD is suggestive but sparse, and it merits further investigation in large studies with measures of childhood psychosocial environment and adulthood CHD risk marker measurements.

Consequently, the objective of this study was to evaluate whether the childhood family psychosocial environment, measured with a risky family questionnaire, is associated with calculated 10-year risk for CHD (using the Framingham algorithm) in participants of the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Another objective was to evaluate whether risky family score is associated with individual modifiable CHD risk factor components of the Framingham algorithm, including smoking, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, systolic blood pressure, and diastolic blood pressure.

MATERIALS AND METHODS

Study Sample

The CARDIA Study is a multicenter, longitudinal study of CHD risk markers (15). At baseline assessment (1985 and 1986), the cohort included 5115 black and white adults aged 18 to 30 years, recruited from four metropolitan areas (Birmingham, Ala; Chicago, Ill; Minneapolis, Minn; and Oakland, Calif). Participants have been regularly examined since

baseline, including Examination 6, which occurred at the 15-year follow-up during the years 2000 to 2001 (ages 33–45 years). Study protocols were approved by institutional review boards at each institution, and written informed consent forms were obtained from participants.

Of the 3671 participants assessed at Examination 6, 3567 had variables required for calculation of the Framingham algorithm. Thirteen participants were excluded for not having risky family score variables, leaving 3554 (1584 men and 1970 women) for analyses. Participants with missing data ($n = 117$) were more likely ($p < .05$) to be of black race/ethnicity, have a lower level of education, have a higher score in the Center for Epidemiologic Studies Depression (CES-D) scale, have a higher anger-out score, have a lower social support score, and were less likely to take antihypertensive medications compared with the included participants. Included and excluded participants were similar ($p > .05$)with regard to age, body mass index, HDL, total cholesterol level, systolic and diastolic blood pressure, diabetes, smoking, childhood SEP, cholesterol-lowering medications, risky family score, and the predicted 10-year CHD risk. All study variables were ascertained at Examination 6 (2000–2001).

Independent Variable

Using a risky family questionnaire adapted from Felitti et al. (5) and further developed by Taylor et al. (4), participants answered questions about their parents or other adults in their household during the participants' childhood and adolescence (before the age of 18 years) using a seven-item scale, each item ranging from 1 (rarely or none of the time) to 4 (most or all of the time). Items were rescored from 0 to 3 and summed (after reverse scoring where appropriate) leading to an overall scale range from 0 to 21, where higher values represent more adverse experiences. Questions included whether participants felt loved, supported, and cared for; were verbally abused; were shown physical warmth and affection; were physically abused; lived with a substance abuser; and lived in a well-organized, well-managed household and whether their family knew what they were up to as children and adolescents. Cronbach α was 0.77. Primary analyses used nontransformed ordinal scale values. Because individual items differed in their variability, in sensitivity analyses, each item was z scored, before summing across items to create the summary score.

To evaluate the discriminant validity of the risky family variable, we investigated the variable's independence from other psychosocial variables (depressive symptomatology, social support, and anger-out expression) that could potentially alter the accuracy of retrospective reporting on family environment, using a confirmatory principal component factor analysis (16). After evaluating a scree plot of eigenvalues, four derived factors were identified, namely, a) all risky family questionnaire variables, b) all anger-out expression questionnaire variables, c) all negative social contacts questionnaire variables, and d) all depressive symptomatology (CES-D) questionnaire variables, as well as all positive social contacts questionnaire variables, based on which variables with orthogonally rotated factor loadings (i.e., correlation coefficients) greater than 0.30 clustered together. A correlation test was performed to confirm that these four derived factors were not correlated with one another. Pearson correlation coefficients ranged from 0.00 to 0.13. The factor analysis was

repeated constraining it to three derived factors, which were then identified as follows: a) all risky family questionnaire variables, b) all anger-out expression questionnaire variables, and c) all social support questionnaire variables (including positive and negative social contacts), as well as all depressive symptomatology (CES-D) questionnaire variables. Again, these three derived factors were not strongly correlated with one another, where Pearson correlation coefficients ranged from 0.01 to 0.12. Other studies from the literature have further evaluated the validity and reliability of retrospective reporting for constructs including childhood SEP (17), parental support and affection (18,19), and childhood abuse (20).

Dependent Variables

The 10-year risk of CHD was calculated using the validated Framingham risk algorithm that uses sex-specific Cox regression models, which incorporate age, diabetes, smoking, total and HDL cholesterol levels, and systolic and diastolic blood pressure, described elsewhere (21). With respect to the validity of the Framingham algorithm, the C statistic for the prediction of CHD events in the Framingham Heart Study is 0.74 in white men and 0.77 in white women, suggesting good predictive validity (21). External validity tests on white and black participants were performed in other studies and demonstrated reasonable predictive validity (22). The risk algorithm was found to perform well in black women (C statistic = 0.79) and moderately well in black men (C statistic = 0.67) in the Atherosclerosis Risk in Communities Study (22). Resting blood pressure (mean of the second and third measurements) was assessed by certified technicians at three 1-minute intervals using random zero sphygmomanometers (W.A. Baum Co, Copiague, NY). Fasting plasma total and HDL cholesterol levels were measured using enzymatic assays described elsewhere (coefficient of variation: ≤2%for total cholesterol level and ≤3% for HDL cholesterol level) (23). Participants were considered to have diabetes if they reported having diabetes or had fasting glucose concentrations of 126 mg/dL or higher. Trained interviewers obtained information on medication use. Smoking was assessed via self-report as current smoker (yes/no).

Covariates

Race/ethnicity was measured by self-report, and participants were categorized as black or white. Childhood SEP was assessed by self-reported father's occupation, categorized as manual versus nonmanual. Adulthood SEP was assessed by self-reported educational attainment (≤12, 13–16, and ≥17 years), family income (continuous variable), and employment status (employed full-time or part-time, including keeping house or raising children full-time, versus unemployed). Body mass index was derived from weight and height (kg/m^2), measured by certified technicians. Marital status was defined as currently married or living-as-married versus not married. Depressive symptomatology were measured using the 20-item CES-D questionnaire. Anger-out expression was measured by the anger-out subscale of the State-Trait Anger Expression Inventory (Cronbach $\alpha = 0.77$) by Spielberger et al. (24), where higher scores represent greater anger-out expression. Social support was assessed by an eight-item summative scale adapted from Schuster et al. (25), which includes both supportive and negative social interactions (Cronbach $\alpha = 0.80$), where elevated scores correspond with lower social support. Antihypertensive and cholesterol-

lowering medications were assessed via self-report. With regard to the inclusion of antihypertensive and cholesterol-lowering medications as covariates, this was done in an effort to evaluate how medication use may influence the relation between childhood family psychosocial environment and CHD risk outcomes, including the calculated 10-year CHD risk, as well as individual outcomes such as systolic blood pressure, diastolic blood pressure, total cholesterol level, and HDL cholesterol level. If participants with adverse childhood family environments were less likely to seek medical care or less likely to adhere to medication prescriptions, their blood pressure or cholesterol levels may be higher than participants with a nurturing childhood family environment due in part to the lack of medication-controlled blood pressure and cholesterol levels.

Statistical Analyses

Descriptive statistics were generated for dependent variables and covariates in men and women, according to quartiles of risky family score (see Table 1 for risky family score range within quartiles). Multivariable-adjusted regression analyses evaluated associations of the risky family score with the calculated 10-year CHD risk. The risky family score was entered as a continuous variable with a range from 0 to 21 in primary analyses. Sensitivity analyses used summed *z* scores of individual risky family score items as a continuous variable instead of the raw score range from 0 to 21. The calculated 10-year CHD risk was used as a continuous variable. Linear regression analyses were performed to evaluate associations between risky family score and the calculated 10-year CHD risk. The distribution of the 10-year CHD risk was strongly skewed and was hence log (natural) transformed. To maintain the original units of the CHD risk algorithm (units are percent risk for incident CHD during the upcoming 10 years), regression coefficients (β's) were exponentiated and reported in results as the percent change in untransformed calculated CHD risk per 1-unit increase in risky family score [(exp(β) − 1) × 100].

Secondary analyses evaluated associations of risky family score with individual CHD risk factors using multivariable-adjusted linear regression for continuous dependent variables (systolic blood pressure, diastolic blood pressure, total cholesterol level, and HDL cholesterol level) and logistic regression for the categorical dependent variable (smoking). Analyses were not performed for associations of risky family score with diabetes because of the low prevalence of dependent variables and resulting insufficient statistical power for multivariable-adjusted analyses.

For secondary analyses assessing associations of individual questions of the risky family score (score, 0–3) with the calculated 10-year CHD risk, multivariable regression analyses were performed comparing dichotomous measures of the risky family score (score, 0 versus 1–3).

We assessed whether education, income, depressive symptomatology, anger-out expression, social support, or body mass index was a potential mediator in the association between family psychosocial environment and 10-year CHD risk using a multiple mediation model. This model simultaneously estimates the association between each mediator and the family psychosocial environment exposure, along with the change in the outcome (10-year CHD risk) associated with the mediator in the fully adjusted model, and calculates an indirect

effect using the product of the coefficients method described in detail elsewhere (26,27). The indirect effect is the reduction in the association of the exposure on the outcome due to the potential mediator. The indirect effects for each individual mediator are summed to estimate the total indirect effect. Examining the individual indirect effect provides evidence of whether childhood family psychosocial environment may exert its effects uniquely through any of the mediators examined in this study. Confidence intervals (CIs) were estimated by the use of the bias-corrected bootstrapping procedure with 5000 resamples. Bootstrapping avoids the common mistaken assumptions that the indirect effects are normally distributed and symmetrical (26,27). Statistical significance was determined by examining whether zero was within the 95% CIs. This approach does not allow for evaluation of dichotomous mediators; consequently, these mediation analyses did not evaluate the potential role of marital status (married/not married), employment status (employed versus unemployed), use of antihypertensive medication (yes/no), or use of cholesterol-lowering medications (yes/no). Secondary analyses adjusting for these variables provided some evidence on the potential contribution of these continuous variables as mechanisms explaining associations between family psychosocial environment and 10-year CHD risk.

Formal statistical tests for interaction between risky family score and sex and between early family adversity score and race/ethnicity demonstrated no interaction for either sex ($p = .73$) or race ($p = .26$) with early family adversity score for the association with calculated 10-year CHD risk. However, as previous studies suggested possible sex differences in associations of early family adversity with cardiovascular outcomes (28,29), analyses were performed sex-specifically. Analyses were conducted using SAS version 9.1.3 (SAS Institute, Cary, NC).

RESULTS

The age range of participants was 33 to 45 years (mean, 40.0 years). In unadjusted analyses in women, risky family score quartile was inversely related to HDL cholesterol level, marital status, father's occupation grade, own educational attainment, and family income and was directly related to smoking, body mass index, diabetes, cholesterol-lowering medication use, depressive symptomatology, anger-out expression, social support score, and the calculated 10-year CHD risk (Table 1). In unadjusted analyses in men, risky family score quartile was inversely related to marital status, education, father's occupational grade, and family income and was directly related to smoking, depressive symptomatology, anger-out expression, social support score, unemployment status, and calculated 10-year risk for CHD. There was evidence that men with lower risky family scores were more likely to be of white versus black race/ethnicity.

In multivariable-adjusted regression analyses adjusting for the likely confounders age, race/ethnicity, and childhood SEP, a 1-unit increase in the risky family score (range, 0–21) was associated with a 1.0% (95% CI = 0.4%–1.7%) increase in the calculated 10-year risk for CHD in women and with a 1.0% (95% CI = 0.2%–1.8%) increase in the calculated 10-year risk for CHD in men (Table 2). In these analyses, β is interpreted as the percent change in calculated CHD risk per 1-unit increase in risky family score. For example, the mean calculated 10-year CHD risk for men in our study is 5.10%; consequently, a 1-unit increase in the risky family score would increase a CHD risk of 5.10% by 1.0%, resulting in a new

10-year CHD risk of 5.15%. With the risky family score range from 0 to 21, a plausible 5-point increase in score would correspond to a 5.1% ([exp(5 × 0.01) − 1] × 100) increase in calculated CHD risk, bringing the absolute calculated CHD risk in men from 5.10% to 5.36%. In an effort to evaluate the potential mechanisms by which childhood psychosocial family environment may influence CHD risk, multiple mediation tests demonstrated that, in women, education, income, depressive symptomatology, and anger-out expression had significant indirect effects, suggesting that they may be possible mediators between risky family score and CHD risk (Table 3). In men, only own educational attainment demonstrated significant indirect effects consistent with being a potential mediator. Secondary analyses adjusted for covariates provided similar findings where adjustment for adulthood SEP (education, income, and employment status) and psychosocial variables (depressive symptomatology, social support, and anger-out expression) reduced effect sizes between risky family score and CHD risk (Table 2). Adjusting for either marital status or CHD risk factors not included in the CHD risk algorithm (body mass index, cholesterol-lowering medications, and antihypertensive medications) had minimal impact on the effect size (Table 2). Sensitivity analyses used summed *z* scores of individual risky family score items as a continuous variable instead of the raw score range from 0 to 21 and showed similar findings (Appendix 1).

In an effort to evaluate which components of the Framingham algorithm (systolic and diastolic blood pressure, total and HDL cholesterol levels, diabetes, and smoking) may be most strongly related to childhood family psychosocial environment, we used multivariable-adjusted regression analyses to evaluate associations of risky family score with these variables. As shown in Table 2, there were strong positive associations between risky family score and cigarette smoking in women and men, after adjusting for age, race/ethnicity, and childhood SEP. There was an inverse association in women of risky family score with HDL cholesterol level; there was no association in men (Table 2).

To evaluate which of the seven components of the risky family questionnaire may be particularly strongly associated with CHD risk, we evaluated associations of each questionnaire item with calculated 10-year CHD risk score (Table 4). In analyses adjusting for age, race/ethnicity, and childhood SEP, in women, it seemed that responses to “How often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened,” “Did your family know what you were up to,” “How often did a parent or other adult in the household push, grab, shove, or hit you so hard you had marks or were injured,” and “Would you say that the household you grew up in was well-organized and well-managed” were particularly related to the CHD risk score (Table 3). In men, responses to “Did your family know what you were up to” and “Would you say that the household you grew up in was well-organized and well-managed” were associated with the CHD risk score. This demonstrated that it was not only potentially abusive factors (e.g., how often marked from getting hit, how often sworn at or insulted) that were related to CHD risk but also family situations related to parental attentiveness and household organization/management.

DISCUSSION

This study suggested that childhood family psychosocial environment is modestly positively associated with calculated 10- year CHD risk, as measured using the Framingham CHD risk algorithm. It seems that the components of the Framingham algorithm most strongly related to family psychosocial environment were cigarette smoking in men and women, and HDL in women. Mediating mechanisms may include the potential negative impact of childhood family psychosocial environment on later-life SEP (e.g., education in men and women, income in women) and/or psychosocial functioning (e.g., depression and anger-out expression in women), which may in turn lead to higher CHD risk, particularly through smoking (in men and women) and low level of HDL cholesterol (in women).

Prior Literature

The findings from this study on associations between risky family score and CHD risk were in general agreement with most other related investigations. For example, Dong et al. (6) demonstrated in 17,337 participants of the Adverse Childhood Experiences Study that there were strong positive cross-sectional associations of self-reported adverse childhood experiences with self-reported ischemic heart disease, where participants who had the highest Adverse Childhood Experiences score (score, 7–8) demonstrated an odds ratio (OR) of ischemic heart disease of 3.6 (95% CI = 2.4–5.3) compared with participants with the lowest score (score, 0), after adjusting for age, sex, race, and education. There was evidence that traditional and psychosocial risk factors were important explanatory mechanisms because further adjustment for smoking, physical inactivity, body mass index, diabetes, hypertension, anger, and depressed affect reduced the OR to 2.3 (95% CI = 1.5–1.9). In the Harvard Mastery of Stress Study (116 men), feelings of warmth and closeness from parents measured at approximately age 20 years were prospectively inversely associated with physician-diagnosed CHD and/or hypertension ($p < .004$) measured approximately 35 years later (analyses only statistically accounted for age) (30). In the Dunedin Study, Melchior et al. (31) demonstrated some inconsistent associations of childhood maltreatment with cardiovascular risk clustering. In the CARDIA Study, a number of investigations evaluated associations of childhood SEP and risky families with cardiovascular risk markers using structural equation modeling path analyses. Findings demonstrated that childhood SEP was related to early childhood family adversity, which was in turn associated with inhibited psychosocial functioning/negative emotionality, which was associated with worsened metabolic functioning (13), C-reactive protein (32), and blood pressure (33). This current study adds to the previous CARDIA investigations by demonstrating associations with a validated CHD risk (Framingham algorithm) that included behaviors (smoking) and biomarkers (total cholesterol level, HDL cholesterol level, diabetes, systolic blood pressure, and diastolic blood pressure), as well as demonstrating multivariable-adjusted associations between risky family score and some of the individual CHD risk factors not previously reported, including smoking, total cholesterol level, and HDL cholesterol level. Some sex differences have been reported in other studies. For example, Batten et al. (28) showed in the National Comorbidity Study (2697 women and 2697 men) that women who had been exposed to childhood maltreatment had elevated OR of self-reported cardiovascular risk/ disease (measured as self-reported high blood pressure/hypertension, myocardial infarction,

or stroke; OR = 8.8, $p < .001$ for maltreatment versus no maltreatment), whereas men did not (OR = 0.90, $p > .05$). Similarly, Almeida et al. (29) found in the New England Family Study that emotional care was related to calculated CHD risk (using the Framingham algorithm) in women ($n = 168$; $p = .004$) and not in men ($n = 99$; $p = .24$). In studies that performed sex-specific analyses, there were generally consistent inverse associations between childhood family psychosocial environment and CHD risk in women, whereas in men, findings tend to be inconsistent (28–30). Our study demonstrated inverse associations in women and men; the associations in men may have been found due in part to fairly high statistical power ($n = 1584$) compared with at least one study that showed a null association ($n = 99$) (29). Overall, the current study adds to the literature confirmatory evidence that childhood family psychosocial environment may be related to CHD risk. Our results suggest a fairly small effect, unlike some previous studies that suggest large effects (6,28). Outcomes for cardiovascular risk vary substantially between studies, and studies that evaluate prevalent/incident CHD often show stronger associations than studies that evaluate CHD risk, such as through CHD risk score algorithms or multimarker cardiovascular clustering. Studies that evaluate incident CHD events will help elucidate the potential causal relation of childhood family psychosocial environment with CHD.

Strengths and Limitations

Limitations of the study include that not all potential confounders (such as parental marital status or parental mental health) were measured. Further studies that have this information will provide additional information on the importance of other potential explanatory pathways. In addition, there is the potential for misclassification of items in the risky family questionnaire based on factors such as retrospective reporting bias or effects of mood/affect. As described in the “Methods” section, we evaluated the discriminant validity of the instrument through principal components factor analysis and found no overlap of the risky family variable with depressive symptomatology, social support score, or anger-out expression score. Other literature has further evaluated the validity and reliability of retrospective reporting for constructs including childhood SEP (17), parental support and affection (18,19), and childhood abuse (20). Overall, the possibility of misclassification by retrospective reporting bias or effects of mood/affect remains; however, the validation techniques demonstrated that the measure is reasonably robust for independence from reporting on depression, social support score, and anger-out expression score. An additional study weakness is that the CHD risk algorithm is not as accurate a measure of CHD as the measurement of CHD events themselves. However, given the relatively young age of the participants (33–45 years), it is too early in the life course to evaluate associations with CHD events in this study. Childhood family psychosocial environment may affect a wide range of CHD risk factors. Consequently, by using a validated CHD prediction algorithm that encompasses a variety of CHD risk factors, it allows evaluation of a variety of systems that may be simultaneously influenced by childhood family psychosocial environment. The relations between childhood family psychosocial environment and the individual components of the CHD risk algorithm provide additional, more specific information as to the risk of early family adversity on each individual CHD risk factor. Finally, although there was no evidence of racial/ethnic differences in the main effects of childhood family psychosocial environment on CHD risk (formal tests for interaction with race/ethnicity

demonstrated $p = .26$), future analyses could explore whether the mechanisms differ by race/ethnicity, including specifically for black men, white men, black women, and white women.

With regard to strengths, this study used a large sample size ($n = 3554$) and was diverse in race/ethnicity (black and white), sex, and SEP. There are few available data on the relation between childhood family psychosocial environment and CHD risk; consequently, this study helps to move the field forward in providing evidence of potential positive associations between childhood psychosocial environment and 10-year risk for CHD. The biologic outcome measures and covariates were measured using rigorous quality control/quality assurance protocols.

CONCLUSIONS

This study demonstrated that a measure of childhood family psychosocial environment was positively associated with the calculated 10-year risk for CHD using the Framingham risk algorithm. The components of the CHD risk factor algorithm most related to childhood family psychosocial environment were cigarette smoking in women and men, and HDL cholesterol level in women. Mechanisms may include the potential negative impact of childhood family psychosocial environment on later-life SEP (e.g., education in men and women) and/or psychosocial functioning (e.g., depression and anger-out expression in women), which may in turn lead to higher CHD risk, particularly through smoking (in men and women) and low level of HDL cholesterol (in women).

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Glossary

CHD	coronary heart disease
SEP	socioeconomic position
CARDIA	Coronary Artery Risk Development in Young Adults
HDL	high-density lipoprotein
CES-D	Center for Epidemiologic Studies Depression scale
CV	Coefficient of variation

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APPENDIX 1

Logistic and Linear Regression Analyses Demonstrating Associations of Risky Family *z* Score With Calculated 10-Year CHD Risk and CHD Risk Factors

	Model Adjustment													
	Age, Race/Ethnicity		**Age, Race/Ethnicity, Childhood SEP**		**Age, Race/Ethnicity, Childhood SEP, Marital Status**		**Age, Race/Ethnicity, Childhood SEP, CHD Risk Factors**		**Age, Race/Ethnicity, Childhood SEP, Adulthood SEP**		**Age, Race/Ethnicity, Childhood SEP, Psychosocial Functioning**		**All Covariates**	
	PE	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**
10-y CHD risk, %														
Women	**0.9**	**0.4 to 1.4**	**1.0**	**0.4 to 1.5**	**1.0**	**0.4 to 1.5**	**0.8**[a]	**0.2 to 1.3**	0.5	−0.06 to 1.1	0.3	−0.3 to 0.9	0.1	−0.5 to 0.7
Men	**0.9**	**0.2 to 1.6**	**0.9**	**0.1 to 1.6**	**0.9**	**0.1 to 1.6**	**0.8**[a]	**0.1 to 1.5**	0.5	−0.2 to 1.3	0.6	−0.3 to 1.4	0.2	−0.6 to 1.0
Systolic blood pressure, mm Hg														
Women	0.05	−0.08 to 0.17	−0.02	−0.16 to 0.13	−0.03	−0.18 to 012	−0.05	−0.19 to 0.08	−0.09	−0.24 to 0.05	−0.09	−0.25 to 0.07	−0.10	−0.25 to 0.05
Men	0.04	−0.12 to 0.21	0.05	−0.13 to 0.24	0.02	−0.16 to 0.21	−0.01	−0.19 to 0.16	−0.01	−0.19 to 0.18	0.05	−0.16 to 0.25	−0.02	−0.22 to 0.18
Diastolic blood pressure, mm Hg														
Women	0.03	−0.07 to 0.13	0.01	−0.10 to 0.12	0.00	−0.11 to 0.11	0.05	−0.03 to 0.12	−0.02	−0.14 to 0.10	−0.04	−0.16 to 0.09	0.03	−0.06 to 0.11
Men	−0.03	−0.16 to 0.10	−0.01	−0.15 to 0.14	−0.02	−0.17 to 0.13	−0.03	−0.13 to 0.07	−0.03	−0.19 to 0.12	−0.02	−0.18 to 0.14	−0.04	−0.15 to 0.08
Total cholesterol level, mg/dL														
Women	−0.11	−0.41 to 0.19	−0.20	−0.54 to 0.14	−0.18	−0.52 to 0.17	−0.15	−0.48 to 0.18	−0.18	−0.53 to −0.17	−0.25	−0.63 to 0.13	−0.15	−0.53 to 0.22
Men	0.17	−0.30 to 0.64	0.20	−0.30 to 0.70	0 to 25	−0.25 to 0.76	0.19	−0.31 to 0.69	0.23	−0.28 to 0.74	0.14	−0.42 to 0.70	0.09	−0.48 to 0.65
HDL cholesterol level, mg/dL														
Women	**−0.16**	**−0.29 to −0.03**	**−0.19**	**−0.34 to −0.04**	**−0.20**	**0.35 to 0.05**	−0.09	−0.23 to 0.04	−0.12	−0.28 to 0.03	**−0.17**	**−0.32 to −0.02**	−0.05	−0.21 to 0.10
Men	0.00	−0.15 to 0.15	0.02	−0.15 to 0.18	0.00	−0.17 to 0.17	0.03	−0.13 to 0.18	−0.00	−0.17 to 0.16	0.01	−0.17 to 0.16	−0.04	−0.21 to 0.14
Current smoker														
Women	**1.06**	**1.03 to 1.08**	**1.06**	**1.03 to 1.08**	**1.05**	**1.02 to 1.08**	**1.05**	**1.02 to 1.08**	**1.03**	**1.00 to 1.06**	1.02	0.99 to 1.05	1.00	0.97 to 1.04
Men	**1.06**	**1.03 to 1.09**	**1.06**	**1.03 to 1.09**	**1.05**	**1.02 to 1.08**	**1.06**	**1.03 to 1.10**	1.03	0.99 to 1.06	1.03	0.99 to 0.07	1.00	0.96 to 1.04

Point estimates (PEs) represent either odds ratios (only for the categorical variables education and current smoker; odds ratios represent change in odds of dependent variable per 1-unit increase in the risky family *z* score) or regression coefficients (for all other dependent variables, representing increase in dependent variable per 1-unit increase in risky family *z* score).

CHD risk factors include smoking, body mass index, systolic blood pressure, total cholesterol level, HDL cholesterol level, diabetes, cholesterol-lowering medications, and antihypertensive medications.

Adulthood SEP variables includes education, family income, and employment status.

Psychosocial functioning variables include depressive symptomatology, anger-out expression, positive social contacts, and negative social contacts.

When covariate is the dependent variable, it is not adjusted for itself.

[a]Adjusted for CHD risk factors not included in the CHD risk algorithm, including body mass index, cholesterol-lowering medications, and antihypertensive medications.

CHD = coronary heart disease; SEP = socioeconomic position; CI = confidence interval; HDL = high-density lipoprotein.

TABLE 1

Means and Proportions (95% Confidence Intervals) According to Quartile of Risky Family Score: CARDIA Study, United States, 2000 to 2001

	Women					Men				
	Quartile of Risky Family Score					Quartile of Risky Family Score				
	1	2	3	4		1	2	3	4	
	Range, 0–1 (*n* = 549)	Range, 2–3 (*n* = 385)	Range, 4–6 (*n* = 460)	Range, 7–21 (*n* = 576)	Overall	Range, 0–1 (*n* = 382)	Range, 2–3 (*n* = 383)	Range, 4–6 (*n* = 411)	Range, 7–19 (*n* = 408)	Overall
Age, y	39.8 (39.5–40.1)	40.2 (39.8–40.6)	39.9 (39.6–40.3)	40.2 (39.9–40.5)	40.0 (39.9–40.2)	39.9 (39.6–40.3)	40.0 (39.6–40.3)	39.9 (39.5–40.2)	40.2 (39.9–40.6)	40.0 (39.9–40.2)
Race/ethnicity, % white	55.4 (51.1–59.6)	47.7 (42.6–52.8)	44.0 (39.4–48.7)	53.9 (49.7–58.0)	50.8 (48.5–53.0)	62.7 (57.7–67.6)	58.0 (52.8–63.0)	52.6 (47.6–57.5)	52.6 (47.6–57.5)	56.4 (53.9–58.9)
Smoker, %	14.6 (11.7–17.8)	16.6 (13.0–20.7)	22.4 (18.7–26.5)	26.2 (22.7–30.0)	20.2 (18.5–22.0)	16.0 (12.4–20.0)	19.3 (15.5–23.6)	29.0 (24.6–33.6)	30.4 (26.0–35.1)	23.9 (21.8–26.0)
HDL cholesterol level, mg/dL	55.9 (54.8–57.1)	56.6 (55.1–58.1)	54.3 (53.1–55.5)	54.1 (52.9–55.4)	55.2 (54.5–55.8)	45.2 (44.0–46.4)	44.7 (43.4–45.9)	45.7 (44.4–46.9)	45.2 (43.9–46.5)	45.2 (44.5–45.8)
Total cholesterol level, mg/dL	181 (178–183)	183 (180–186)	182 (179–185)	181 (178–183)	181 (180–183)	188 (184–192)	189 (186–193)	188 (184–192)	190 (186–193)	189 (187–191)
Body mass index, kg/m^2	28.4 (27.7–30.0)	29.0 (28.3–30.0)	29.3 (28.6–30.0)	29.5 (28.8–30.1)	29.0 (28.7–29.4)	28.0 (27.5–28.4)	28.4 (27.9–28.9)	27.9 (27.4–28.3)	28.4 (27.9–28.9)	28.2 (27.9–28.4)
Systolic blood pressure, mm Hg	111 (109–112)	111 (110–113)	112 (110–113)	111 (110–113)	111 (110–112)	114 (113–115)	116 (114–117)	117 (116–119)	116 (114–117)	116 (115–116)
Diastolic blood pressure, mm Hg	72.6 (71.6–73.5)	72.2 (71.0–73.4)	73.2 (72.1–74.2)	72.8 (71.8–73.7)	72.7 (72.2–73.2)	75.8 (74.8–76.9)	77.3 (76.1–78.5)	77.2 (76.0–78.3)	76.6 (75.5–77.6)	76.7 (76.1–77.2)
Diabetes, %	6.9 (4.9–9.4)	6.0 (3.8–8.8)	8.9 (6.5–11.9)	10.2 (7.9–13.0)	8.2 (7.0–9.5)	3.4 (1.8–5.7)	3.9 (2.2–6.4)	4.6 (2.8–7.1)	6.1 (4.0–8.9)	4.6 (3.6–5.7)
Taking antihypertensive medication, %	6.9 (4.9–9.4)	7.8 (5.3–10.9)	9.8 (7.2–12.9)	7.1 (5.1–9.5)	7.8 (6.7–9.0)	7.1 (4.7–10.0)	8.4 (5.8–11.6)	9.0 (6.4–12.2)	6.6 (4.4–9.5)	7.8 (6.5–9.2)
Taking cholesterol-lowering medication, %	0.9 (0.3–2.1)	0.0 (0.0–1.0)	1.3 (0.5–2.8)	2.3 (1.2–3.8)	1.2 (0.8–1.8)	3.4 (1.8–5.7)	3.9 (2.2–6.4)	4.1 (2.4–6.5)	3.7 (2.1–6.0)	3.8 (3.0–4.8)
Father's occupation, % manual	36.6 (32.2–41.1)	41.5 (36.1–47.0)	48.1 (42.9–53.3)	49.5 (44.8–54.1)	43.8 (41.4–46.3)	31.4 (26.6–36.6)	39.5 (34.1–45.0)	43.5 (38.1–48.9)	43.6 (38.3–49.1)	39.4 (36.8–42.1)
Education, % >16 y	24.9 (21.3–28.8)	21.8 (17.8–26.2)	19.8 (16.2–23.7)	17.4 (14.4–20.7)	20.9 (19.1–22.8)	29.2 (24.7–34.1)	19.8 (16.0–24.2)	18.5 (14.8–22.5)	15.1 (11.8–19.0)	20.5 (18.6–22.6)
Current employment, % employed	90.3 (87.5–92.7)	88.6 (85.0–91.6)	92.0 (89.1–94.3)	88.2 (85.2–90.7)	89.7 (88.3–91.0)	95.5 (92.9–97.4)	93.2 (90.2–95.5)	91.5 (88.4–94.0)	88.5 (85.0–91.4)	92.1 (90.7–93.4)
Family income, US $1000	72.7 (69.4–76.0)	68.1 (64.0–72.2)	61.4 (57.9–65.0)	55.7 (52.5–58.9)	64.2 (62.4–66.0)	82.5 (78.4–86.7)	71.6 (67.5–75.6)	68.0 (64.2–71.8)	62.6 (58.5–66.6)	71.0 (68.9–73.0)
Marital status, % married	66.2 (62.0–70.1)	57.4 (52.3–62.4)	59.8 (55.1–64.3)	51.3 (47.1–55.5)	58.6 (56.4–60.8)	71.8 (66.9–76.2)	69.7 (64.8–74.3)	58.6 (53.7–63.4)	55.2 (50.2–60.0)	63.6 (61.1–65.9)
CES-D score	6.9 (6.3–7.5)	8.4 (7.7–9.2)	10.5 (9.7–11.2)	12.3 (11.6–13.1)	9.6 (9.3–10.0)	5.8 (5.3–6.3)	7.6 (7.0–8.2)	8.7 (8.0–9.3)	11.5 (10.6–12.3)	8.4 (8.1–8.8)
Anger-out expression score	5.4 (5.2–5.7)	6.0 (5.6–6.3)	6.2 (5.9–6.5)	6.5 (6.2–6.7)	6.0 (5.9–6.2)	4.9 (4.6–5.2)	5.4 (5.1–5.7)	5.6 (5.3–5.8)	6.0 (5.7–6.4)	5.5 (5.3–5.6)
Social support score	4.2 (4.0–4.5)	5.6 (5.3–5.9)	6.6 (6.3–6.9)	8.5 (8.2–8.9)	6.3 (6.1–6.5)	4.1 (3.8–4.4)	5.4 (5.1–5.7)	6.1 (5.8–6.4)	8.5 (8.1–8.9)	6.1 (5.9–6.2)
Calculated 10-y CHD risk	1.6 (1.5–1.7)	1.6 (1.5–1.7)	1.7 (1.6–1.8)	1.8 (1.7–2.0)	1.8 (1.7–1.9)	4.8 (4.5–5.1)	5.2 (4.3–5.5)	5.2 (4.9–5.5)	5.5 (5.2–5.9)	5.1 (5.0–5.3)

CARDIA = Coronary Artery Risk Development in Young Adults; HDL = high-density lipoprotein; CES-D = Center for Epidemiologic Studies Depression scale; CHD = coronary heart disease.

TABLE 2

Logistic and Linear Regression Analyses Demonstrating Associations of Risky Family Score With Calculated 10-Year CHD Risk and CHD Risk Factors

	Model Adjustment													
	Age, Race/Ethnicity		**Age, Race/Ethnicity, Childhood SEP**		**Age, Race/Ethnicity, Childhood SEP, Marital Status**		**Age, Race/Ethnicity, Childhood SEP, CHD risk factors**		**Age, Race/Ethnicity, Childhood SEP, Adulthood SEP**		**Age, Race/Ethnicity, Childhood SEP, Psychosocial Functioning**		**All Covariates**	
	PE	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**	**PE**	**95% CI**
10-y CHD risk, %														
Women	**1.0**	**0.4 to 1.6**	**1.0**	**0.4 to 1.7**	**0.8**	**0.2 to 1.4**	**1.0**[a]	**0.4 to 1.6**	0.5	−0.1 to 1.2	0.3	−0.4 to 1.0	0.1	−0.5 to 0.8
Men	**1.0**	**0.3 to 1.8**	**1.0**	**0.2 to 1.8**	**0.9**	**0.2 to 1.7**	**1.0**[a]	**0.1 to 1.8**	0.6	−0.2 to 1.4	0.6	−0.3 to 1.5	0.3	−0.6 to 1.1
Systolic blood pressure, mm Hg														
Women	0.04	−0.10 to 0.19	−0.03	−0.19 to 0.13	−0.06	−0.21 to 0.09	−0.04	−0.20 to 0.12	−0.11	−0.27 to 0.05	−0.11	−0.29 to 0.07	−0.11	−0.28 to 0.06
Men	0.04	−0.14 to 0.22	0.05	−0.15 to 0.26	−0.02	−0.21 to 0.17	0.02	−0.18 to 0.22	−0.02	−0.22 to 0.19	0.04	−0.18 to 0.27	−0.03	−0.25 to 0.19
Diastolic blood pressure, mm Hg														
Women	0.03	−0.09 to 0.14	0.00	−0.12 to 0.13	0.05	−0.04 to 0.13	−0.00	−0.13 to 0.12	−0.03	−0.16 to 0.10	−0.06	−0.19 to 0.09	0.02	−0.07 to 0.12
Men	−0.05	−0.19 to 0.11	−0.01	−0.17 to 0.15	−0.03	−0.14 to 0.08	−0.03	−0.19 to 0.14	−0.04	−0.21 to 0.12	−0.03	−0.21 to 0.16	−0.04	−0.16 to 0.08
Total cholesterol level, mg/dL														
Women	−0.11	−0.45 to 0.22	−0.22	−0.59 to 0.16	−0.16	−0.53 to 0.20	−0.19	−0.57 to 0.19	−0.19	−0.58 to 0.20	−0.26	−0.69 to −0.16	−0.16	−0.57 to 0.25
Men	0.19	−0.32 to 0.71	0.22	−0.33 to 0.75	0.21	−0.34 to 0.76	0.27	−0.29 to 0.83	0.25	−0.31 to 0.81	0.14	−0.48 to 0.76	0.09	−0.53 to 0.71
HDL cholesterol level, mg/dL														
Women	**−0.18**	**−0.32 to −0.03**	**−0.21**	**−0.37 to −0.04**	−0.10	−0.26 to 0.05	**−0.21**	**−0.38 to −0.04**	−0.13	−0.30 to 0.04	−0.10	−0.28 to 0.08	−0.06	−0.23 to 0.11
Men	0.01	−0.18 to 0.15	0.01	−0.17 to 0.20	0.02	−0.15 to 0.19	−0.01	−0.19 to 0.18	−0.01	−0.20 to 0.17	−0.03	−0.24 to 0.17	−0.05	−0.24 to 0.14
Current smoker														
Women	**1.06**	**1.04 to 1.09**	**1.06**	**1.03 to 1.09**	**1.06**	**1.03 to 1.09**	**1.05**	**1.02 to 1.08**	1.03	1.00 to 1.06	1.02	0.99 to 1.06	1.00	0.97 to 1.04
Men	**1.07**	**1.03 to 1.10**	**1.07**	**1.03 to 1.11**	**1.07**	**1.04 to 1.11**	**1.06**	**1.02 to 1.09**	1.03	0.99 to 1.07	1.03	0.99 to 1.08	1.00	0.96 to 1.05

Point estimates (PEs) represent either odds ratios (for smoking, where odds ratios represent change in odds of smoking per 1-unit increase in the child adversity score) or regression coefficients (for all other dependent variables, representing increase in dependent variable per 1-unit increase in risky family score). Range of risky family score is from 0 to 21. Bold text represents statistically significant effect size at $p < .05$.

CHD risk factors include smoking, body mass index, systolic blood pressure, total cholesterol level, HDL cholesterol level, diabetes, cholesterol-lowering medications, and antihypertensive medications.

Adulthood SEP variables includes education, family income, and employment status.

Psychosocial functioning variables include depressive symptomatology, anger-out expression, positive social contacts, and negative social contacts.

When covariate is the dependent variable, it is not adjusted for itself.

[a]Adjusted for CHD risk factors not included in the CHD risk algorithm, including body mass index, cholesterol-lowering medications, and antihypertensive medications.

CHD = coronary heart disease; SEP = socioeconomic position; CI = confidence interval; HDL = high-density lipoprotein.

TABLE 3

Mediation Tests Evaluating the Indirect Effects of Risky Family Score on the Calculated 10-Year Coronary Heart Disease Risk

	Women		Men	
Mediators	**PE, %**	**95% CI**	**PE, %**	**95% CI**
Own education	**0.19**	**0.1 to 0.3**	**0.43**	**0.25 to 0.66**
Family income	**0.13**	**0.03 to 0.27**	0.11	0.00 to 0.26
CES-D score	**0.22**	**0.04 to 0.43**	0.21	−0.03 to 0.05
Anger-out expression score	**0.07**	**0.01 to 0.17**	0.07	−0.03 to 0.20
Social support score	0.08	−0.22 to 0.39	−0.02	−0.41 to 0.41
Body mass index	0.19	−0.06 to 0.43	0.08	−0.22 to 0.41
Total indirect effect	**0.88**	**0.45 to 1.32**	**0.87**	**0.35 to 1.47**

Models include all potential mediators, adjusted for age, race/ethnicity, and childhood socioeconomic position. Bold text represents statistically significant effect size at $p < .05$.
Point estimates (PEs) represent percent change in CHD risk during the next 10 years per unit of risky family score, due to the mediating variable.

CI = confidence interval; CES-D = Center for Epidemiologic Studies Depression scale.

TABLE 4

Regression Analyses of the Association Between Individual Risky Family Questions and 10-Year CHD Risk

	Model Adjustment													
	Age, Race/Ethnicity		Age, Race/Ethnicity, Childhood SEP		Age, Race/ Ethnicity, Childhood SEP, Marital Status		Age, Race/Ethnicity, Childhood SEP, CHD Risk Factors		Age, Race/Ethnicity, Childhood SEP, Adulthood SEP		Age, Race/Ethnicity, Childhood SEP, Psychosocial Functioning		All Covariates	
	β[a]	95% CI	β[a]	95% CI	β[a]	95% CI	β[a]	95% CI	β[a]	95% CI	β[a]	95% CI	β[a]	95% CI
Did your family know what you were up to?														
Women	**6.9**	**1.8 to 12.3**	**5.9**	**0.4 to 11.7**	**6.3**	**0.4 to 12.6**	**5.9**	**0.4 to 11.6**	3.0	−2.1 to 8.3	1.6	−3.8 to 7.4	1.1	−4.1 to 6.3
Men	**6.9**	**1.1 to 13.1**	**7.0**	**0.8 to 13.7**	4.9	0.0 to 10.2	**6.8**	**0.6 to 13.5**	3.4	−2.8 to 9.9	5.1	−1.1 to 11.9	1.5	−4.4 to 7.7
How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?														
Women	5.0	−0.1 to 10.4	4.6	−1.0 to 10.4	3.1	−2.0 to 8.4	4.4	−1.1 to 10.5	1.1	−4.2 to 6.8	−0.8	−6.4 to 5.1	−1.7	−6.8 to 3.8
Men	4.2	−1.7 to 10.4	3.4	−2.9 to 10.2	3.4	−2.6 to 9.6	3.1	−3.2 to 10.0	1.5	−4.7 to 8.1	0.5	−6.1 to 7.5	−0.4	−6.5 to 6.0
How often did a parent or other adult in the household push, grab, shove, or hit you so hard you had marks or were injured?														
Women	**11.9**	**5.2 to 18.9**	**10.5**	**3.2 to 18.3**	**8.8**	**2.1 to 15.7**	**10.5**	**3.0 to 18.2**	6.5	−0.5 to 14.0	4.7	−2.4 to 12.3	3.1	−3.3 to 9.9
Men	1.3	−5.9 to 9.0	2.4	−5.4 to 11.1	2.1	−5.3 to 10.1	2.2	−5.6 to 10.8	1.0	−6.8 to 9.4	−0.1	−8.0 to 8.4	−0.9	−8.1 to 6.9
How often did a parent or other adult in the household express physical affection for you, such as hugging or other physical gesture of warmth and affection?														
Women	1.9	−3.1 to 7.1	2.1	−3.2 to 7.9	1.4	−3.5 to 6.6	2.1	−3.2 to 7.9	0.6	−4.7 to 6.1	−2.4	−7.7 to 3.4	−2.3	−7.1 to 2.9
Men	0.6	−5.2 to 6.6	1.2	−5.2 to 7.9	0.6	−5.3 to 6.7	0.8	−5.4 to 7.5	−0.5	−6.7 to 6.0	−0.8	−7.0 to 8.1	−2.4	−8.1 to 3.8
Did you live with anyone who was a problem drinker or alcoholic, or who used street drugs?														
Women	−0.1	−5.3 to 5.3	0.5	−5.2 to 6.5	1.1	−4.1 to 6.6	0.2	−5.4 to 6.3	−0.6	−5.8 to 5.2	−1.1	−6.7 to 4.8	−0.2	−5.4 to 5.2
Men	3.7	−2.7 to 10.3	4.0	−3.0 to 11.3	6.0	−0.6 to 13.0	3.8	−3.1 to 11.2	2.6	−4.2 to 9.9	2.2	−4.6 to 9.5	3.4	−3.1 to 10.3
How often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened?														
Women	**9.5**	**4.2 to 15.1**	**9.6**	**3.9 to 15.7**	**7.4**	**2.1 to 12.9**	**9.6**	**3.9 to 15.7**	**6.6**	**1.1 to 12.7**	4.5	−1.3 to 10.5	2.4	−2.8 to 7.9
Men	4.8	−1.0 to 11.0	5.5	−0.8 to 12.2	5.1	−0.8 to 11.3	5.4	−0.9 to 12.2	4.0	−2.3 to 10.5	3.5	−3.0 to 10.3	2.5	−3.3 to 8.9
Would you say that the household you grew up in was well-organized and well-managed?														
Women	**7.8**	**2.6 to 13.2**	**7.6**	**1.9 to 13.5**	3.7	−1.3 to 9.0	**7.5**	**1.8 to 13.5**	5.0	−0.4 to 10.5	3.8	−1.8 to 9.7	0.4	−4.6 to 5.5
Men	**7.7**	**1.8 to 13.9**	**6.6**	**0.3 to 13.3**	4.9	−0.9 to 11.1	**6.6**	**0.3 to 13.2**	5.1	−1.1 to 11.6	4.6	−1.7 to 11.4	2.2	−3.6 to 8.3

Bold text represents statistically significant effect size at $p < .05$.

CHD risk factors include body mass index, cholesterol-lowering medications, and antihypertensive medications.

Adulthood SEP variables includes education, family income, and employment status.

Psychosocial functioning variables include depressive symptomatology, anger-out expression, positive social contacts, and negative social contacts.

[a] Represents percent change in nontransformed 10-year CHD risk for the three riskiest scores (score, 1–3) compared with the least risk score (score, 0).

CHD = coronary heart disease; SEP = socioeconomic position; CI = confidence interval.