

Childhood Family Psychosocial Environment and Coronary Heart Disease Risk

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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). **Key words:** family, risky family, childhood, life course, coronary heart disease, epidemiology.

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.

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

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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: $\leq 2\%$ for total cholesterol level and $\leq 3\%$ 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 $[(\exp(\beta) - 1) \times 100]$.

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