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Childhood family psychosocial environment and carotid intima media thickness: The CARDIA study

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ABSTRACT

Little is known about whether the childhood family psychosocial environment (characterized by cold, unaffectionate interactions, conflict, aggression, neglect and/or low nurturance) affects coronary heart disease (CHD) risk. Objectives were to evaluate associations of childhood family psychosocial environment with carotid intima media thickness (IMT), a subclinical measure of atherosclerosis. The study population included 2659 CARDIA study participants, aged 37–52 years. Childhood family psychosocial environment was measured using a risky family questionnaire via self-report. Carotid IMT was calculated using the average of 20 measurements of mean common carotid, bulb and internal carotid IMT, assessed using high-resolution B-mode ultrasound images. Utilizing linear regression analyses adjusted for age, a 1-unit (range 0–21) increase in risky family score was associated with 0.0036 (95% CI: 0.0006,0.0066 mm) and 0.0020 (95% CI: 0.0002,0.0038) mm increase in mean IMT in white males and females, respectively. Formal mediation analyses and covariate adjustments suggested childhood socioeconomic position and smoking may be important mechanisms in white males and females, as well as education and depressive symptomatology in white males. No associations were found in black participants. Formal statistical tests for interaction between risky family score and sex, and between risky family score and race/ethnicity, demonstrated borderline evidence of interactions for both sex ($p = 0.12$) and race/ethnicity ($p = 0.14$) with risky family score for associations with mean IMT. In conclusion, childhood family psychosocial environment was positively associated with IMT in white participants, with little evidence of association in black participants. Mechanisms in white participants may include potential negative impacts of socioeconomic constraints on parenting quality, potentially influencing offspring's cardiovascular risk factors (e.g. smoking), socioeconomic position (e.g. education), and/or psychosocial functioning (e.g. depression), which may in turn lead to atherosclerotic processes. Borderline racial/ethnic differences in findings should be replicated, but add to literature exploring race/ethnicity-specific associations of parenting approaches with health outcomes.

Introduction

Coronary heart disease (CHD) remains a major cause of mortality world-wide (Mendis, Puska, & Norrving, 2011). There is increasing evidence that early life factors may contribute to the development of CHD, such as findings of early atherosclerotic lesions in adolescents and young adults (Berenson et al., 1998), development of CHD risk factors such as obesity, blood pressure and cholesterol in infants and children (Lynch & Smith, 2005), and evidence that early life markers such as birth weight and parental socioeconomic position may be risk markers for CHD (Lynch & Smith, 2005). An early life potential determinant of CHD that has been minimally explored is the childhood family psychosocial environment. "Risky families" is a term proposed and developed by Taylor et al. which is defined as a childhood family environment composed of cold, unaffectionate interactions, conflict, aggression,

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neglect and/or low nurturance (Taylor, Lerner, Sage, Lehman, & Seeman, 2004). The risky family questionnaire measures the family psychosocial environment including levels of family conflict, harsh restrictive parenting styles, and chaotic or neglectful parenting (Repetti, Taylor, & Seeman, 2002). This is distinct from other measures of the family psychosocial environment such as the Adverse Childhood Experiences (ACE) questionnaire that measures only exposure to more extreme parenting styles including abuse and neglect (Dong et al., 2004), and the Parental Bonding Instrument (PBI) which focuses on measuring the degree of parental care and overprotection (Parker, Tupling, & Brown, 1979). The risky family questionnaire allows for the measurement of family psychosocial environment along a continuum from caring and organized to harsh, disorganized, neglectful and abusive (Repetti et al., 2002). Early evidence suggests that risky families, or other measures of the childhood family psychosocial environment, may be associated with CHD (Dong et al., 2004; Felitti et al., 1998). Furthermore, a small number of studies suggest that the childhood family psychosocial environment may be associated with CHD risk factors/risk markers such as smoking, blood pressure and metabolic syndrome (Almeida et al., 2010; Dong et al., 2004; Lehman, Taylor, Kiefe, & Seeman, 2005, 2009; Loucks, Almeida, Taylor, & Matthews, 2011; Taylor, Lehman, Kiefe, & Seeman, 2006). However, associations of childhood family psychosocial environment with subclinical estimates of atherosclerosis, such as carotid intima media thickness (IMT), to our knowledge have not been explored.

Carotid IMT serves as a reasonable surrogate marker of atherosclerosis and biological marker of CVD risk (Lorenz, Markus, Bots, Rosvall, & Sitzer, 2007; Pignoli, Tremoli, Poli, Oreste, & Paoletti, 1986). Ultrasonic carotid IMT measurements correlate well with atherosclerotic histology (Pignoli et al., 1986). Carotid IMT is associated with cardiovascular risk factors and coronary artery disease, and serves as a strong predictor of future cardiovascular events (Lorenz et al., 2007). A meta-analysis demonstrated that for a carotid IMT difference of 0.10 mm, the future risk of myocardial infarction increased by 15% (RR = 1.15; 95% CI, 1.12,1.17); and the relative risk of stroke increased by 18% (RR = 1.18; 95% CI, 1.16,1.21) (Lorenz et al., 2007).

The primary objectives of this study were to evaluate whether the childhood family psychosocial environment, measured with a "risky family" questionnaire, is associated with carotid intima media thickness in black and white participants of a large, prospective study of United States adults, specifically the Coronary Artery Risk Development in Young Adults (CARDIA) study. Previous studies suggested possible sex differences in associations of early family adversity with cardiovascular outcomes, with evidence of stronger associations in females than males (Almeida et al., 2010; Batten, Aslan, Maciejewski, & Mazure, 2004). There has been very little exploration of racial/ethnic differences in the relation between childhood family psychosocial environment and cardiovascular disease risk. We hypothesized there would be no racial/ethnic differences in findings. It is important to understand sex- and race/ethnicity-specific associations, consequently analyses were stratified by sex and race/ethnicity.

Methods

Study sample

The CARDIA study is a multicenter, longitudinal study of CHD risk markers. At baseline assessment (1985–1986) the cohort included 5115 black and white adults aged 18–30 years, recruited from 4 metropolitan areas (Birmingham, AL, Chicago, IL, Minneapolis, MN and Oakland, CA). Participants have been regularly examined since baseline, including Examination 6 which occurred at the 15-year follow-up during the years 2000–2001 (ages 33–45 years) and Examination 7 which occurred at the 20-year follow-up during the years 2005–2006 (ages 37–52 years). Study protocols were approved by institutional review boards at each institution, and informed consent was obtained from participants.

Of the 3671 participants assessed at Examination 6, 21 participants were excluded for not having risky family score variables and 1 transgender participant was excluded, leaving 3649 participants. Of these, 490 participants did not attend Examination 7 when carotid IMT was assessed; an additional 250 attended Examination 7 but were missing carotid IMT. A further 250 participants were missing $\geq$1 covariate (171 of these were missing father and mother's education), and consequently were not included in analyses, leaving 2659 participants for complete case analyses. Due to limited availability of all required variables at each exam period, all study variables were ascertained at Examination 7, with the exception of the exposure variable (risky family questionnaire), family income, CES-D, anger expression and social support, which were obtained at Examination 6.

Independent variable

Using a risky family questionnaire adapted from Felitti et al. (Felitti et al., 1998) and further developed by Taylor et al. (Taylor et al., 2004), participants answered questions about their parents or other adults in the household during participants' childhood and adolescence (prior to age 18) using a 7-item scale, each item ranging from 0 (rarely or none of the time) to 3 (most or all of the time). The scale was created specifically in the context of the CARDIA study, for which questions were developed for a community-based sample, to assess family conflict, neglect and cold non-nurturant behavior. Items were summed (after reverse scoring where appropriate) leading to an overall scale range of 0–21, 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, lived in a well-organized, well-managed household, and whether their family knew what they were up to as children and adolescents (specific wording of questions shown in Appendix Table A).

In order 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 factor analysis. After evaluating a scree plot of eigenvalues, four derived factors were identified as (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. Other literature has further evaluated validity and reliability of retrospective reporting for constructs including childhood SES (Krieger, Okamoto, & Selby, 1998), parental support and affection (Brewin, Andrews, & Gotlib, 1993; Parker, 1989), and childhood abuse (Dill, Chu, Grob, & Eisen, 1991).

Dependent variable

At year 20 (2005–2006), carotid ultrasounds were performed by centrally-trained technicians using standard procedures (GE Logiq

700, GE Healthcare, Issaquoah, IL.) and were read at a single center (Tufts Medical Center, Boston, MA), described in more detail elsewhere (Polak et al., 2010). High-resolution B-mode ultrasound images were measured including the near and far walls of the common carotid artery (CCA), the carotid artery bulb (bulb), and the internal carotid artery (ICA), on the left and right sides of the neck. One image was acquired for each side of the CCA, and two images for each side of the bulb and ICA. The first image was taken at 45° to the vertical, and the second image was taken at 20°–25° to the vertical. Any atherosclerotic plaque was included as part of the intima media and a note was made about the extent of stenosis that existed anywhere in the right or left carotid artery. Pearson correlation coefficients based on the 58 replicate studies were 0.86 for CCA, 0.72 for the bulb, and 0.88 for the ICA (Polak et al., 2010).

Primary analyses utilized the mean of the mean measurements across all segments (4 for the CCA and 8 segments for the bulb and ICA, respectively) of both the right and the left sides, similar to the approach used in the ARIC study (Nambi et al., 2010). Secondary analyses utilized mean of the maximum measurements across all segments described above, to evaluate maximum intima media thickness, described in more detail elsewhere (Polak et al., 2010).

Covariates

With regard to race/ethnicity, the CARDIA study recruited participants in 2 nonspecific racial/ethnic categories, specifically non-Hispanic Black vs. non-Hispanic White. Race was self-reported as non-Hispanic Black or non-Hispanic White. Childhood socioeconomic position was assessed using self-reported father's or mother's education, categorized as >16 vs. ≤16 years, utilizing the highest education level when both variables were available. Adulthood socioeconomic position was assessed by self-reported educational attainment, using the categories ≤12 years (approximating ≤ high school degree), 13–16 years (estimating college degree, associates degree or some college), and ≥17 years (representing > college degree, such as MD, JD, PhD, MSc). Resting systolic blood pressure (mean of second and third measurements) was assessed by certified technicians at three 1-min intervals using random zero sphygmomanometers (WA Baum Co, Copaigue, NY). Fasting plasma total and HDL cholesterol were measured using enzymatic assays described elsewhere (CV ≤ 2% for total cholesterol and ≤3% for HDL cholesterol) (Warnick, 1986). Participants were considered to have diabetes if they reported having diabetes or fasting glucose concentrations ≥126 mg/dL. Trained interviewers obtained information on antihypertensive and cholesterol-lowering medication use. Smoking was assessed via self-report, as current smoker (yes/no) in regression analyses, and current daily number of cigarettes smoked in mediation analyses. Body mass index was derived from weight and height (kg/m^2), measured by certified technicians. Depressive symptomatology was measured using the 20-item CES-D (Center For Epidemiological Studies Depression) questionnaire. Anger-out expression was measured by the anger-out subscale of the Spielberger Trait Anger Expression Inventory (Cronbach's $\alpha = 0.77$), where higher scores represent greater anger-out expression (Spielberger, Syndeman, Owen, & Marsh, 1999). Social support was assessed by an 8-item summative scale adapted from Schuster et al. (Schuster, Kessler, & Aseltine, 1990) that includes both supportive and negative social interactions (Cronbach's $\alpha = 0.80$), where elevated scores correspond with lower social support.

Statistical analyses

Descriptive statistics were generated for dependent variables and covariates in males and females, according to quartiles of risky family score. Multivariable-adjusted linear regression analyses evaluated associations of risky family score with mean carotid IMT. The risky family score was entered as a continuous variable with a range of 0–21 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 of 0–21 in order to allow comparability with studies that used z-scores (Lehman et al., 2005, 2009; Taylor et al., 2006). In addition, secondary multivariable-adjusted linear regression analyses were performed to assess associations of each of the seven individual risky family questions (score range 0–3) with mean carotid IMT, which allowed the evaluation of whether there are specific components of the risky family questionnaire (e.g. felt loved, supported and cared for *vs.* verbally abused) that may be particularly important for cardiovascular risk.

We assessed whether several CHD risk factors/markers were potential mediators in the association between family psychosocial environment and carotid IMT using a multiple mediation model. This model simultaneously estimates associations between each mediator and the family psychosocial environment exposure, along with difference in the outcome (mean carotid IMT) associated with the mediator in the fully adjusted model, and calculates indirect effects using the product of coefficients method described elsewhere (Hayes, 2009; Preacher & Hayes, 2008). The indirect effect is the reduction in association of exposure with outcome due to the tested mediator. Indirect effects attributed to each individual mediator are summed to estimate the total indirect effect. Examining individual indirect effects provides evidence of whether childhood family psychosocial environment may exert its effects uniquely through any of the mediators examined in this study. Confidence intervals were estimated by use of the bias-corrected bootstrapping procedure with 5000 resamples (Hayes, 2009; Preacher & Hayes, 2008). This approach does not allow for evaluation of dichotomous mediators, consequently mediation analyses did not evaluate potential roles of antihypertensive medication use (yes/no) or cholesterol-lowering medication use (yes/no). Secondary analyses adjusting for these two variables provided some evidence on potential contributions as mediators. Analyses were conducted using SAS version 9.1.3 (SAS Institute, Cary, NC).

Results

Participants with missing data ($n = 2453$) were more likely ($p < 0.05$) to be male (47.8 vs. 43.4%), black race/ethnicity (62.2 vs. 41.8%), smoke (27.0 vs. 17.0%), have lower HDL cholesterol (53.2 vs. 54.7 mg/dL), higher systolic blood pressure (117.5 vs. 115.1 mmHg), higher diastolic blood pressure (73.4 vs. 71.9 mmHg), take antihypertensive medications (21.9% vs. 16.0%), have diabetes (7.4% vs. 4.9%), and less likely to have a parent with >16 years education (13.5 vs. 14.1%), less likely to complete >16 years of education themselves (13.3 vs. 25.5%), had lower annual family income ($55,700 vs. $72,800), higher CES-D score (10.5 vs. 8.7), higher anger score (6.0 vs. 5.7), higher lack of social support (6.9 vs. 6.0), and higher mean IMT (0.73 mm vs. 0.71 mm). Included and excluded participants were similar ($p > 0.05$) with regard to age, BMI, total cholesterol, and taking cholesterol-lowering medications.

The age range of participants was 37–52 years (mean 45 years). Bivariate analyses demonstrated that risky family score was associated with smoking, parental education, own family income, depressive symptomatology, anger expression and social support in all racial/ethnic and gender groups. Other covariates were associated with risky family score, depending on race/ethnicity and gender, shown in Tables 1 and 2.

Using multivariable-adjusted regression analyses adjusting for age, risky family score was positively associated with mean IMT in white males and white females (Table 3). For example, a 1-unit increase in risky family score (range 0–21) was associated with 0.0020 (95% CI: 0.0002,0.0038) and 0.0036 mm (95% CI: 0.0006,0.0066 mm) increase in mean carotid IMT in white females and white males, respectively (Table 3). Further adjustment for childhood socioeconomic position induced a moderate reduction in effect size, specifically a 25% (i.e. 1–(0.0015/0.0020)) and 15% (i.e. 1–(0.0030/0.0036)) effect size reduction in white females and white males, respectively (Table 3). Effects of adjustment for own education, psychosocial variables and CHD risk markers are shown in Table 3. There were no associations between risky family score and carotid IMT in black males or black females. Similar findings were shown using maximum carotid IMT instead of mean carotid IMT (Appendix Table B), and using Z-scores instead of the ordinal score for the risky family independent variable (Appendix Table C). Formal statistical tests for interaction between risky family score and sex, and between risky family score and race/ethnicity, demonstrated borderline evidence of interactions for both sex ($p = 0.12$) and race/ethnicity ($p = 0.14$) with risky family score for the association with mean IMT.

In an effort to evaluate potential mechanisms by which childhood psychosocial family environment may influence IMT, multiple mediation tests demonstrated that in females, smoking had indirect effects, suggesting it may be a possible mediator between risky family score and carotid IMT (Table 4). There was marginal evidence of indirect effects for HDL cholesterol, education and anger-out expression in white females. In white males, smoking, education and depressive symptomatology demonstrated significant indirect effects consistent with being potential mediators (Table 4).

In order to evaluate which of the 7 components of the risky family questionnaire may be particularly strongly associated with CHD risk, analyses evaluated associations of each questionnaire item with mean carotid IMT (Appendix Table A). Parental monitoring (responses to question "Did your family know what you were up to?") was significantly associated with mean IMT in white males after adjusting for age and childhood SEP (Appendix Table A). Parental love, support and care (responses to question "How often did a parent or other adult in the household make you feel that you were loved, supported and cared for?") was significantly associated with mean IMT in white males and females after adjusting for age and childhood SEP (Appendix Table A). Finally, parental aggression (responses to question "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?") was significantly associated with mean IMT in white males after adjusting for age (Appendix Table A). Overall, this demonstrated that it was not only potentially abusive factors (e.g. how often sworn at or insulted) that were associated with carotid IMT, but also family situations related to parental monitoring/attentiveness. There were no associations of any of the individual risky family score components with mean carotid IMT in black males or black females (data not shown).

Table 1
Females: descriptive statistics of outcome (mean carotid intima media thickness) and covariates according to quartile of risky family score, stratified by race/ethnicity. Point estimates are means or percentages, with 95% confidence intervals in parentheses.

	White Race/Ethnicity				Black Race/Ethnicity			
	Quartile of risky family score				Quartile of risky family score			
	1	2	3	4	1	2	3	4
	Range: 0–1 ($n = 258$)	Range: 2–3 ($n = 158$)	Range: 4–6 ($n = 163$)	Range: 7–21 ($n = 247$)	Range: 0–1 ($n = 176$)	Range: 2–3 ($n = 153$)	Range: 4–6 ($n = 169$)	Range: 7–21 ($n = 180$)
Age, y	45.5[a] (45.1,45.9)	45.7 (45.2,46.3)	45.6 (45.0,46.1)	46.2[a] (45.8,46.6)	43.9 (43.4,44.5)	44.9 (44.3,45.5)	44.8 (44.2,45.3)	44.3 (43.7,44.9)
Smoker, %	8.9[a] (5.7,13.1)	7.0 (3.5,12.1)	10.4 (6.1,16.2)	20.7[a] (15.8,26.2)	11.9[a] (7.5,17.7)	19.0 (13.1,26.1)	23.7 (17.5,30.8)	24.4[a] (18.4,31.4)
HDL cholesterol, mg/dL	62.6[a] (60.8,64.4)	64.1 (61.2,67.0)	60.9 (58.4,63.4)	60.3[a] (58.1,62.5)	57.1 (54.9,59.2)	58.7 (56.3,61.1)	58.3 (55.7,61.0)	57.8 (55.1,60.5)
Total cholesterol, mg/dL	183.4[a] (179.7,187.0)	191.0 (185.9,196.0)	189.3 (184.3,194.4)	187.9[a] (184.0,191.8)	181.8 (176.8,186.9)	188.1 (182.5,193.8)	185.3 (180.2,190.4)	183.4 (178.5,188.3)
Body mass index, kg/m^2	26.4[a] (25.6,27.1)	27.7 (26.5,28.9)	27.0 (25.9,28.1)	27.7[a] (26.9,28.6)	33.0 (31.9,34.2)	32.3 (31.0,33.7)	31.9 (30.6,33.1)	32.6 (31.5,33.8)
Systolic blood pressure, mmHg	108.4 (107.2,109.8)	109.2 (107.2,111.2)	108.4 (106.4,110.3)	108.9 (107.5,110.4)	118.5 (115.9,121.0)	117.7 (114.9,120.6)	117.4 (114.9,119.8)	116.0 (113.8,118.3)
Diabetes, %	2.7 (1.1,5.5)	0.0 –	3.1 (1.0,7.0)	1.2 (0.3,3.5)	6.3[a] (3.2,10.9)	6.5 (3.2,11.7)	4.7 (2.1,9.1)	13.3[a] (8.7,19.2)
Taking anti-hypertensive medication, %	7.4 (4.5,11.3)	5.1 (2.2,9.7)	9.8 (5.7,15.5)	7.7 (4.7,11.8)	27.8 (21.4,35.1)	27.5 (20.6,35.2)	23.7 (17.5,30.8)	29.4 (22.9,36.7)
Taking cholesterol-lowering medication, %	7.0[a] (4.2,10.8)	2.5 (0.7,6.4)	4.9 (2.1,9.4)	3.7[a] (1.7,6.8)	8.0 (4.4,13.0)	6.6 (3.2,11.8)	5.3 (2.5,9.9)	7.8 (4.3,12.7)
Parental education, y	15.3[a] (15.0,15.7)	15.0 (14.5,15.5)	15.3 (14.8,15.9)	14.1[a] (13.7,14.5)	13.2[a] (12.8,13.6)	13.1 (12.6,13.5)	12.4 (11.9,12.7)	12.3[a] (11.9,12.7)
Own education, %>16 y	39.9[a] (33.9,46.2)	42.4 (34.6,50.5)	37.4 (30.0,45.3)	27.5[a] (22.1,33.6)	14.8 (9.9,20.9)	15.0 (9.8,21.7)	10.7 (6.4,16.3)	12.8 (8.3,18.6)
CES-D score	5.9[a] (5.2,6.7)	7.6 (6.5,8.6)	8.2 (7.2,9.2)	9.8[a] (8.7,10.9)	7.9[a] (6.8,8.9)	9.0 (7.9,10.2)	11.4 (10.0,12.8)	15.1[a] (13.6,16.6)
Anger score	5.5[a] (5.1,5.8)	6.4 (5.9,6.9)	6.4 (5.9,6.9)	6.4[a] (6.0,6.8)	5.0[a] (4.5,5.5)	5.4 (4.8,5.9)	6.1 (5.6,6.6)	6.5[a] (5.9,7.0)
Social support score	4.0[a] (3.7,4.3)	5.3 (4.8,5.7)	5.7 (5.2,6.2)	7.5[a] (7.0,8.0)	4.2[a] (3.8,4.7)	5.8 (5.3,6.4)	7.1 (6.6,7.7)	9.6[a] (8.9,10.3)
Intima media thickness	0.65[a] (0.64,0.67)	0.66 (0.64,0.68)	0.66 (0.64,0.69)	0.68[a] (0.66,0.70)	0.71 (0.69,0.73)	0.71 (0.68,0.73)	0.70 (0.68,0.72)	0.70 (0.68,0.72)

CES-D, Center for Epidemiologic Studies Depression Scale; HDL, high density lipoprotein; y, years.
[a] Represents point estimates for highest *vs.* lowest risky family score quartile where 95% confidence intervals do not overlap.

Table 2
Male descriptive statistics of outcome (mean carotid intima media thickness) and covariates according to quartile of risky family score, stratified by race/ethnicity. Point estimates are means or percentages, with 95% confidence intervals in parentheses.

	White Race/Ethnicity				Black Race/Ethnicity			
	Quartile of risky family score				Quartile of risky family score			
	1	2	3	4	1	2	3	4
	Range: 0–1 (n = 203)	Range: 2–3 (n = 181)	Range: 4–6 (n = 167)	Range: 7–21 (n = 170)	Range: 0–1 (n = 98)	Range: 2–3 (n = 108)	Range: 4–6 (n = 114)	Range: 7–21 (n = 114)
Age, y	45.7 (45.3,46.2)	45.7 (45.2,46.2)	45.9 (45.5,46.4)	45.9 (45.4,46.4)	44.4 (43.7,45.1)	44.8 (44.1,45.6)	44.7 (44.0,45.4)	44.9 (44.3,45.6)
Smoker, %	8.9[a] (5.3,13.6)	13.8 (9.1,19.7)	13.8 (8.9,20.0)	21.8[a] (15.8,28.7)	21.4[a] (13.8,30.9)	20.4 (13.2,29.2)	29.0 (20.8,38.2)	32.5[a] (24.0,41.9)
HDL cholesterol, mg/dL	45.6 (43.9,47.3)	45.6 (43.7,47.5)	48.0 (46.0,50.0)	45.6 (43.2,48.0)	49.2 (46.5,51.9)	48.6 (45.9,51.3)	49.6 (46.0,53.3)	50.1 (47.3,52.9)
Total cholesterol, mg/dL	186.6 (181.4,191.7)	186.1 (180.8,191.5)	191.1 (185.8,196.4)	187.7 (181.8,193.7)	181.9 (173.9,189.9)	183.8 (175.9,191.7)	184.6 (177.6,191.6)	183.4 (177.8,189.0)
Body mass index, kg/m^2	28.7 (27.5,29.9)	28.6 (27.9,29.3)	27.9 (27.2,28.6)	28.6 (27.9,29.4)	30.5 (29.1,32.0)	30.0 (28.7,31.3)	30.6 (29.2,31.9)	29.6 (28.5,30.8)
Systolic blood pressure, mmHg	116.3 (114.7,117.9)	116.9 (115.1,118.7)	116.5 (114.7,118.4)	115.5 (113.8,117.2)	119.4 (117.3,121.6)	123.6 (120.8,126.3)	122.6 (119.6,125.3)	121.5 (119.3,123.7)
Diabetes, %	3.5 (1.4,7.1)	5.0 (2.3,9.2)	4.2 (1.7,8.5)	3.5 (1.3,7.5)	7.1 (2.9,14.2)	7.4 (3.3,14.1)	8.8 (4.3,15.5)	7.0 (3.1,13.4)
Taking anti-hypertensive medication, %	9.9 (6.1,14.8)	17.2 (12.0,23.6)	15.0 (9.9,21.3)	11.2 (6.9,16.9)	19.4 (12.1,28.6)	19.4 (12.5,28.2)	21.2 (14.1,29.9)	17.5 (11.1,25.8)
Taking cholesterol-lowering medication, %	13.3 (9.0,18.8)	19.4 (13.9,26.0)	13.2 (8.5,19.4)	11.8 (7.4,17.7)	9.2 (4.3,16.7)	11.1 (5.9,18.6)	5.3 (2.0,11.1)	7.1 (3.1,13.5)
Parental education, y	15.6[a] (15.2,16.0)	15.2 (14.8,15.7)	14.7 (14.2,15.1)	14.7[a] (14.2,15.2)	13.4[a] (12.8,13.9)	13.0 (12.5,13.4)	13.1 (12.7,13.6)	12.6[a] (12.1,13.1)
Own education, %>16 y	43.8[a] (36.9,51.0)	34.3 (27.4,41.7)	32.3 (25.3,40.0)	28.8[a] (22.2,36.3)	10.2 (5.0,18.0)	3.7 (1.0,9.2)	10.0 (4.9,16.6)	9.7 (4.9,16.6)
CES-D score	5.5[a] (4.8,6.2)	7.1 (6.3,7.9)	7.9 (7.0,8.9)	9.6[a] (8.4,10.9)	5.7[a] (4.8,6.6)	8.0 (6.9,9.1)	9.3 (8.0,10.5)	12.0[a] (10.4,13.6)
Anger score	5.0[a] (4.6,5.4)	5.7 (5.2,6.1)	5.8 (5.3,6.2)	6.4[a] (5.8,6.9)	4.4[a] (3.8,5.0)	5.0 (4.5,5.5)	5.3 (4.7,5.8)	5.5[a] (4.9,6.1)
Social support score	3.9[a] (3.5,4.3)	5.2 (4.7,5.6)	5.9 (5.4,6.3)	8.1[a] (7.4,8.7)	4.2[a] (3.6,4.9)	5.3 (4.8,5.9)	6.4 (5.7,7.0)	8.7[a] (7.9,9.6)
Intima media thickness	0.72[a] (0.70,0.74)	0.74 (0.72,0.76)	0.75 (0.73,0.78)	0.76[a] (0.73,0.78)	0.75 (0.73,0.78)	0.77 (0.74,0.81)	0.76 (0.72,0.79)	0.76 (0.74,0.79)

CES-D, Center for Epidemiologic Studies Depression Scale; HDL, high density lipoprotein; y, years.
[a] Represents point estimates for highest *vs.* lowest risky family score quartile where 95% confidence intervals do not overlap.

Discussion

This study demonstrated that in white males and females, increasingly adverse childhood family psychosocial environment was positively associated with mean carotid IMT after adjusting for age. Further adjustment for childhood SEP caused a moderate reduction in effect sizes. Mediation analyses provided evidence that smoking in white males and females, as well as education and depressive symptomatology in white males, may be mechanisms that explain how the childhood family psychosocial environment could influence carotid IMT. No associations between family psychosocial environment and carotid IMT were found in black participants.

Prior literature

To our knowledge, this is the first study to evaluate associations of the childhood family psychosocial environment with subclinical cardiovascular measures such as carotid IMT. Most (Dong et al., 2004; Fuller-Thomson, Brennenstuhl, & Frank, 2010; Russek & Schwartz, 1997) but not all (Korkeila et al., 2010) studies that evaluated associations of the childhood family psychosocial environment with prevalent/incident CHD used self-reported measures of CHD. Carotid IMT provides a more direct assessment than self-reported CHD, that has the benefit of being free from biases due to self-report, although carotid IMT has the limitation of being an imperfect predictor of CHD events (Lorenz et al., 2007). With regard to other studies that evaluated associations with risk for CHD (although these other studies did not evaluate carotid IMT), this present study's findings in white participants are in general agreement with other studies that showed adverse childhood psychosocial family environment was related to higher risk for CHD after adjusting for age (Almeida et al., 2010; Batten et al., 2004; Dong et al., 2004; Fuller-Thomson et al., 2010; Korkeila et al., 2010; Loucks et al., 2011; Russek & Schwartz, 1997). With regard to our findings of associations in white but not black participants, most studies to date on the relation between the childhood family psychosocial environment with CHD risk have either performed analyses only in white participants, or adjusted for race/ethnicity (Almeida et al., 2010; Batten et al., 2004; Dong et al., 2004; Fuller-Thomson et al., 2010; Korkeila et al., 2010; Loucks et al., 2011; Russek & Schwartz, 1997). Consequently, little is known about associations in black participants. In the CARDIA study, Lehman et al. (Lehman et al., 2005, 2009) and Taylor et al. (Taylor et al., 2006) evaluated race/ethnicity- and sex-specific associations of the pathways between childhood SEP, risky family score and a number of CHD risk markers using structural equation models, with some evidence of better model fit for white than black participants (Lehman et al., 2005, 2009; Taylor et al., 2006). The reasons why effects were shown in our study for white but not black participants are uncertain. Loss to follow-up was higher in the CARDIA study for black compared with white participants, and it is possible this influenced findings through bias. There is

Table 3
Associations of risky family score with mean carotid intima media thickness (IMT) measured at 20 sites of the bifurcation, internal and common carotid artery. Point estimates represent regression coefficients (95% confidence intervals); units are mm increase in intima media thickness per one unit increase in risky family score (range of risky family score 0–21).

	Model adjustment					
	Age	Age, childhood SEP[a]	Age, adulthood SEP[a]	Age, psychosocial[a]	Age, CHD risk markers[a]	All covariates
White Females	0.0020[b] (0.0002,0.0038)	0.0015 (−0.0003,0.0033)	0.0017 (−0.0001,0.0035)	0.0015 (−0.0005,0.0034)	0.0013 (−0.0004,0.0030)	0.0011 (−0.0007,0.0049)
White Males	0.0036[b] (0.0006,0.0066)	0.0030 (0.0000,0.0060)	0.0020 (−0.0010,0.0050)	0.0032 (−0.0001,0.0066)	0.0030[b] (0.0001,0.0059)	0.0017 (−0.0016,0.0049)
Black Females	−0.0004 (−0.0027,0.0019)	−0.0004 (−0.0027,0.0020)	−0.0004 (−0.0027,0.0019)	−0.0009 (−0.0036,0.0018)	−0.0008 (−0.0030,0.0015)	−0.0003 (−0.0030,0.0024)
Black Males	0.0000 (−0.0038,0.0037)	−0.0003 (−0.0040,0.0035)	−0.0004 (−0.0041,0.0034)	−0.0005 (−0.0047,0.0037)	0.0003 (−0.0034,0.0040)	−0.0009 (−0.0050,0.0032)

[a] Childhood SEP was assessed using father's and mother's education. Adulthood SEP was assessed using own education. Psychosocial functioning variables include depressive symptomatology, anger expression, positive social contacts and negative social contacts. CHD risk markers include smoking, body mass index, systolic blood pressure, total cholesterol, HDL cholesterol, diabetes, cholesterol-lowering medications and antihypertensive medications.
[b] Represents point estimates for which 95% confidence intervals do not encompass null (0.0000 mm).

literature on racial/ethnic differences in associations of non-abusive corporeal punishment with externalizing behaviors and conduct problems in youth, and concern has been expressed that much of the literature and recommendations are catered to white, middle-class Americans (Lansford, Deater-Deckard, Dodge, Bates, & Pettit, 2004; Pardini, Fite, & Burke, 2008). For example, in a prospective study of 585 children from age 5 through 13 years, greater use of physical discipline was associated with higher levels of subsequent externalizing behaviors in European American adolescents, but lower levels of externalizing behaviors in African American adolescents (Lansford et al., 2004). This study suggested that different ecological niches may affect the way in which parents use physical discipline, the meaning that children attached to experiences of physical discipline, and its effects on the adjustment of offspring. However racial/ethnic-specific findings are not consistent in the literature, and more research using prospective studies and representative racial/ethnic groups will improve knowledge in this area (Pardini et al., 2008). All studies to date, to the best of our knowledge, that have evaluated contributions of the childhood family psychosocial environment to CHD risk in black participants have been in the CARDIA study (Lehman et al., 2005, 2009; Taylor et al., 2006); replication using race/ethnicity-specific analyses in other study populations will help further understand associations between the childhood family psychosocial environment and carotid IMT in African Americans as well as other racial/ethnic groups.

Mechanisms

How could a childhood family environment composed of cold, unaffectionate interactions, conflict, aggression, neglect and/or low nurturance causally influence thickening of the carotid artery, atherosclerosis and eventual CHD? Studies have demonstrated that exposure to emotional neglect, abuse, and cold, distant parent–child relationships increases the risk of depression and anxiety (Francis, Champagne, Liu, & Meaney, 1999); depression itself is a risk marker for CHD (Rugulies, 2002). Mediation analyses in this study suggested that depressive symptomatology in white males, and possibly anger-out expression in white females may be explanatory mechanisms. Furthermore, certain behaviors (e.g. smoking, binge drinking, overeating) can be used as mental health coping strategies. Adverse early family psychosocial environments are typically associated with elevated smoking (Dong et al., 2004; Loucks et al., 2011), binge drinking (Kauhanen, Leino, Lakka, Lynch, & Kauhanen, 2011) and obesity (Midei & Matthews, 2011); smoking and obesity are established CHD risk factors, while early evidence suggests binge drinking may be a CHD risk marker (Bagnardi, Zatonski, Scotti, La Vecchia, & Corrao, 2008). Parental monitoring and family connectedness can be important factors in limiting adolescent smoking, alcohol consumption and overeating (Cromley, Neumark-Sztainer, Story, & Boutelle, 2010; Mahabee-Gittens, Xiao, Gordon, & Khoury, 2012; Simons-Morton, Haynie, Crump, Eitel, & Saylor, 2001). For example, with regard to cigarette

Table 4
Mediation tests evaluating indirect effects of risky family score on mean IMT, stratified by sex and race/ethnicity.

Mediators	White females		White males		Black females		Black males	
	IE	95% CI	IE	95% CI	IE	95% CI	IE	95% CI
Body mass index	0.0002	(−0.0003,0.0007)	−0.0000	(−0.0004,0.0007)	0.0000	(−0.0004,0.0003)	−0.0005	(−0.0013,0.0000)
Systolic blood pressure	−0.0002	(−0.0006,0.0002)	−0.0001	(−0.0007,0.0003)	−0.0001	(−0.0005,0.0001)	−0.0001	(−0.0005,0.0002)
Smoking	0.0003[a]	(0.0001,0.0007)	0.0005[a]	(0.0001,0.0013)	0.0004[a]	(0.0001,0.0090)	−0.0001	(−0.0006,0.0001)
Total cholesterol	0.0001	(−0.0002,0.0004)	0.0001	(−0.0002,0.0006)	−0.0000	(−0.0003,0.0002)	0.0002	(−0.0002,0.0001)
HDL cholesterol	0.0003	(0.0000,0.0006)	−0.0001	(−0.0006,0.0002)	−0.0000	(−0.0003,0.0001)	0.0001	(−0.0007,0.0002)
Fasting glucose	0.0001	(−0.0002,0.0005)	0.0001	(−0.0008,0.0009)	0.0001	(−0.0001,0.0004)	−0.0001	(−0.0006,0.0003)
Own education	0.0001	(0.0000,0.0004)	0.0013[a]	(0.0006,0.0021)	0.0000	(−0.0002,0.0004)	0.0002	(−0.0001,0.0010)
CES-D score	0.0001	(−0.0002,0.0006)	0.0010[a]	(0.0003,0.0020)	−0.0006	(−0.0015,0.0003)	0.0003	(−0.0008,0.0014)
Anger-out expression score	0.0002	(0.0000,0.0005)	0.0000	(−0.0005,0.0006)	0.0003	(−0.0001,0.0009)	0.0001	(−0.0002,0.0007)
Social support score	0.0005	(−0.0002,0.0011)	0.0003	(−0.0012,0.0018)	0.0008	(−0.0005,0.0023)	0.0006	(−0.0011,0.0024)
Total indirect effect	0.0006[a]	(0.0001,0.0012)	0.0021[a]	(0.0010,0.0036)	−0.0004	(−0.0014,0.0006)	0.0002	(−0.0010,0.0015)

Models include all potential mediators, adjusted for age and childhood socioeconomic position.
CES-D, Center for Epidemiologic Studies Depression Scale.
Indirect effect (IE) represents difference in mm mean IMT per unit increase of risky family score due to the addition of mediator into the model.
[a] Represents point estimates for which 95% confidence intervals do not encompass null (0.0000 mm).

consumption, adolescence is recognized as a sensitive period in the life course, as a large proportion of smokers start during this time (American Lung Association Epidemiology and Statistics Unit, 2011). The addictive properties of cigarettes make it difficult to quit for the remainder of the life course, leading to increased risk for CHD. Parental monitoring and caring, positive parent–child relationships can reduce the likelihood that youth initiate smoking, thereby potentially limiting this important CHD risk factor for the remainder of the life course (Mahabee-Gittens et al., 2012). Mediation analyses in this study show fairly strong evidence for smoking as a potential mechanism between the risky family score and carotid IMT in both white males and females, suggesting this may be an important mechanism. Furthermore, adverse childhood family environments including childhood neglect are associated with poor educational outcomes (Chapple & Vaske, 2010). Education itself is a risk marker for CHD and may causally influence CHD potentially through pathways such as health literacy and income-related access to health care, healthy environments and foods, amongst others (Manrique-Garcia, Sidorchuk, Hallqvist, & Moradi, 2011). Mediation analyses showed an indirect effect of education in white males, suggesting it may be an additional mechanism.

It is important to note that the childhood psychosocial environment, particularly parenting quality, is dependent on the larger context in which the family is living. Low SEP of families, including longitudinal descent of families into low SEP, is associated with elevated risk for physical mistreatment or abuse, and appears to move parenting to become more harsh, punitive, irritable, inconsistent and coercive (McLoyd, 1998). In animal models, similar environmental conditions such as scarcity of food, social instability and low dominance have also been shown to worsen quality of maternal care (Francis et al., 1999). Adjusting for childhood SEP in the current analyses moderately reduced effect sizes by 15–25%, suggesting that the early life socioeconomic family context may be an explanatory mechanism that could lead to worsened parenting, thereby leading to CHD risks such as depression, smoking, obesity, and low education, finally leading to increased carotid IMT. After adjusting for childhood SEP, the 95% confidence intervals marginally encompassed the referent category. Statistical power is very much affected by sample size and measurement error, therefore independent associations of risky family environment with carotid IMT may still be clinically meaningful, despite limitations of sample size and ability to accurately measure childhood psychosocial environment in this study. Structural equation modeling approaches have found associations consistent with the pathways by which childhood SEP is related to risky family score, which in turn is related to CHD risk markers such as metabolic syndrome (Lehman et al., 2005), C-reactive protein (Taylor et al., 2006) and blood pressure (Lehman et al., 2009). Overall, evidence suggests that the early family socioeconomic environment has an impact on the quality of parental care, which could then potentially influence CHD risk.

Strengths and limitations

Limitations of the study include that 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 a confirmatory factor analysis, and found no overlap of the risky family variable with depressive symptomatology, social support or anger-out expression. Other literature has further evaluated validity and reliability of retrospective reporting for constructs including childhood SES (Krieger et al., 1998), parental support and affection (Brewin et al., 1993; Parker, 1989), and childhood abuse (Dill et al., 1991). Overall, the possibility remains for misclassification by retrospective reporting bias, or effects of mood/affect; however the validation techniques demonstrated that the measure is reasonably robust for independence from reporting on depression, social support and anger-out expression.

With regard to strengths, this study used a fairly large sample size ($n = 2659$), and was diverse in race/ethnicity (black and white), sex, and socioeconomic position. It provided both race/ethnicity- and sex-specific analyses, which are an important gap in the literature. Furthermore, there is little 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 a subclinical measure of CHD (carotid IMT). The biological outcome measures and covariates were measured utilizing rigorous quality control/quality assurance protocols.

Clinical and population health implications

If the early family psychosocial environment does indeed impact risk for CHD, there is substantial literature on interventions to help prevent or mitigate the effects of these environments on children, described in more detail elsewhere (Garner & Shonkoff, 2012; Mercy & Saul, 2009; Repetti et al., 2002). A number of specific programs such as the Nurse-Family Partnership, Perry Preschool, and Triple P programs have been evaluated for providing a nurturing environment to children and their families to improve developmental and health outcomes, described in further detail elsewhere (Garner & Shonkoff, 2012; Mercy & Saul, 2009; Repetti et al., 2002). Finally, it is important to consider the social context in which the childhood family psychosocial environment manifests. Parents living in poverty, with few employment opportunities, low education and inadequate support systems are more likely to express more adverse parenting styles, such as neglect, abuse and lower parental monitoring (Repetti et al., 2002). Efforts to address some these potential upstream fundamental determinants of health may help to break the intergenerational cycles that tend to link early life adversity with later life disease.

With regard to the clinical significance of findings, according to our results and those of the aforementioned meta-analysis (Lorenz et al., 2007), a plausible 10-unit difference in risk family score (e.g. for a child in a low *vs.* higher risk family; risky family score range 0–21) represents an increased risk of 3.0–5.4% for myocardial infarction and 3.6–6.5% increased risk for stroke. These represent significant but fairly small increased risk for cardiovascular events, which makes some sense given the multitude of risk factors for cardiovascular disease (U.S. Department of Health and Human Services, 2002).

Conclusions

This study demonstrated that in white males and females, increasingly adverse childhood family psychosocial environment was positively associated with mean carotid IMT after adjusting for age. Further adjustment for childhood SEP induced a moderate reduction in effect sizes. Mediation analyses suggested that smoking in white males and females, in addition to education and depressive symptomatology in white males, may be mechanisms through which the childhood family psychosocial environment could influence carotid IMT. No associations were found between family psychosocial environment and carotid IMT in black participants. These findings suggest a possible role of the childhood family psychosocial environment influencing carotid IMT in white participants.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2013.12.015.

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