Relation of Childhood Socioeconomic Status and Family Environment to Adult Metabolic Functioning in the CARDIA Study

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Objective: Low SES and a conflict-ridden, neglectful, or harsh family environment in childhood have been linked to a high rate of physical health disorders in adulthood. The objective of the present investigation was to evaluate a model of the pathways that may help to explain these links and to relate them to metabolic functioning (MF) in the Coronary Artery Risk Development In Young Adults (CARDIA) dataset. **Methods:** Participants ($n = 3225$) in the year 15 assessment of CARDIA, age 33 to 45 years, completed measures of childhood socioeconomic status (SES), risky early family environment (RF), adult psychosocial functioning (PsyF, a latent factor measured by depression, hostility, positive and negative social contacts), and adult SES. Indicators of the latent factor MF were assessed, specifically, cholesterol, insulin, glucose, triglycerides, and waist circumference. **Results:** The overall prevalence of metabolic syndrome was 9.7%. Structural equation modeling indicated that childhood SES and RF are associated with MF via their association with PsyF (standardized path coefficients: childhood SES to RF −0.13, RF to PsyF 0.44, PsyF to MF 0.09, all $p < .05$), but also directly (coefficient from childhood SES to MF −0.12, $p < .05$), with good overall model fit. When this model was tested separately for race-sex subgroups, it fit best for white women, fit well for African-American women and white men, but did not fit well for African-American men. **Conclusions:** These results indicate that childhood SES and early family environment contribute to metabolic functioning through pathways of depression, hostility, and poor quality of social contacts. **Key words:** stress, family, health, SES, comorbidities, HPA.

SES = socioeconomic status; **RF** = risky family environment; **MF** = metabolic functioning; **PsyF** = psychosocial functioning; **CHD** = coronary heart disease; **HPA** = hypothalamic pituitary axis; **LDL** = low-density lipoprotein; **HDL** = high-density lipoprotein; **CARDIA** = Coronary Artery Risk Development In Young Adults; **RMSEA** = root mean-square error of approximation; **NFI** = normed fit index; **CFI** = confirmatory fit index; **SEM** = structural equation modeling.

INTRODUCTION

A substantial body of research indicates that low socioeconomic status (SES) is associated with a broad array of health risks (1), including an elevated risk for metabolic dysregulation (2). One possible pathway may be via familial stress in childhood. Low SES is associated with chronic social and financial stress (3) and can exacerbate family tensions, strain, and conflict (4–6). Economic adversity in the family is associated with a poor or deteriorating quality of parenting, including higher levels of family conflict, a harsh restrictive parenting style, and chaotic or neglectful parenting (4–6). Families characterized by overt conflict and aggression and/or by a cold and unaffectionate interaction style, in turn, have offspring with an increased risk for a wide variety of health problems in adulthood (7).

For example, in a study of 13,494 adults, Felitti and colleagues (8) found a strong graded relationship between breadth of exposure to abuse or household dysfunction during childhood and risk for adult health disorders, including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. Similarly, Walker and colleagues (9) found a history of physical or sexual abuse to be associated with a broad array of physical symptoms and medical diagnoses in adulthood. Prospective studies have similarly found that conflict in the family during upbringing is associated with an increased risk, years later, of diagnosable medical conditions (7,10). Accumulating evidence indicates that these adverse clinical outcomes can result from more mild, subabusive family environments characterized as harsh or conflict-ridden as well (7). Low SES can contribute to these dynamics.

Repetti et al. (7) proposed that these "risky" family (RF) environments lead to compromised emotional, social, and biological stress responses that can, in turn, lead to adverse alterations in biological systems, culminating in increased rates of morbidity and mortality from chronic disorders. One such indicator of potential health problems, metabolic syndrome (11), involves deficits in metabolic functioning (MF) (12,13). MF is typically defined by fasting glucose, cholesterol, triglycerides, blood pressure, and abdominal obesity. Metabolic syndrome is defined as exceeding recommended standards on three or more of these indicators (11) and is prognostic for heart disease (14), diabetes, inflammatory diseases (15,16), and all-cause mortality (17,18). The prevalence of metabolic syndrome in the United States is approximately 22% (19), making it an important contributor to chronic illness.

The present study examined whether a risky early RF can explain SES-related variability in MF in a large, well-characterized sample of African-American and white men and women. The basis for this prediction is as follows: The chronic stress experienced by children from RFs leads to chronic or recurrent activation of psychological and biological

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stress regulatory systems. As such, through mechanisms outlined below, an RF environment may have cascading effects that result in, among other predisease predictors, increased risk for both clinical and subclinical dysregulation in MF.

Risky Families Model

The RF conceptual model is presented in Figure 1 (7). In the model, low SES is conceptualized as a contributor to chronic familial stress. Childhood SES is temporally prior, has been tied to subsequent variables in the model, and is minimally affected by them in return; specifically, in addition to being related to parenting style, it has been related to the development of chronic negative emotional states, including hostility and depression (20) and to problems in the enlistment and/or use of social support (3).

A risky early family environment is temporally before the subsequent variables in the model and has been independently correlated with each of the subsequent steps as well, including higher levels of hostility, anxiety, and depression (7); lower levels of social support (7); and major physical health disorders (8). These variables predict subsequent psychosocial functioning (PsyF), specifically, depression, hostility, and positive/negative social relationships. Early environments marked by harsh or chaotic parenting are reliably associated with deficits in emotion regulation skills, including both internalizing and externalizing behavior in children (7).

The fact that these emotional problems are seen early in life, coupled with the relation of these emotional states to disease states later in life, makes them potential candidates for mediators of the relation between early family environment and health outcomes. Specifically, hostility has been tied to abdominal obesity (21), to the development of metabolic syndrome among children and adolescents (22), and to an increased risk for coronary heart disease (CHD) and hypertension (23). Clinical depression has been tied to atypical hypothalamic pituitary axis (HPA) diurnal rhythms and responses to stress, to sustained suppressed immunity (24), and to mortality (25). Major depression, depressive symptoms, and history of depression have been identified as predictors of cardiac events (26), an association that may be partly mediated by metabolic syndrome, at least among women (27).

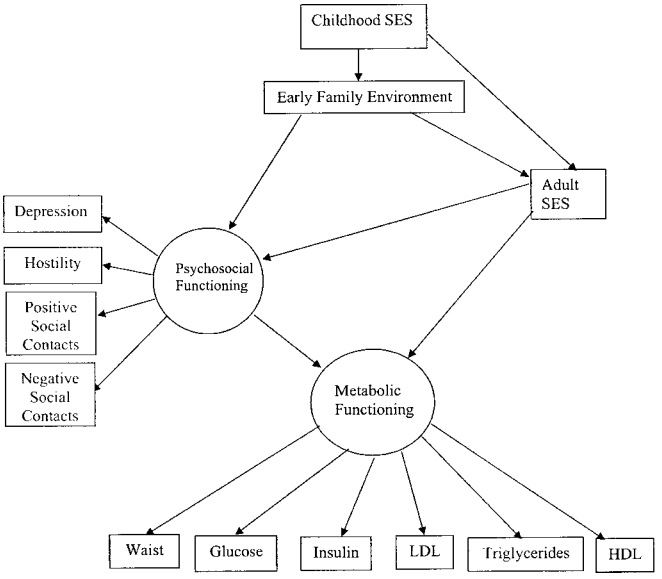

Figure 1. Hypothesized relations of early family environment to metabolic functioning.

Offspring from harsh or chaotic families commonly also show deficits in social skills, compromising their social support systems (7). A lack of social support is a predictor of all-cause mortality in humans (28) and is significantly related to the likelihood of developing health disorders and to delayed recovery from illness (29). A lack of social support has also been tied to problematic glucose metabolism (2) and to a heightened risk of infectious disorders (30), among other adverse outcomes.

As these risks cascade across the lifespan, one would expect to see an increase in risk factors for major chronic diseases. MF, as noted, is responsive to stress and encompasses multiple risk factors prognostic for major chronic diseases. Accordingly, outcome variables examined in the model were MF as indexed by waist circumference, fasting glucose level, fasting insulin level, low-density lipoprotein (LDL), triglycerides, and high-density lipoprotein (HDL) cholesterol. We tested whether the full model proposed in Figure 1 would be substantiated and hypothesized support for each of the proposed pathways by which a risky childhood environment is expected to influence MF.

METHODS

Participants

The current research made use of the year 15 Coronary Artery Risk Development In Young Adults (CARDIA) dataset. CARDIA is an ongoing epidemiologic study of coronary artery risk development in young adults, in which African-American and white participants from a wide range of SES backgrounds took part in 6 assessments over 15 years. The CARDIA study was designed to track predictors of coronary artery disease as young people transition to adulthood. To be included in the study, participants must have identified themselves as either African-American or white, had a permanent address in 1 of 4 urban areas (Birmingham, AL; Chicago, IL; Minneapolis, MN; or Oakland, CA), have been without symptoms of long-term disease or disability, and were not pregnant (see 31,32 for further detail on the methodology of the CARDIA study). The original assessment was in 1985 to 1986 when participants were 18 to 30 years old.

At the time of the most recent assessment (2000–2001), participants ranged in age from 33 to 45, an age at which the beginnings of MF dysregulation can potentially be observed. Of the initial sample of 5115 participants, 3672 were examined at the year 15 assessment. Participation among the living members of the original sample was 73.5% in year 15; attrition analyses suggested that those who remained at year 15 were more likely than the baseline sample to be white, well-educated, older women. Those included in the final sample for this study had complete data on the 13 measures used in the analyses, including a report of childhood SES that was obtained during the 1985 to 1986 baseline assessment. The final sample consisted of 3225 participants: 619 African-American men; 850 African-American women; 812 white men; 944 white women.

Procedure

Participants for the year 15 CARDIA assessment were asked not to exercise on the day of the examination, to fast for 12 hours, and to refrain from smoking for one-half hour before the examination. The examination took approximately 3 hours, during which participants completed informed consents and provided blood and urine samples, blood pressure measure-

ments, and anthropomorphic measurements. They then were given a small snack, participated in interviews on their health behavior and health history, and then completed a set of self-administered questionnaires, including the psychosocial and risky family measures used for this study.

Measures

Early Family Environment

The Risky Families (RF) questionnaire was adapted from an instrument developed by Felitti et al. (8). Respondents answered seven questions about their family environment before age 18 on 4-point scales from 1 (rarely or none of the time) to 4 (most or all of the time), specifically, whether the individual felt loved and cared for, was shown physical affection, was verbally abused, was physically abused, lived with a substance abuser, lived in a well-organized, well-managed household, and whether family members knew what the child was up to. Evidence concerning reliability and validity of the measure appears in (33). Because individual items differed in their variability, each item was z scored before a composite measure was formed; Cronbach's α was 0.77. Higher values represent a riskier family environment; we observed the full range of possible values on this measure.

PsyF

Depression was assessed using the 20-item Center for Epidemiologic Studies Depression Scale (34). Possible values ranged from 0 to 60; we observed values from 0 to 54. Cronbach's α was 0.89. Hostility was measured by the 8-item anger-out (Cronbach's $\alpha = 0.76$) subscale of the Spielberger Trait Anger Expression Inventory (35). Social contacts were assessed by an 8-item scale adapted from Schuster et al. (36) that includes both supportive and unsupportive interactions. Four items composed the positive social contacts subscale ($\alpha = 0.83$), and four items measured negative social contacts ($\alpha = 0.72$). The subscales were correlated ($r = -0.37$, $p < .001$). Response scales for hostility, positive social contacts, and negative social contacts ranged from 1 to 4, and values along that entire continuum were observed in this sample.

Childhood SES was assessed at the baseline 1984 CARDIA assessment by using parental educational attainment as a proxy for childhood SES. Each participant indicated the highest level of education obtained by the participant's parents or primary caregivers. The mean of standardized z scores of the primary male and female caregivers' levels of education ($r = 0.56$, $p < .001$) was used as a measure of childhood SES. Adult SES (year 15) was measured by four items. Participants marked an X on a 9-rung ladder to indicate their standing in life compared with the country and compared with the community (37) and also reported their annual income (in dollars) and number of years of education. Before combining these measures (Cronbach's $\alpha = 0.68$), a standardized z score was computed for each measure.

MF

Waist Girth

Waist girth was measured horizontally at the narrowest point between the iliac crest and the rib cage. The final measure is an average of two assessments, rounded to the nearest 0.5 cm.

Cholesterol

Total cholesterol, HDL cholesterol, and triglyceride assays were assessed enzymatically using the Abbott Biochromatic Analyzer. Cholesterol was determined by a Trinder-type method, and triglycerides by an ultraviolet method (38). HDL was separated from plasma by chemical precipitation with dextran sulfate-magnesium (mw 50,000) (39,40), and the resulting supernatant was assayed for cholesterol. LDL was derived using the formula by Friedwald et al. (41).

Fasting Insulin and Glucose

Assays for both glucose and insulin were conducted by Linco Research, Inc (Dr. Ronald Gingerich, P.I.). Insulin levels were determined by radioimmunoassay using an overnight equilibrium incubation format. These insulin values do not include a significant contribution of proinsulin and thus represent "true" insulin levels. Glucose measurements were obtained using a Cobas Mira Plus chemistry analyzer (Roche Diagnostic Systems) using the hexokinase ultraviolet method (42).

We used the National Cholesterol Education Program criteria (9), namely, abdominal obesity greater than 102 cm (men) and 88 cm (women); triglycerides >149; HDL cholesterol <40 mg/dl (men) or <50 mg/dl (women); blood pressure ≥130/85 mm Hg; and fasting glucose >109 mg/dl, for determining metabolic syndrome.

RESULTS

Table 1 presents summary statistics for our overall sample and for each of the four race-sex subgroups. Using National

TABLE 1. Psychosocial, Metabolic, and Demographic Characteristics of Participants

	Entire Sample (n = 3225)	African-American Women (n = 850)	White Women (n = 944)	African-American Men (n = 619)	White Men (n = 812)
Variables in the RF model, mean (SD)					
Mother's education (years)	13.15 (2.23)	12.53 (2.04)	13.51 (2.32)	12.66 (1.98)	13.77 (2.25)
Father's education (years)	13.22 (2.57)	12.24 (2.17)	13.95 (2.68)	12.08 (1.86)	14.10 (2.62)
Early family environment	1.66 (0.58)	1.65 (0.57)	1.70 (0.66)	1.67 (0.53)	1.61 (0.54)
Depression	9.03 (7.9)	11.18 (9.2)	8.15 (7.6)	9.25 (7.1)	7.63 (6.6)
Hostility	1.72 (0.40)	1.74 (0.44)	1.77 (0.39)	1.65 (0.38)	1.71 (0.37)
Positive social contacts	3.51 (0.56)	3.46 (0.63)	3.61 (0.51)	3.46 (0.56)	3.51 (0.54)
Negative social contacts	2.05 (0.62)	2.18 (0.68)	2.03 (0.58)	2.05 (0.63)	1.95 (0.57)
Waist	89.3 (15.4)	90.0 (15.5)	81.6 (14.3)	94.1 (15.6)	93.8 (12.3)
Glucose	86.4 (20.2)	86.9 (27.5)	82.6 (12.9)	88.7 (20.9)	88.5 (16.5)
Insulin	14.3 (10.8)	16.8 (13.2)	11.73 (7.1)	15.7 (12.6)	13.7 (9.4)
LDL cholesterol (gm/dl)	112.9 (31.9)	109.0 (30.4)	106.8 (29.0)	116.1 (34.3)	121.5 (32.5)
Triglycerides	98.8 (59.5)	80.9 (45.1)	91.7 (52.0)	103.3 (59.1)	122.3 (71.9)
HDL cholesterol	50.8 (14.5)	54.3 (13.7)	56.3 (14.9)	47.6 (13.7)	43.3 (11.1)
Sample descriptive information (in 2000 to 2001), mean (SD)					
Age	40.1 (3.6)	39.5 (3.9)	40.6 (3.4)	39.4 (3.7)	40.6 (3.3)
% With college degrees	47.8	31.9	64.9	25.0	61.7
% Income <$75,000	62.1	79.2	53.2	73.5	46.2
% Meeting criteria for MS	9.7	10.2	8.5	8.7	11.3

Cholesterol Education Program criteria, 9.7% of the sample was judged to have metabolic syndrome. This rate is lower than the national average because the CARDIA sample is still relatively young. Rates of metabolic syndrome did not vary significantly across the four race/sex subgroups.

Statistical Analysis

The main goal of our analyses was to determine whether the risky families model shown in Figure 1 explains variability in MF in the CARDIA dataset. A series of structural equation models, conducted using EQS 6.0, tested this multivariate model. The data were first evaluated for multivariate normality, and outliers were identified for the analyses described below. Second, a latent metabolic factor was tested initially for the entire CARDIA sample, and then a separate analysis was conducted to test for invariance of the latent factor across the four race/sex subgroups. Third, the latent metabolic factor was incorporated into a test of the whole model (shown in Figure 1) and tested using both the entire CARDIA sample and the race/sex subgroups. Fourth, modifications to this model were made based on the results of Lagrange multiplier tests and examination of standardized discrepancies; theoretically indicated covariations among variables were added to improve model fit.

Examination of Assumptions

To address skew, all biomarkers (waist, glucose, insulin, LDL, HDL, and trigylcerides) were logarithmically transformed before their inclusion in analyses. However, because transformed data were still not normally distributed, multivariate outliers were examined, and robust standard errors were used for indices of model fit (43).[1] The results presented here used the entire sample; unless otherwise noted, analyses excluding multivariate outliers were similar to those conducted using the entire sample.

Consistent with the recommendations of McDonald and Ho (44), we used several indicators of model fit to determine the adequacy of the theoretical models. Models that fit the data had standardized discrepancies close to 0, and root mean-square error of approximation (RMSEA) of 0.05 or less; those with RMSEA lower than 0.08 were acceptable. Normed fit index (NFI) and confirmatory fit index (CFI) estimates greater than 0.90 were indicators of acceptable model fit. The Satorra-Bentler scaled χ^2 (43) is also reported, although nonsignificance of the scaled χ^2 value is an inappropriate criterion of model fit in the present analyses because of the large sample size. Model χ^2, NFI, CFI, and RMSEA are reported for each of the major analyses.

MF Confirmatory Factor Analysis

A confirmatory factor analysis was conducted to determine whether waist, glucose, insulin, LDL, triglycerides, and HDL were appropriately modeled as indicators of a single latent metabolic factor. It should be noted that elevated blood pressure is also prognostic for metabolic syndrome. However, the analyses indicated that blood pressure was empirically distinct from the lipid measures and should not be modeled as an indicator of the MF latent variable. Other researchers (e.g., Matthews KA, personal communication, November 24, 2004) have also found blood pressure to be statistically distinct from other indicators of MF in healthy samples. Blood pressure was therefore not considered further in the analyses. An exploratory factor analysis, conducted for the entire sample as well as separately for each of the race/sex subgroups, indicated that a single metabolic factor was appropriate. The path to insulin was fixed to one; triglycerides and HDL cholesterol were allowed to covary. This model fit the data (Satorra-Bentler χ^2 (8) = 100.30, $p < .05$; NFI = 0.97; CFI = 0.97; RMSEA = 0.060), and each indicator made a statistically significant contribution to the latent factor.

Next, a multiple groups structural equation modeling (SEM) was conducted to determine whether the factor loadings were invariant across the four race/sex subgroups. If factor loadings across the subgroups are equivalent, then it is appropriate for the model to be tested using the entire sample. If the loadings differ across the subgroups, then the meaning of the latent dependent measure is not consistent, and an analysis on the entire sample is less meaningful. An initial multiple groups analysis confirmed that the latent model fit after the grouped data structure was considered (Satorra-Bentler χ^2 (32) = 107.24, $p < .05$; NFI = 0.97; CFI = 0.98; RMSEA = 0.027). However, when constraints of factor loading equality were placed on this model, Lagrange multiplier tests indicated that factor loadings differed across the subgroups.[2] Based on these results, the model was analyzed with the entire sample and separately for each of the four race/sex subgroups.

Test of the RF Model

The correlation matrix that formed the basis of the RF model is shown below the diagonal of Table 2. Initially, the RF model was analyzed as shown in Figure 1. That is, it was modeled without any covariation among the 6 biomarkers and without a direct link between childhood SES and MF. This model did not yield an acceptable fit (Satorra-Bentler χ^2 (61) = 836.24, $p < .05$; NFI = 0.88; CFI = 0.88; RMSEA = 0.063). An examination of standardized residuals and the Lagrange multiplier tests suggested that intercorrelations among the cholesterol measures would improve model fit. Therefore, a second model, in which triglycerides were allowed to correlate with both HDL and LDL, was tested.

[1] Multivariate outliers, as detected through Mahalanobis distance greater than 36.12, were identified separately for each of the four race/sex subgroups. All analyses were conducted with the entire sample and with a sample that omitted 76 outliers.

[2] Although space prohibits a more detailed discussion of subgroup differences in factor loadings, differences can be summarized as follows. Factor loadings for waist circumference, LDL, and HDL were larger for white women than for other groups, and the overall model fit best for white women. In addition, glucose contributed more to the latent factor for black women than for other groups and triglycerides were a stronger contributor for both white men and women than for black participants.

TABLE 2. Correlation Matrix (Below Diagonal) for Entire Sample and Discrepancies for Final Model (Above Diagonal)[a]

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Childhood SES	—	0.000	0.006	0.063	−0.026	0.036	0.000	−0.002	−0.003	−0.028	0.030	0.060	−0.042
2. Early family environment	−0.13**	—	−0.066	−0.025	−0.071	0.001	0.000	−0.039	−0.043	−0.010	−0.046	−0.011	0.022
3. Depression	−0.11**	0.27**	—	0.057	0.012	0.002	−0.071	−0.009	0.011	0.029	−0.055	−0.017	0.053
4. Hostility	0.01	0.12**	0.24**	—	0.104	0.128	0.061	−0.013	−0.012	−0.022	−0.044	−0.002	0.048
5. Positive social contacts	0.09**	−0.41**	−0.41**	−0.08**	—	0.007	−0.004	−0.025	−0.002	−0.015	0.026	0.005	0.011
6. Negative social contacts	−0.07**	0.31**	0.38**	0.29**	−0.37**	—	0.060	0.010	0.014	0.025	−0.053	0.011	0.030
7. Adult SES	0.33**	−0.24**	−0.37**	−0.07**	0.30**	−0.21**	—	−0.015	0.005	−0.008	0.080	0.033	−0.030
8. Waist	−0.13**	0.02	0.06**	0.02	−0.09**	0.07**	−0.11**	—	−0.023	−0.001	0.013	−0.004	−0.021
9. Glucose	−0.07**	−0.01	0.05*	0.05	−0.04*	0.05*	−0.05*	0.37**	—	0.045	0.005	0.008	0.017
10. Insulin	−0.13**	0.04*	0.09**	0.02	−0.07**	0.08**	−0.09**	0.60**	0.38**	—	−0.048	0.003	0.038
11. LDL cholesterol	−0.01	−0.03	−0.04*	−0.04*	0.01	−0.03	0.05*	0.23**	0.13**	0.14**	—	0.021	−0.061
12. Triglycerides	−0.01	0.03	0.02	0.02	−0.04*	0.05*	−0.02	0.41**	0.24**	0.36**	0.24**	—	−0.006
13. HDL cholesterol	0.04*	−0.01	0.01	0.03	0.05*	−0.01	0.03	−0.46**	−0.23**	−0.34**	−0.20**	−0.51**	—

* $p < .05$; ** $p < .001$.

[a] Zero-order correlation coefficients for all variables used in the analysis are shown below the diagonal. Standardized residuals computed for the final model are presented above the diagonal; lower residuals indicate better model fit. $n = 3225$.

Although this model was a much better fit with the data (Satorra-Bentler χ^2 (59) = 512.10, $p < .05$; NFI = 0.92; CFI = 0.93; RMSEA = 0.049; χ^2 change = 324.14, $p < .001$), Lagrange multiplier tests and standardized residuals suggested that childhood SES had a substantial direct effect on adult MF. Although this link had not been anticipated in the original model, it is consistent with research indicating the role of childhood SES on both childhood and adult health outcomes (45,46) and was therefore tested. The addition of a direct childhood SES-MF link was tested through the final model, and was a good fit with the data (Satorra-Bentler χ^2 (58) = 479.27, $p < .05$; NFI = 0.93; CFI = 0.94; RMSEA = 0.047; χ^2 change = 32.83, $p < .001$). Standardized discrepancies for the final model are shown above the diagonal in Table 2, and summary statistics for model fit are in Table 3.

As shown in the first column of Table 3 and in Figure 2(a), most of the links proposed in the RF model (7) were supported. As predicted, children from lower SES families were more likely to report growing up in a household characterized by conflict or by neglect (RF). RF environment was, in turn, associated with more maladaptive PsyF and lower adult SES. Maladaptive PsyF, as indicated by depression, hostility, and low positive/high negative social contacts, predicted increased metabolic risk. Of interest, although the direct path from childhood SES to MF was statistically significant, adult SES did not maintain a significant association with MF.

An alternative model addressed the possibility that a negative response bias (as embodied in the PsyF variable) might predict the reconstruction of RF. Accordingly, we repeated the SEM analysis but gave PsyF causal priority over RF. This model was a significantly poorer fit to the data ($\chi^2 = 29.39$, $p < .001$). Among other problems, there was no significant direct path from childhood SES to PsyF, and thus the alternative model does not address the mediation of the childhood SES-MF association.

Because the loadings of the biomarkers onto MF were not invariant across the race/sex subgroups, the final model was also tested separately for each subgroup.

Subgroup Analyses of the RF Model

With the exception of African-American men, the RF model resulted in an acceptable fit for each subgroup. There were differences among the groups in the importance of the various links in the model. Figure 2, b-d, shows the standardized path coefficients for each of the subgroups, and Table 3 summarizes model fit and unstandardized path coefficients for these three subgroups.[3]

For each of the three subgroups, lower childhood SES was associated with RF. In every case, RF predicted maladaptive PsyF and lower adult SES. Likewise, adult SES was associated with childhood SES, as well as with PsyF in each of the subsamples. However, for white men, neither PsyF nor childhood SES or adult SES was associated with adult MF. In contrast, PsyF and both childhood SES and adult SES predicted MF for white women. For African-American women, childhood SES and PsyF predicted MF, but adult SES was not associated with MF. Although the fit of the model for African-American men was not as good (Satorra-Bentler χ^2 (58) = 178.73, $p < .05$; NFI = 0.86; CFI = 0.90; RMSEA = 0.058) and path coefficients may be misleading, the overall pattern of path coefficients was similar to the model for white men (Figure 2b); however, the path between adult SES and MF was statistically significant for African-American men. Modifications did not result in a model that fit the data for African-

[3]The results of analyses conducted excluding multivariate outliers from the full and subsamples led to similar results; however, model fit was somewhat worse for white women and better for African-American women with the smaller samples. The link between childhood SES and MF dropped below the threshold for statistical significance in the smaller sample of African-American women.

TABLE 3. Fit Indices and Unstandardized Path Coefficients (With Robust Standard Errors) for Final Model Paths, Latent Factors, and Covariances

	Entire Sample (n = 3225)	African-American Women (n = 850)	White Women (n = 944)	White Men (n = 812)
Fit indices				
χ^2 (58 *df*)	530.36	161.11	200.71	188.97
Satorra-Bentler χ^2 (58 *df*)	479.27	134.21	189.68	169.02
NFI (robust)	0.93	0.92	0.91	0.90
CFI (robust)	0.94	0.95	0.93	0.93
RMSEA (robust)	0.047	0.039	0.049	0.049
Path model[a]				
Childhood SES → Early family environment	−0.088* (0.012)	−0.097* (0.026)	−0.099* (0.025)	−0.102* (0.022)
Early family environment → Adult SES	−0.221* (0.020)	−0.221* (0.040)	−0.193* (0.030)	−0.302* (0.042)
Childhood SES → Adult SES	0.223* (0.012)	0.133* (0.028)	0.193* (0.021)	0.155* (0.022)
Adult SES → Psychosocial functioning	−2.57* (0.18)	−3.33* (0.37)	−2.63* (0.38)	−1.19* (0.29)
Early family environment → Psychosocial functioning	3.41* (0.21)	4.58* (0.44)	2.47* (0.31)	3.37* (0.42)
Childhood SES → Metabolic functioning	−0.052* (0.009)	−0.050* (0.021)	−0.051* (0.015)	−0.032 (0.017)
Psychosocial functioning → Metabolic functioning	0.007* (0.002)	0.009* (0.004)	0.009* (0.004)	0.009 (0.006)
Adult SES → Metabolic functioning	−0.018 (0.013)	−0.014 (0.030)	−0.098* (0.025)	−0.041 (0.027)
Latent factors[a]				
Psychosocial functioning[b] →				
Hostility	0.023* (0.002)	0.024* (0.004)	0.020* (0.004)	0.027* (0.005)
Positive social contacts	−0.073* (0.004)	−0.073* (0.006)	−0.074* (0.006)	−0.093* (0.011)
Negative social contacts	0.072* (0.004)	0.074* (0.006)	0.069* (0.007)	0.079* (0.010)
Metabolic functioning[c] →				
Waist	0.348* (0.011)	0.291* (0.022)	0.378* (0.019)	0.231* (0.014)
Glucose	0.195* (0.009)	0.215* (0.023)	0.158* (0.015)	0.160* (0.017)
LDL cholesterol	0.193* (0.017)	0.127* (0.031)	0.272* (0.033)	0.073* (0.027)
Triglycerides	0.651* (0.027)	0.565* (0.047)	0.715* (0.046)	0.721* (0.055)
HDL cholesterol	−0.369* (0.015)	−0.296* (0.027)	−0.394* (0.024)	−0.257* (0.025)
Covariances				
LDL cholesterol: triglycerides	0.015* (0.003)	0.004 (0.005)	0.014* (0.005)	0.023* (0.005)
Triglycerides: HDL cholesterol	−0.036* (0.002)	−0.011* (0.003)	−0.021* (0.004)	−0.045* (0.004)

* $p < .05$.

[a] The error variance of each measured variable was fixed to 1. All error and disturbance variances were statistically significant.

[b] The path from psychosocial functioning to depression (not shown) was fixed to 1 for all analyses.

[c] The path from metabolic functioning to insulin (not shown) was fixed to 1 for all analyses.

CFI = comparative fit index; NFI = normed fit index; RMSEA = root mean-square error of approximation; SES = socioeconomic status.

American men. For this reason, path coefficients are not shown for this group.

DISCUSSION

This investigation tested a causal model that relates low SES and harsh parenting to adverse physiological and health-related outcomes via their impact on negative emotional states and social contacts (7), as portrayed in Figure 1. The results indicate that the model provided a good fit to the data for MF. Low childhood SES was associated with harsh parenting, which in turn was related to the psychosocial variables of depression, hostility, and poor social relations and to poorer standing on indicators of MF.

The results are consistent with evidence that ties low SES and early cold or harsh parenting to adverse health outcomes in adulthood (7,33). In previous writings (7,33), we had reasoned that the intermediate products of this association may be dysregulations in psychological and biological stress regulatory systems, which may represent early signs of potential pathology and translate into concrete risk factors for disease, in this case, adverse changes in MF. A previous study from our laboratory documented the applicability of this model to signs of potential HPA axis dysregulation (an elevated flat cortisol trajectory during a laboratory stress task) and to heightened heart rate and blood pressure responses to a laboratory stress task in men only (33). The present investigation

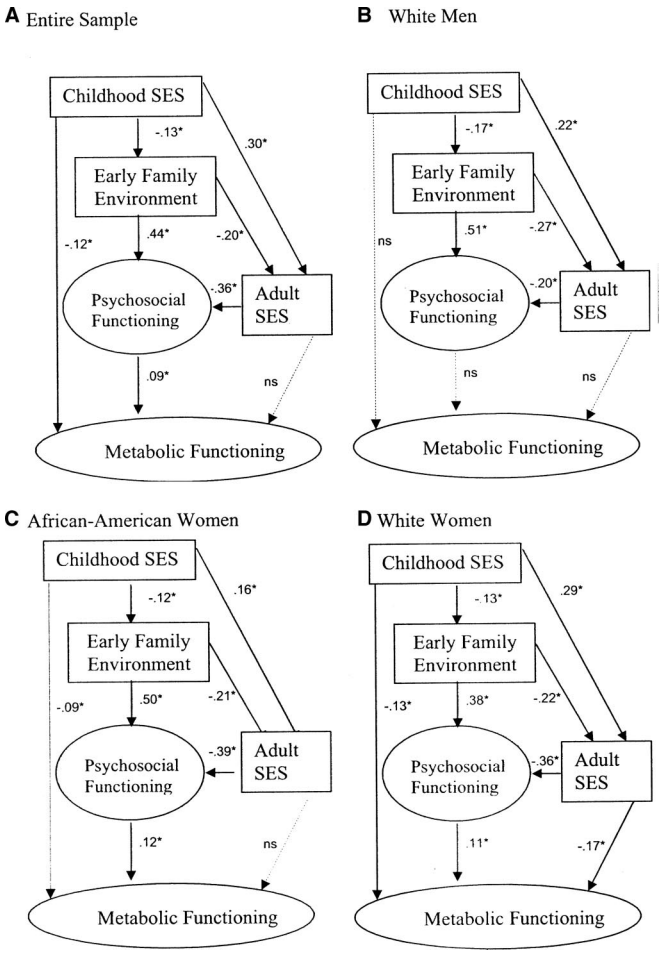

Figure 2. Final model standardized path coefficients for the entire sample (A), white men (B), African-American women (C), and white women (D). In the diagrams, solid lines represent statistically significant paths, whereas dashed lines represent paths that were included in the model but that were not statistically significant. Circles are used to represent latent variables, whereas boxes indicate measured variables. Consistent with SEM conventions, the arrows are shown as going from the latent construct to boxes indicating measured variables in the model. Standardized path coefficients for the entire sample on the latent variables are as follows: psychosocial functioning (depression = 0.64, hostility = 0.28, positive social contacts = −0.65, negative social contacts = 0.58); metabolic functioning (waist circumference = 0.84, glucose = 0.47, insulin = 0.72, LDL cholesterol = 0.26, triglycerides = 0.50, HDL cholesterol = −0.53).

is the first study to tie the RF model to a specific cluster of metabolic risk factors for disease.

In addition to support for mediation via RF, there was a direct path from childhood SES to MF, indicating that factors in addition to RF explain this link. These may include low birthweight, environmental toxins, poor diet and poor health care, and chronic stress associated with low SES (in addition to family environment) (47).

McEwen (12) and Seeman and colleagues (13) proposed an allostatic load model of stress in which they maintain that chronic or recurrent stress interacts with genetic or acquired risks, leading to cascading, potentially irreversible interactions between these predispositions and environmental factors; over time, these interactions and their accumulating effects can lead to large individual differences in susceptibility to stress, in biological markers of the cumulative effects of stress, and in stress-related physical and mental disorders. Although our findings do not directly test the allostatic load model, we have provided evidence that a chronically stressful early family environment can influence the development of symptomatology prognostic for metabolic syndrome.

It should be noted that, despite the overall support for the model, some of the path coefficients, especially from PsyF to MF, are weak. One reason is that the tested model does not include variability in MF that is due to genetic factors. Previous research suggests that genetic contributions account for as much as 40% of the variability in some of markers of MF (16). Health behaviors, which also contribute to MF, were also not included in the present analyses; the indicators in the CARDIA dataset were not strongly associated with MF overall but were instead differentially associated with individual indicators of MF in ways that could not be adequately statistically modeled. In addition, assessments of HPA axis functioning were not available in the present dataset. Over time, excess adrenal steroids can result in elevated blood glucose, abdominal obesity, and high cholesterol (48), and so HPA axis dysregulation represents a potential mediator in the model as well (33). In spite of the exclusion of direct or interactive genetic contributions, health behaviors, and indicators of HPA axis functioning, our data indicate that childhood environment and psychosocial variables in the RF model contribute to metabolic dysregulation. This result is especially noteworthy because our sample is still relatively young.

Our model was supported by the full CARDIA sample. However, this model was most predictive of variability in MF among women, especially white women. The better fit of the model in explaining women's MF is consistent with previous research indicating that lipid variables, as used in these analyses, are especially important contributors to MF and risk of disease for women (49). The model was least predictive for African-American men. African-American men were significantly more likely than the other three groups to report that their families "did not know what they were up to"; possibly, the impact of the family environment on African-American men's psychosocial and biological functioning was diluted by relatively less family influence, potentially coupled with greater peer group influence. This explanation is conjectural at present. Potential unexamined physiological differences may also contribute to the nonresults for this subgroup.

Limitations

There are several limitations to the present analysis. First is the fact that, with the exception of childhood SES, the data are largely cross-sectional, not longitudinal. Consequently, we have inferred causal paths from correlational data. Even within the constraints of SEM, inferring causality from correlational evidence has attendant risks, and so longitudinal evidence will be valuable for pursuing these links. Second, reconstructions of family environments may include an emotional overlay suggestive of response bias.

To address this issue, we point to the relatively poor fit of an alternative SEM model that gave PsyF causal priority over RF; this suggests that negativity is not leading to reports of a RF. We also note that the instrument on which our assessment of RF is based (8) has demonstrated a dose-response relationship to a broad array of hard health outcomes (e.g., cancer, CHD), and a response bias is highly unlikely to yield such effects. Nonetheless, reconstruction of a family environment is a limitation. A third limitation is that, as noted, the individual paths are weak, which is likely due to unmeasured genetically based risk factors, among other possibilities. A fourth limitation is that the relatively young and healthy sample does not directly address the applicability of the model to the clinical outcome of metabolic syndrome. A fifth limitation is the already noted unsuccessful effort to include health behaviors in the model.

CONCLUSIONS

Although the effects are modest in size, this investigation relates low childhood SES and a harsh early family environment to metabolic dysregulation among young adults. The results underscore the role of childhood SES and early family environment in predicting morbidity in adulthood and extend those findings to include dysregulation in MF. The results also point to the psychosocial and biological mechanisms that may underlie the relation of SES and early family environment to metabolic syndrome and, thus, help to explain SES-related health disparities. In addition, the results point to the potential importance of intervening with troubled families to enhance the likelihood of improving offspring long-term health.

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