Sex Differences in the Association of Childhood Socioeconomic Status With Adult Blood Pressure Change: The CARDIA Study

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Objective: To examine sex differences in the relation of childhood socioeconomic status (CSES) to systolic (SBP) and diastolic blood pressure (DBP) trajectories during 15 years, spanning young (mean [M] [standard deviation {SD}] = 30 [3] years) and middle (M [SD] = 45 [3] years) adulthood, independent of adult SES. **Methods:** A total of 4077 adult participants reported father's and mother's educational attainments at study enrollment (Year 0) and own educational attainment at enrollment and at all follow-up examinations. Resting BP also was measured at all examinations. Data from examination Years 5 (when participant M [SD] age = 30 [3] years), 7, 10, 15, and 20 are examined here. Associations of own adult (Year 5), mother's, and father's educations with 15-year BP trajectories were examined in separate multilevel models. Fully controlled models included time-invariant covariates (age, sex, race, recruitment center) and time-varying covariates that were measured at each examination (marital status, body mass, cholesterol, oral contraceptives/hormones, and antihypertensive drugs). Analyses of parental education controlled for own education. **Results:** When examined without covariates, higher education — own (SBP $\gamma = -0.03$, DBP $\gamma = -0.03$), mother's (SBP $\gamma = -0.02$, DBP $\gamma = -0.02$), and father's (SBP $\gamma = -0.02$, DBP $\gamma = -0.01$) — were associated with attenuated 15-year increases in BP ($p < .001$). Associations of own (but not either parent's) education with BP trajectories remained independent of standard controls. Sex moderated the apparent null effects of parental education, such that higher parental education—especially mother's, predicted attenuated BP trajectories independent of standard covariates among women (SBP $\gamma = -0.02$, $p = .02$; DBP $\gamma = -0.01$, $p = .04$) but not men (SBP $\gamma = 0.02$, $p = .06$; DBP $\gamma = 0.005$, $p = .47$; p interaction SBP $< .001$, p interaction DBP $= .01$). **Conclusions:** Childhood socioeconomic status may influence women's health independent of their own adult status. **Key words:** blood pressure, childhood socioeconomic status, multilevel modeling, sex differences.

BMI = body mass index; **BP** = blood pressure; **CARDIA** = Coronary Artery Risk Development in Young Adults Study; **CSES** = childhood socioeconomic status; **CVD** = cardiovascular disease; **DBP** = diastolic blood pressure; **OC/HRT** = oral contraceptives/hormone replacement therapy; **SBP** = systolic blood pressure; **SES** = socioeconomic status.

INTRODUCTION

Childhood socioeconomic status (CSES) is a powerful and reliable predictor of future cardiovascular disease (CVD) risk, independent of adult SES (1,2). Much of the research on CSES and CVD has been conducted with middle-aged and older adult samples and has focused on the prediction of clinical manifestations of CVD such as myocardial infarction (3) and mortality (2,4,5). Although important in establishing the link between relative socioeconomic deprivation during childhood and increased risk of cardiovascular pathology during adulthood, this research reveals little about the mechanisms that account for the association of CSES with CVD risk or the stage of disease development at which the influences of CSES on cardiovascular pathogenesis are most likely to become apparent.

Elevated blood pressure (BP), even in the high-normal range, is an important risk factor for CVD as well as for reduced life expectancy even in the absence of cardiovascular pathology (6). Thus, variation in adult BP may be one pathway through which CSES influences CVD risk. That a BP-related mechanism might explain the CSES-CVD association is supported by existing data that have shown higher CSES to correlate with lower average adult BP, whether measured during young, middle, or older adulthood (7–12). Moreover, in most cases, the association of CSES with adult BP was independent of adult SES (7,8,10). A limitation of this research is that the reported associations are essentially cross-sectional in that they describe the relation of CSES with future BP at a single specific adult age.

Additional support for a link between CSES and adult BP has been provided by prospective longitudinal analyses (including an earlier analysis of Coronary Artery Risk Development in Young Adults Study (CARDIA) data (12)) that have shown CSES to predict later *change* in BP (8,12). Even these findings are limited, however, in that neither study controlled for adult SES in their analyses. Because adult SES is associated with both CSES (13) and BP (14), failing to control for this factor leaves the possibility that the reported associations were actually attributable to concurrent SES. Knowing whether the effects of adult SES can be ruled out can provide a clue as to when during the life course childhood socioeconomic experiences begin to have their effect on adult cardiovascular risk.

Insofar as CSES relates prospectively to adult BP, the implications of this association may be more relevant to the future health of women than of men. Elevated BP may pose a greater risk for future clinical disease among women relative to men. For example, among participants in the Framingham Heart Study hypertension was found to account for 59% of congestive

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Work on this article was supported (or partially supported) by contracts from the University of Alabama at Birmingham, Coordinating Center, N01-HC-95095; the University of Alabama at Birmingham, Field Center, N01-HC-48047; the University of Minnesota, Field Center and Diet Reading Center (Year 20 Exam), N01-HC-48048; Northwestern University, Field Center, N01-HC-48049; and the Kaiser Foundation Research Institute, N01-HC-48050.

Preparation of the article was also facilitated by the Pittsburgh Mind-Body Center (HL076852, R24HL076858) and R01-HL095296-01 from the National Heart, Lung, and Blood Institute.

Received for publication June 6, 2011; revision received March 19, 2012.

DOI: 10.1097/PSY.0b013e31825e32e8

0033-3174/12/7407–0728

heart failure cases among women compared with 39% of cases among men (15). Likewise, elevated systolic BP was found to be a stronger risk factor for incident fatal or nonfatal myocardial infarction among female participants in the Reykjavik Study relative to their male counterparts (16). In addition to the evidence suggesting that hypertension may be more detrimental for women than for men, there is additional evidence to suggest that CSES may matter more for women's cardiovascular health. Specifically, findings from several studies that stratified their analyses by sex suggest that associations between CSES and CVD morbidity and mortality are stronger for women than for men (3–5).

The existing literature has not adequately tested whether the associations of CSES and BP and BP change vary by sex. Neither of the two studies that explicitly tested for a CSES-by-sex interaction observed a modifying effect of sex (10,17). However, analyses in both of these studies were limited to father's occupational class as the measure of CSES. It is possible that father's occupation does not reflect the dimensions of CSES that are most likely to differ between men and women in having an influence on future BP. For example, analyses of data from both the United States and international sources have shown maternal education to relate more strongly to girls' physical development relative to paternal education (18). Thus, it may be the association of the maternal contribution to CSES with adult BP that is sex specific.

Here we address several questions regarding the nature of the association between CSES and *longitudinal change* in adult BP that have not yet been answered by existing research. First, is the association of CSES with the rate of increase in adult BP independent of adult SES? Second, does the magnitude of the association between CSES and adult BP change throughout early and middle adulthood differ for men and women? Third, are mothers' contributions to CSES as important as—or possibly more important than fathers' contributions in regard to influencing adult BP change, and does the relative importance of either parent's contribution to CSES differ between women and men?

We elaborate on existing research on CSES and BP change by addressing the above questions using multilevel modeling. This approach offers several advantages. First, multilevel modeling allows us to take full advantage of CARDIA's longitudinal data collection by permitting the examination of BP trajectories over 15 years. Second, multilevel modeling is robust to missing data, thus allowing us to maximize our sample. Third, time-varying covariates can be incorporated into the analytic models, thus providing more precise control for potential confounding factors. Fourth, multilevel analysis permits the modeling of autocorrelated errors between subsequent measurements.

METHODS

Participants

In 1985 to 1986, 5115 adults aged 18 to 30 years[1] were recruited into CARDIA at four sites: Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. The sampling strategy resulted in a population-based cohort that was balanced by race (52% black), sex (55% female), and education (40% with ≤12 years of schooling) both overall and within each clinical center (see Friedman et al. (19) for additional detail). Follow-up examinations were conducted in 1987 to 1988 (Year 2), 1990 to 1991 (Year 5), 1992 to 1993 (Year 7), 1995 to 1996 (Year 10), 2000 to 2001 (Year 15), and 2005 to 2006 (Year 20). Retention rates for each of the seven examinations were 91%, 86%, 81%, 79%, 74%, and 72% of the surviving members of the baseline sample, respectively. Institutional review committee approval was obtained at each participating site, and written informed consent was obtained from participants at each examination.

Measures

Childhood SES

Parental education was assessed at Year 0 with two items that asked participants to report the highest grade (or year) of regular school completed by a) "your father/or the man responsible for you as a child" and b) "your mother/or the woman responsible for you as a child." Responses ranged from 0 (no formal schooling) to 20 or higher (≥4 years of graduate education). For the present analyses, two separate continuous variables were created representing father's and mother's total years of education, respectively.

Adult SES

Participant educational attainment was used as the measure of adult SES. Education was measured at all examination years using the same years of education scale that was used to report parental education. For the present analyses, own adult education was represented with a continuous variable indicating participants' total years of education at Year 5. Year 5 data were chosen because participants, who, when attending that examination, were 23 to 35 years and thus were likely finished with their education and living independently of their parents.

Blood Pressure

Systolic (SBP) and diastolic blood pressure (DBP) were measured at all examinations, with the participant in a seated position and after a 5-minute rest. Using the participant's right arm, three measurements were taken at 1-minute intervals, with the latter two averaged. Both SBP and DBP were recorded as Phase I and Phase V Korotkoff sounds. From Years 0 to 15, BP was measured with a Hawksley random zero sphygmomanometer (Hawksley, Sussex, United Kingdom); at Year 20, the *OmROn* HEM907HXL (Omron Corporation, Kyoto, Japan) was used to assess BP, calibrated in a CARDIA substudy to levels that would have been obtained using the sphygmomanometer (calibrated RZ SBP = 3.74 + 0.96 × OMRON average

[1]Although CARDIA was designed to recruit individuals between the ages of 18 and 30 years, twelve 17-year-olds, thirty-six 31-year-olds, and ten 32–35-year-olds were enrolled in the study.

SBP, $R^2 = 0.94$; calibrated RZ DBP = 1.30 + 1.97 × OMRON average DBP, $R^2 = 0.91$).

Antihypertensive Medication

At all CARDIA examinations, participants reported whether they currently were taking antihypertensive medication.

Covariates

With the exception of age, sex, and race, covariates were time varying. Thus, the measures described below were obtained at all CARDIA follow-up examinations that are included in the present report (i.e., 5, 7, 10, 15, and 20). All scales are documented on the CARDIA Web site, http://www.cardia.dopm.uab.edu/.

Demographics

Participant age, sex, and race (white, black) were assessed by self-report at Year 0. Marital status (married, widowed, divorced, separated, or never married) was assessed at all examination years. For the purposes of analysis, marital status was coded as a dichotomous variable (married = 1; not married = 0).

Behavioral Risk Factors

Data on smoking status (1 = current, 0 = former/never) and alcohol consumption (number of drinks per week) were collected at all CARDIA examinations. Because of the documented J-shaped association of alcohol consumption with CVD risk (20), we recoded alcohol consumption into a dichotomous variable indicating whether participants were moderate drinkers (≤14 drinks per week for men, ≤7 drinks per week for women) or not (nondrinkers; men consuming ≥14 drinks per week, women consuming ≥7 drinks per week). Physical activity scores were computed by multiplying participants' reported frequencies of engagement in 13 categories of exercise and recreational sport activity by the intensity of the activity (expressed in exercise units (EU)). Total activity scores (combines scores for both heavy and moderate intensity activities) are used in the present analyses. Additional details on scoring procedures are available elsewhere (21).

Physiological Risk Factors

Body mass index (BMI), high-density lipoprotein cholesterol, total cholesterol, and triglycerides were measured at all seven CARDIA examinations. Low-density lipoprotein cholesterol concentration was computed using the Friedewald formula (22). Procedures for the collection and measurement of physiological risk factors previously have been reported (19). A $\log_{10}$ transformation was used to normalize the distributions of BMI and total cholesterol before analysis.

Oral Contraceptive/Hormone Use

At all examinations, women reported whether they currently were taking oral contraceptives/hormone replacement therapy (OC/HRT). From these data, a single dichotomous variable was created to indicate whether women were currently taking either OCs or HRT.

Exclusions

Of the 5115 individuals who originally enrolled in CARDIA, 4352 were still enrolled at the Year 5 examination and 4329 had data on SBP, DBP, and all of the standard covariates (see next paragraphs) from at least one examination between Years 5 and 20. From this group, 275 participants were excluded from the present analyses for having no data on either parent's educational attainment. The following report is based on the remaining 4077 participants. Relative to the 1038 participants (5115 at enrollment – 4077 in present sample) who enrolled in CARDIA at Year 0 but whose data were not examined here, the present sample was older (mean age at enrollment = 25.0 versus 24.4 years), more educated (median years of education at enrollment = 14 versus 12), more likely to be female (55% versus 53% women), and less likely to be black (47% versus 69%).

Statistical Analyses

All statistical analyses were performed using SAS v9.1 (SAS Institute, Inc, Cary, NC). Descriptive statistics are presented as mean (standard deviation), median (interquartile range), or frequency (%). Also, *t* tests were used to compare means of continuous variables with Satterthwaite adjustment to correct for unequal variances between groups.

Linear multilevel modeling (SAS Proc MIXED, SAS Institute, Inc., Cary, NC) was used to examine the associations of own adult and father's and mother's educations with 15-year adult BP trajectories. Repeated BP measurements obtained at CARDIA examination Years 5 (baseline), 7, 10, 15, and 20 were examined as dependent variables, and each of the three education variables was examined as predictors. Unless otherwise indicated, all analyses included a set of standard controls composed of both time-invariant covariates (age, sex, race, and CARDIA center) and a time-varying covariates that were measured at each of the aforementioned CARDIA follow-up examinations (marital status, BMI, total cholesterol, use of OCs or HRT (women only), and use of antihypertensive medication). Because BP trajectories may differ as a function of age, sex, and race, the interactions of each of these variables with time were included as covariates in addition to their main effects. Also, because some participants reported education data for only one parent (see Table 1 footnote), analyses involving either parental education variable included an additional dummy variable indicating whether the other parent's educational attainment was known and the interaction of that variable with time. All predictor variables and covariates were centered at the sample mean before analysis. We specified an unstructured covariance structure (TYPE = UN) for the estimation of intercept (average baseline BP) and slope (BP trajectory) values (between-person) and a first-order autoregressive error structure (AR1) to model autocorrelation between adjacent BP measurements (within-person). Separate models were run for SBP, DBP, and for own, father's, and mother's educations.

Effect modification analyses were used to examine whether associations of own adult, father's, and mother's educations with adult BP trajectories differ between men and women. These

TABLE 1. Participant Characteristics at Baseline (CARDIA Year 5) Separately by Sex

	Men (n = 1838)	Women (n = 2239)	$p_{sex\ diff}$
Adult predictors			
Age, M (SD), y	29.94 (3.56)	29.98 (3.68)	.70
SBP, M (SD), mm Hg	111.85 (11.13)	104.40 (10.76)	.001
DBP, M (SD), mm Hg	71.75 (9.60)	67.25 (9.66)	.001
Own (adult) education, median (IQR), y	14.0 (12.0–16.0)	15.0 (13.0–16.0)	.40
African American, n (%)	826 (44.9)	1091 (48.7)	.02
Married, n (%)	736 (40.0)	918 (41.0)	.63
BMI, median (IQR), kg/m^2	25.1 (22.9–28.1)	24.2 (21.5–29.0)	.42
Total cholesterol, median (IQR), mg/dL	175.4 (154.0–200.2)	173.8 (153.4–194.8)	.003
Taking antihypertensive drugs, n (%)	24 (1.3)	40 (1.8)	.22
Using OC/HRT (women), n (%)		610 (27.2)	
Childhood predictors, M (SD)			
Mother's education, y	13.17 (2.70)	12.89 (2.85)	.002
Father's education, y	13.25 (3.56)	13.07 (3.72)	.16

CARDIA = Coronary Artery Risk Development in Young Adults Study; M = mean; SD = standard deviation; SBP = systolic blood pressure; DBP = diastolic blood pressure; IQR = interquartile range; BMI = body mass index; OC/HRT = oral contraceptives/hormone replacement therapy.
Mother's education: men, n = 1798; women, n = 2206.
Father's education: men, n = 1646; women, n = 1931.

models were identical to the main effect models described previously, save for the addition of the relevant sex-by-education and sex-by-education-by-time cross-product terms. P values are presented for interaction terms.

RESULTS

Participant Characteristics

Table 1 presents sample characteristics at baseline (CARDIA Year 5) separately for men and women. On average, men had higher baseline SBP and DBP than did women. In terms of education, men and women did not differ on their own total years of schooling or on the amount of education attained by their fathers. By comparison, men's mothers were more educated than women's mothers.

Main Analyses

Associations Between SES and BP Change in the Entire Sample

Tables 2 and 3 display the effects of own, father's, and mother's educations on average baseline BP and 15-year slopes of adult

TABLE 2. Effects of Own, Father's, and Mother's Educations on Adult SBP Intercepts and 15-Year Slopes

	Model 1		Model 2		Model 3		Interaction With Sex
	Estimate	SE	Estimate	SE	Estimate	SE	p
Own education							
Effect on intercept	−0.68	0.07***	−0.36	0.07***			.02
Effect on slope	−0.03	0.01***	−0.02	0.01*			.11
Father's education							
Effect on intercept	−0.41	0.05***	−0.13	0.05**	−0.08	0.05	.53
Effect on slope	−0.02	0.004***	−0.005	0.005	−0.002	0.005	.02
Mother's education							
Effect on intercept	−0.33	0.06***	−0.16	0.06**	−0.09	0.06	.50
Effect on slope	−0.02	0.01***	−0.006	0.01	−0.003	0.01	.001

SBP = systolic blood pressure; SE = standard error; CARDIA = Coronary Artery Risk Development in Young Adults Study; BMI = body mass index; OC/HRT = oral contraceptives/hormone replacement therapy; SES = socioeconomic status.
Model 1: includes terms for time, education, and time-by-education interaction.
Model 2: Model 1 + time-invariant covariates (age, sex, race, CARDIA center), time-varying covariates (BMI, total cholesterol, OC/HRT use, antihypertensive medication use, marital status), and interactions of age, sex, and race with time; childhood SES models also control for whether the other parent's education is known and the interaction of that variable with time.
Model 3: Model 2 + own adult education and time-by-own adult education interaction.
* $p < .05$, ** $p < .01$, *** $p < .001$.

TABLE 3. Effects of Own, Father's, and Mother's Educations on Adult DBP Intercepts and 15-Year Slopes

	Model 1		Model 2		Model 3		Interaction with Sex
	Estimate	SE	Estimate	SE	Estimate	SE	*p*
Own education							
Effect on intercept	−0.27	0.06***	−0.13	0.06*			.004
Effect on slope	−0.03	0.01***	−0.02	0.01**			.15
Father's education							
Effect on intercept	−0.30	0.04***	−0.06	0.04	−0.03	0.04	.33
Effect on slope	−0.01	0.004***	−0.005	0.004	−0.003	0.004	.51
Mother's education							
Effect on intercept	−0.24	0.05***	−0.06	0.05	−0.03	0.05	.67
Effect on slope	−0.02	0.004***	−0.01	0.005	−0.005	0.005	.01

DBP = diastolic blood pressure; SE = standard error; CARDIA = Coronary Artery Risk Development in Young Adults Study; BMI = body mass index; OC/HRT = oral contraceptives/hormone replacement therapy; SES = socioeconomic status.
Model 1: includes terms for time, education, and time-by-education interaction.
Model 2: Model 1 + time-invariant covariates (age, sex, race, CARDIA center), time-varying covariates (BMI, total cholesterol, OC/HRT use, antihypertensive medication use, marital status), and interactions of age, sex, and race with time; childhood SES models also control for whether the other parent's education is known and the interaction of that variable with time.
Model 3: Model 2 + own adult education and time-by-own adult education interaction.
* $p < .05$, ** $p < .01$, *** $p < .001$.

SBP and DBP, respectively. When examined with Model 1, higher own adult education as well as higher father's and mother's educations were associated with a lower average baseline SBP as well as a smaller average increase in SBP over time. Similar findings emerged for DBP. The apparent protective effects of own adult education on SBP and DBP remained when the standard controls were included in the model (Model 2). Both parental education variables also continued to predict a lower average baseline SBP when examined with Model 2. However, addition of the standard controls reduced the effects of father's and mother's educations on SBP slope, average baseline DBP, and DBP slope to nonsignificance. All associations of parental education with SBP and DBP lost significance when own adult education was included in the models (Model 3).

Modifying Effect of Sex

To determine whether the associations of own adult education with SBP and DBP differed between men and women, the following interaction terms were included in the Model 2 analyses in addition to the main effects of own adult education and sex and the education-by-time and sex-by-time interactions: sex-by-education and sex-by-education-by-time. Similarly, to

TABLE 4. Associations of Own, Father's, and Mother's Educations With SBP and DBP Intercepts and Slopes by Sex

	SBP				DBP			
	Men		Women		Men		Women	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Own education								
Effect on intercept	−0.25	0.10*	−0.48	0.10***	−0.11	0.10	−0.16	0.08*
Effect on slope	−0.007	0.01	−0.02	0.01*	−0.007	0.01	−0.02	0.01**
Father's education								
Effect on intercept	−0.11	0.08	−0.05	0.07				
Effect on slope	0.004	0.01	−0.005	0.01				
Mother's education								
Effect on intercept	−0.12	0.09	−0.07	0.08	−0.06	0.08	−0.03	0.07
Effect on slope	0.02	0.01†	−0.02	0.01*	0.005	0.01	−0.01	0.01*

SBP = systolic blood pressure; DBP = diastolic blood pressure; SE = standard error; CARDIA = Coronary Artery Risk Development in Young Adults Study; BMI = body mass index; OC/HRT = oral contraceptives/hormone replacement therapy; SES = socioeconomic status.
Models include time invariant covariates (age, race, CARDIA center), time-varying covariates (BMI, total cholesterol, OC/HRT use (women only), antihypertensive medication use, marital status), and interactions of age and race with time; childhood SES models also control for whether the other parent's education is known and the interaction of that variable with time as well as for own education and own education-by-time interaction.
* $p < .05$, ** $p < .01$, *** $p < .001$.
† $p < .10$

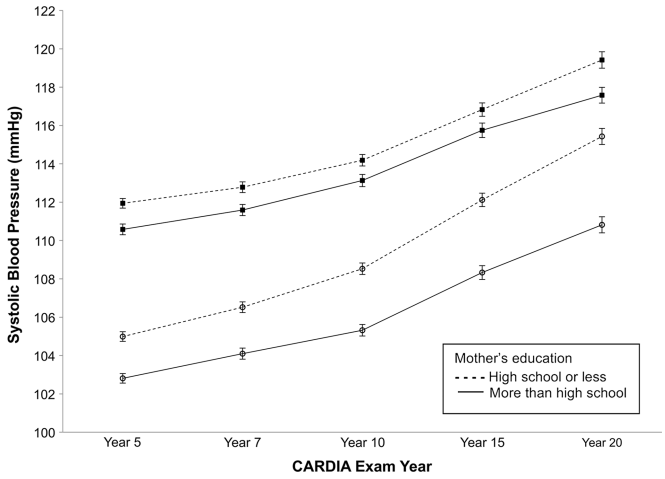

Figure 1. Modifying the effect of sex on the association of mother's education with 15-year change in adult SBP. Data points represent predicted SBP values obtained from Model 3 (with the addition of the sex-by-mother's education and sex-by-mother's education-by-time interaction terms) by CARDIA examination year. Filled squares represent men and open circles represent women. SBP = systolic blood pressure; CARDIA = Coronary Artery Risk Development in Young Adults Study.

determine whether the effects of parental education differed between men and women, the following interaction terms were included in the Model 3 analyses in addition to the main effects of father's (or mother's) education and sex and the relevant parent education-by-time and sex-by-time interactions: sex-by-parent education and sex-by-parent education-by-time. Results of these analyses revealed that sex modified the multivariable cross-sectional associations of own adult education with average SBP and average DBP ($p < .02$ for sex-by-education interactions). Sex also modified the multivariable associations of both father's and mother's educations with SBP slope and mother's education with DBP slope ($p < .02$ for sex-by-parent education-by-time interactions). Analyses by sex revealed that the apparent protective effect of greater own educational attainment on average BP was driven largely by the findings for women (Table 4). This sex difference was especially marked for DBP wherein the protective effect was present among women but not among men. The attenuating effect of higher maternal education on 15-year SBP and DBP trajectories similarly was apparent for women only (Table 4). By comparison, sex-specific examinations of the association of father's education with SBP slope revealed no significant effects among men or women. For the purposes of illustration, Figures 1 and 2 present trajectories of SBP and DBP, respectively, at low (high school or less) and high (more than high school) levels of mother's education separately for women and for men.

Exploratory Analyses

A previous report from CARDIA found that black women showed the highest incidence of hypertension during the 20-year follow-up (23). Thus, we examined whether race modified the associations of own adult and parental education on women's average BP and BP trajectories. To do so, we included in each multivariate analysis the relevant race-by-education and race-by-education-by-time interactions. Race did not modify the effects of own, father's, or mother's education on average SBP ($p > .10$) or SBP slope ($p > .14$). By comparison, race did modify the effect of father's education on DBP slope ($p < .05$), such that the protective effect of having a father with greater educational attainment was apparent among white women (estimate = −0.01, standard error = 0.01, $p = .03$) but not among black women (estimate = 0.01, standard error = 0.01, $p = .23$). No other modifying effects of race on the associations of own or parental education on average DBP ($p > .21$) or DBP slope ($p > .09$) were significant.

Given the association between SES and the likelihood of engaging in health-promoting (or health risk) behaviors (24), we also examined whether smoking status, moderate drinking, and physical activity, accounted for the protective effects of women's own adult educational attainments on average BP and of women's own and mothers' educations on 15-year BP trajectories. Results of analyses that included all three behaviors as time-varying covariates did not differ substantially from those reported in Table 4 (data not shown), thus suggesting that the effects of greater own and maternal educational attainment on BP cannot be accounted for by women's participation in these behaviors during the 15-year follow-up.

DISCUSSION

In the present report, we used data from the CARDIA study to examine whether CSES—operationalized as both father's and mother's educational attainments—is related to 15-year SBP and DBP trajectories during middle adulthood. A previous report from CARDIA showed that higher educational attainment for the parent with more schooling was associated with an attenuated increase in SBP and DBP across the 10 years spanning the Year 5 and Year 15 follow-up examinations (12). Unsurprisingly, our findings for BP trajectories were similar to those reported by Lehman and colleagues in that higher education for both fathers and mothers was related to lower average

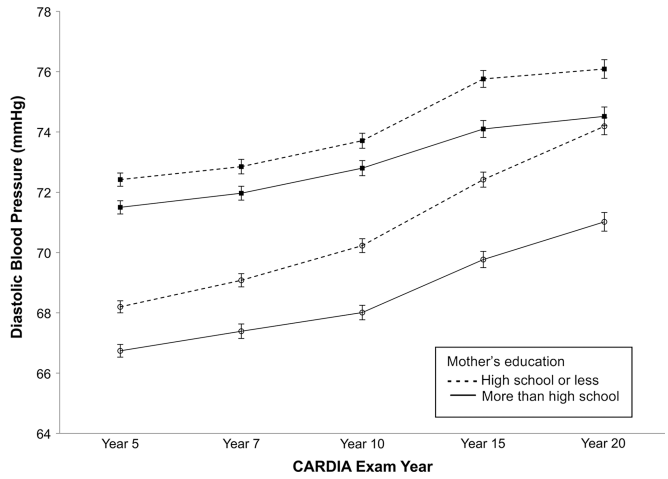

Figure 2. Modifying the effect of sex on the association of mother's education with 15-year change in adult DBP. Data points represent predicted DBP values obtained from Model 3 (with the addition of the sex-by-mother's education and sex-by-mother's education-by-time interaction terms) by CARDIA examination year. Filled squares represent men and open circles represent women. DBP = diastolic blood pressure; CARDIA = Coronary Artery Risk Development in Young Adults Study.

SBP and DBP and to flatter SBP and DBP slopes across the 15 years. Associations with BP slope were lost, however, when standard covariates were included in the models. Associations with average BP were lost as well when participants' own adult educational attainment was included in addition to the standard covariates. These findings extend those of Lehman et al. because they suggest that the relation of higher childhood SES to lower adult BP, *when examined in the entire sample*, may be due to the increased likelihood of children with more highly educated parents to ultimately attain higher educations themselves, with factors associated with adult SES rather than childhood SES influencing BP and BP trajectories. This pattern of findings was qualified, however, when sex was examined as a modifier.

Addressing the question of whether men and women differ in regard to the association of childhood SES with adult BP outcomes was the central aim of the present study, and the evidence we present here clearly supports a stronger effect of CSES among women relative to men, particularly when mother's educational attainment was used as the measure of childhood SES. Among women, increasing maternal education was associated with flatter SBP and DBP trajectories across the 15 years of follow-up. By comparison, maternal education was unrelated to BP trajectories among men. Importantly, the attenuating effect of mother's education on women's BP slopes was independent of women's own adult educational attainment. Thus, among women, factors unique to the experience of having been reared by a mother with higher compared with lower levels of education extend into adulthood to affect BP trajectories throughout young and middle adulthood. Such factors could influence women's development during early critical periods that may have latent effects that appear much later in life or they could establish a trajectory of behavioral and environmental exposures that accumulate over time to influence women's health throughout their life course.

Although the present stronger findings for women are consistent with trends reported in the extant research, the *absence* of a finding for men—rather than an observable but comparatively weaker finding—was unexpected. One explanation might involve female relative to male children being more receptive to the health benefits associated with increased parental education. Higher education is associated with modeling of better health behaviors among parents of both male and female adolescents (25). However, girls are more likely than boys to internalize parental rules (26) and report greater amounts of parental monitoring and more strongly enforced normative expectations of behavior (27). Thus, it is possible that the transmission of cardioprotective behaviors from higher SES parents will be more "taken to heart" by daughters than by sons. Although the three health-related behaviors examined here—smoking status, moderate drinking, and physical activity—did not explain the association of higher own or mother's education with attenuated BP trajectories among women, it remains possible that other health-related factors such as engaging in preventive health care might play some role in the protective effects of higher SES and CSES for women's BP.

That girls may be more likely than boys to model parental health-related behaviors may offer at least a partial explanation for why mother's rather than father's educational attainment predicts women's adult BP trajectories. Because mothers tend to spend more time with their children than do fathers, they may be more influential than fathers are as modelers of healthy (or unhealthy) behaviors. Children's nutrient intakes, for example, are more strongly correlated with their mother's than their father's intakes (28). Moreover, because daughters are more likely to identify with their mothers and sons with their fathers (29), this greater maternal influence would be more marked among girls than among boys.

Because a previous investigation of CARDIA data reported that black women showed the highest incidence of hypertension during the 20-year follow-up (23), we examined whether race modified the association of mother's and father's educations with women's average BP and BP trajectories. Our results showed a modifying effect of race on the association of father's educational attainment with DBP trajectories such that the attenuating effect of having a more highly educated father was apparent among white but not black women. This apparent race difference may in part be due to the income disparity between blacks and whites that occurs at all levels of educational attainment (30). Because having a higher education may not bring with it the same rewards for black fathers relative to their white counterparts, their children similarly may not have access to the same health-promoting resources as their white peers.

All findings reported here were independent of several physiological and medical factors that are known to influence BP. That CSES should influence women's adult BP trajectories independent of adult BMI in particular contrasts with findings from previous research wherein adult BMI explained most of the increase in SBP associated with having a father in a manual occupation (8). It is possible that BMI-related mechanisms linking CSES with adult BP will be more likely to involve weight and weight gain during childhood than during adulthood. Overweight children are more likely than their normal-weight peers both to have parents with low education (31) and to be overweight as adults (32). However, long-term consequences of childhood overweight may be independent of adult weight status. For example, childhood obesity has been found to predict greater carotid artery intima-media thickness at a mean age of 36 years independent of concurrent adult BMI (33).

A limitation of the present study is that only 85% of participants provided education data for both parents. Also, parental education was the only measure of CSES. Other childhood physical environmental factors associated with SES—such as family wealth and income, housing quality, and neighborhood safety additionally could influence later adult health (1). Another limitation is that the association between CSES and adult BP change that we report here is an elaboration of an earlier CARDIA analysis (12). Although not an independent replication of the earlier work, the present study makes several unique contributions including use of multilevel modeling, examination of the individual educational attainments of both mothers

and fathers, control for adult SES, and formally testing the modifying effect of sex. Finally, the effect for women that we report here is small. Consider, for example, two women with equivalent SBP at Year 5 but whose mothers differed in educational attainment by 4 years (i.e., high school graduate versus bachelor's degree). By Year 20, these two women would differ in SBP, with the daughter of the more highly educated mother having an SBP that is 1.2 mm Hg lower than the daughter of the less educated mother. However, at Year 20, women in the CARDIA sample are 38 to 50 years of age. As these women continue to age, this difference will widen, thus lending added risk to women with less educated mothers as they approach the period in life when women's trajectories of risk for clinically manifest CVD begin to steepen.

REFERENCES

1. Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. Ann N Y Acad Sci 2010;1186:37–55.
2. Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. Epidemiol Rev 2004;26:7–21.
3. Hamil-Luker J, O'Rand AM. Gender differences in the link between childhood socioeconomic conditions and heart attack risk in adulthood. Demography 2007;44:137–58.
4. Claussen B, Davey Smith G, Thelle D. Impact of childhood and adult socioeconomic position on cause specific mortality: the Oslo Mortality Study. J Epidemiol Community Health 2003;57:40–5.
5. Naess O, Strand BH, Smith GD. Childhood and adulthood socioeconomic position across 20 causes of death: a prospective cohort study of 800,000 Norwegian men and women. J Epidemiol Community Health 2007;61: 1004–9.
6. Franco OH, Peeters A, Bonneux L, de Laet C. Blood pressure in adulthood and life expectancy with cardiovascular disease in men and women. Hypertension 2005;46:280–6.
7. Blane D, Hart CL, Davey Smith G, Gillis CR, Hole DJ, Hawthorne VM. Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. BMJ 1996;313:1434–8.
8. Hardy R, Kuh D, Langenberg C, Wadsworth ME. Birthweight, childhood social class, and change in adult blood pressure in the 1946 British birth cohort. Lancet 2003;362:1178–83.
9. Kivimaki M, Kinnunen M-L, Pitkanen T, Vahtera J, Elovainio M, Pulkkinen L. Contribution of early and adult factors to socioeconomic variation in blood pressure: thirty-four-year follow-up study of school children. Psychosom Med 2004;66:184–9.
10. Power C, Atherton K, Strachan DP, Shepherd P, Fuller E, Davis A, Gibb I, Kumari M, Lowe G, Macfarlane GJ, Rahi J, Rodgers B, Stansfeld S. Life-course influences on health in British adults: Effects of socio-economic position in childhood and adulthood. Int J Epidemiol 2007;36:532–9.
11. Kivimaki M, Lawlor DA, Davey Smith G, et al. Early socioeconomic position and blood pressure in childhood and adulthood: the Cardiovascular Risk in Young Finns Study. Hypertension 2006;47:39–44.
12. Lehman BJ, Taylor SE, Kiefe CI, Seeman TE. Relationship of early life stress and psychological functioning to blood pressure in the CARDIA study. Health Psychol 2009;28:338–46.
13. Behrman JR, Taubman P. The intergenerational correlation between children's adult earnings and their parents' income: results from the Michigan Panel Survey of Income Dynamics. Rev Income Wealth 1990;36:115–27.
14. Colhoun HM, Hemingway H, Poulter NR. Socio-economic status and blood pressure: an overview and analysis. J Hum Hypertens 1998;12: 91–110.
15. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KKL. The progression from hypertension to congestive heart failure. JAMA 1996;275:1557–62.
16. Jonsdottir LS, Sigfusson N, Gudnason V, Sigvaldason H, Thorgeirsson G. Do lipids, blood pressure, diabetes, and smoking confer equal risk of myocardial infarction in women as in men? The Reykjavik Study. J Cardiovasc Risk 2002;9:67–76.
17. Kivimaki M, Smith GD, Elovainio M, et al. Socioeconomic circumstances in childhood and blood pressure in adulthood: the Cardiovascular Risk in Young Finns Study. Ann Epidemiol 2006;16:737–42.
18. Thomas D. Like father, like son; like mother, like daughter: parental resources and child height. J Hum Resour 1994;29:950–88.
19. Friedman GD, Cutter GR, Donahue RP, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. J Clin Epidemiol 1988;41:1105–16.
20. Corrao G, Bagnardi V, Zambon A, La Vecchia C. A meta-analysis of alcohol consumption and the risk of 15 diseases. Prev Med 2004;38:613–9.
21. Jacobs DR, Hahn L, Haskell WL, Pirie P, Sidney S. Validity and reliability of a short physical activity history: CARDIA and the Minnesota Heart Health Program. J Cardiopulm Rehabil 1989;9:448–59.
22. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499–502.
23. Levine DA, Lewis CE, Williams OD, et al. Geographic and demographic variability in 20-year hypertension incidence. The CARDIA Study. Hypertension 2011;57:39–47.
24. Winkleby MA, Fortmann SP, Barrett DC. Social class disparities in risk factors for disease: eight year prevalence patterns by level of education. Prev Med 1990;19:1–12.
25. Aufseeser D, Jekielek S, Brown B. The Family Environment and Adolescent Well-being: Exposure to Positive and Negative Family Influences. Washington, DC: Child Trends and the National Adolescent Health Information Center; 2006.
26. Kochanska G, Woodard J, Kim S, Koenig JL, JE Y, Barry RA. Positive socialization mechanisms in secure and insecure parent-child dyads: two longitudinal studies. J Child Psychol Psychiatry 2010;51:998–1009.
27. Windle M, Brener N, Cuccaro P, et al. Parenting predictors of early-adolescents' health behaviors: Simultaneous group comparisons across sex and ethnic groups. J Youth Adolesc 2009;39:594–606.
28. Oliveria SA, Ellison RC, Moore LL, Gillman MW, Garrahie EJ, Singer MR. Parent-child relationships in nutrient intake: the Framingham Children's Study. Am J Clin Nutr 1992;56:593–8.
29. Acock A, Yang WS. Parental power and adolescents' parental identification. J Marriage Fam 1984;46:487–95.
30. US Census Bureau. Current Population Survey Annual Social and Economic Supplement/Income, 2011. Available at: http://www.census.gov/hhes/www/cpstables/032011/perinc/new03_000.htm. Accessed January 6, 2012.
31. Wang Y. Cross-national comparison of childhood obesity: the epidemic and the relationship between obesity and socioeconomic status. Int J Epidemiol 2001;30:1129–36.
32. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obes Rev 2008;9:474–88.
33. Freedman DS, Patel DA, Srinivasan SR, et al. The contribution of childhood obesity to adult carotid intima-media thickness: the Bogalusa Heart Study. Int J Obes 2008;32:749–56.