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

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Abstract

Objective—To examine sex differences in the relation of childhood socioeconomic status (CSES) to systolic (SBP) and diastolic (DBP) blood pressure trajectories during 15-years spanning young (30 ± 3 years) and middle (45 ± 3 years) adulthood, independent of adult SES.

Method—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 exams. Resting BP also was measured at all exams. Data from exam Years 5 (when participant mean 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 exam (marital status, body mass, cholesterol, oral contraceptives/hormones, antihypertensives). Parental education analyses 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<0.001). 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 $\gamma= -.02$, p=.02; DBP $\gamma= -.01$, p=0.04) but not men (SBP $\gamma=0.02$, p=0.06; DBP $\gamma=0.005$, p=0.47; p-interaction SBP<0.001, p-interaction DBP=0.01).

Conclusion—CSES may influence women's health independent of their own adult status.

Keywords

blood pressure; childhood socioeconomic status; multilevel modeling; sex differences

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 terms of 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 nor 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 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 heart failure cases among women compared to 39% of cases among men (15). Likewise, elevated systolic BP was found to be a stronger risk factor for incident fatal or non-fatal 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 may not reflect the dimensions of CSES that are most likely to differ between men and women in terms of 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 upon 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; and fourth, multilevel analysis permits the modeling of auto-correlated errors between subsequent measurements.

METHODS

Participants

In 1985–1986, 5115 adults aged 18–30 years[1] were recruited into CARDIA at four sites: Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA. 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 (19) for additional detail). Follow-up examinations were conducted in 1987–1988 (Year 2), 1990–1991 (Year 5), 1992–1993 (Year 7), 1995–1996 (Year 10), and 2000–2001 (Year 15), and 2005–2006 (Year 20). Retention rates for each of the seven exams 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+ (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 exam 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 exam, were

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[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.

23–35 years of age and thus were likely finished with their education and living independently of their parents.

Blood pressure—SBP and DBP were measured at all exams, with the participant in a seated position and following a 5-minute rest. Using the participant's right arm, three measurements were taken at 1-minute intervals, with the latter two averaged. 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+.96*OMRON average SBP, R-square=.94; calibrated RZ DBP=1.30+1.97*OMRON average DBP, R-square=.91).

Antihypertensive medication—At all CARDIA exams, 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 exams that are included in the present report (i.e., 5, 7, 10, 15, and 20). All scales are documented on the CARDIA website, 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 exam years. For 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 exams. Because of the documented J-shaped association of alcohol consumption with cardiovascular disease risk (20), we re-coded alcohol consumption into a dichotomous variable indicating whether participants were moderate drinkers (14 drinks/week for men; 7 drinks/week for women) or not (nondrinkers; men consuming 14 drinks/week; women consuming 7 drinks/week). Physical activity scores were computed by multiplying participants' reported frequencies of engagement in thirteen 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 exams. 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 prior to analysis.

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

Exclusions

Of the 5115 individuals who originally enrolled in CARDIA, 4352 were still enrolled at the Year 5 exam, and 4329 had data on SBP, DBP, and all of the standard covariates (see below) from at least one exam 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 *vs.* 24.4), more educated (median years of education at enrollment=14 *vs.* 12), more likely to be female (55% *vs.* 53% women), and less likely to be black (47% *vs.* 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, SD), median (interquartile range, IQR), or percent (*n*). 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) 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 exam years 5 (baseline), 7, 10, 15, and 20 were examined as dependent variables, and each of the three education variables were examined as predictors. Unless otherwise indicated, all analyses included a set of standard controls comprised 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 exams (marital status, body mass index, total cholesterol, use of oral contraceptives or hormone replacement therapy [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 prior to analysis. We specified an unstructured covariance structure (TYPE=UN) for 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 models were identical to the main effect models described above, 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 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 non-significance. 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-X-time and sex-X-time interactions: sex-X-education and sex-X-education-X-time. Similarly, to 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-X-time and sex-X-time interactions: sex-X-parent education and sex-X-parent education-X-time.

Results of these analyses revealed that sex modified the multivariable cross-sectional associations of own adult education with average SBP and average DBP ($ps < .02$ for sex-X-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 ($ps < .02$ for sex-X-parent education-X-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 (see 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 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 over 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-X-education and race-X-education-time interactions. Race did not modify the effects of own, father's, or mother's education on average SBP ($Ps>.10$) or SBP slope ($Ps>14$). 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 = −01, SE = .01, $P=.03$) but not among black women

(estimate = .01, SE = .01, P=.23). No other modifying effects of race on the associations of own or parental education on average DBP (Ps>.21) or DBP slope (Ps>.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 exams (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 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 as compared to 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 womens' health throughout their lifecourse.

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 cardio-protective behaviors from higher SES parents may 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 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 boys.

Because a previous investigation of CARDIA data reported that black women showed the highest incidence of hypertension over 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 may 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 an average age of 36 years independent of concurrent adult BMI (33).

A limitation of the present study is that only 85% 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). Though 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 four years (i.e., high school graduate vs. 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 mmHg 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.

Acknowledgments

Glossary

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

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Figure 1.
Modifying 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 addition of the sex-X-mother's education and sex-X-mother's education-X-time interaction terms) by CARDIA exam year. Filled squares represent men and open circles represent women.
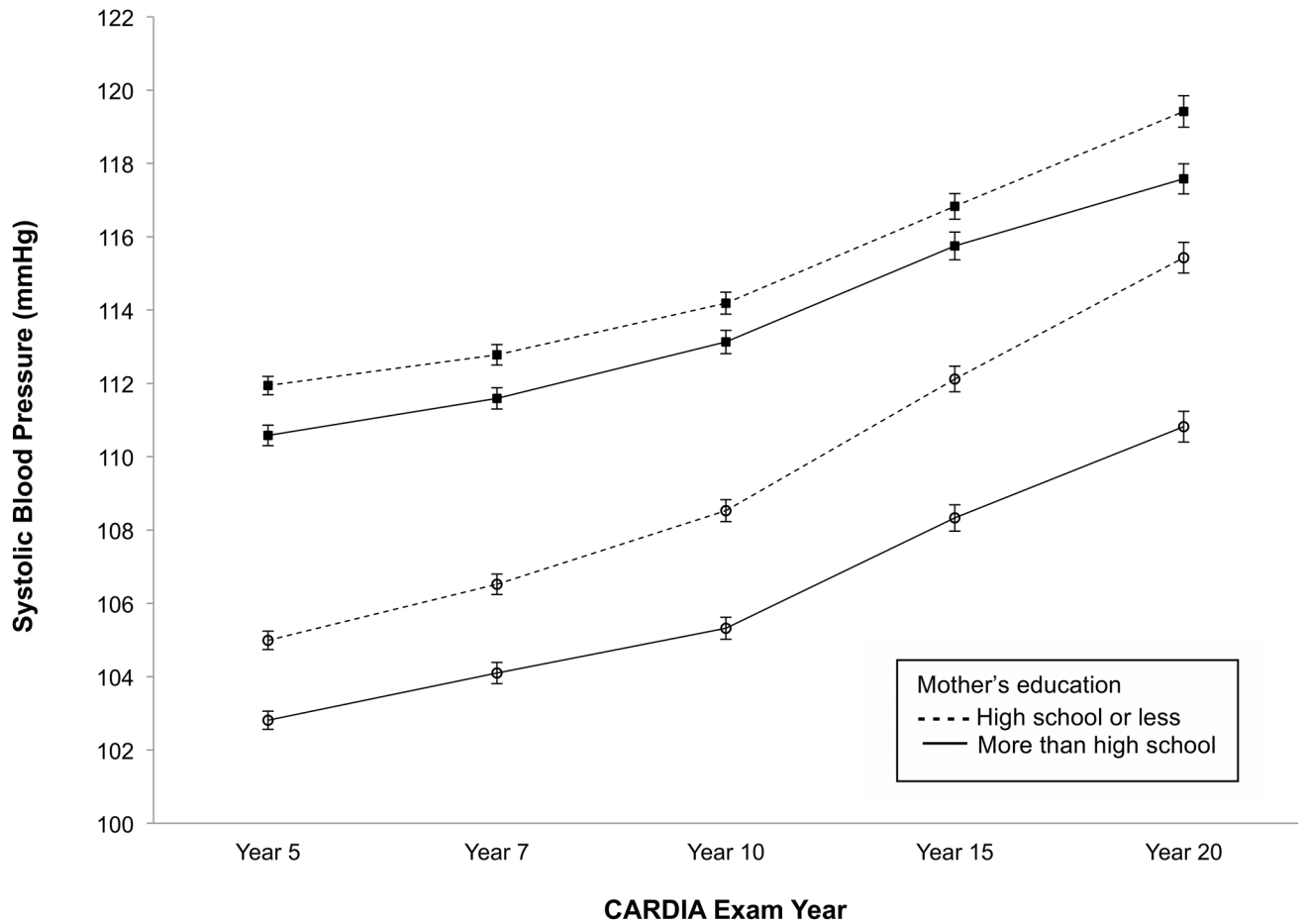
Figure 2.
Modifying effect of sex on the association of mother's education with 15-year change in adult DBP. Data points represent predicted DBP values obtained from Model 3 (with addition of the sex-X-mother's education and sex-X-mother's education-X-time interaction terms) by CARDIA exam year. Filled squares represent men and open circles represent women.
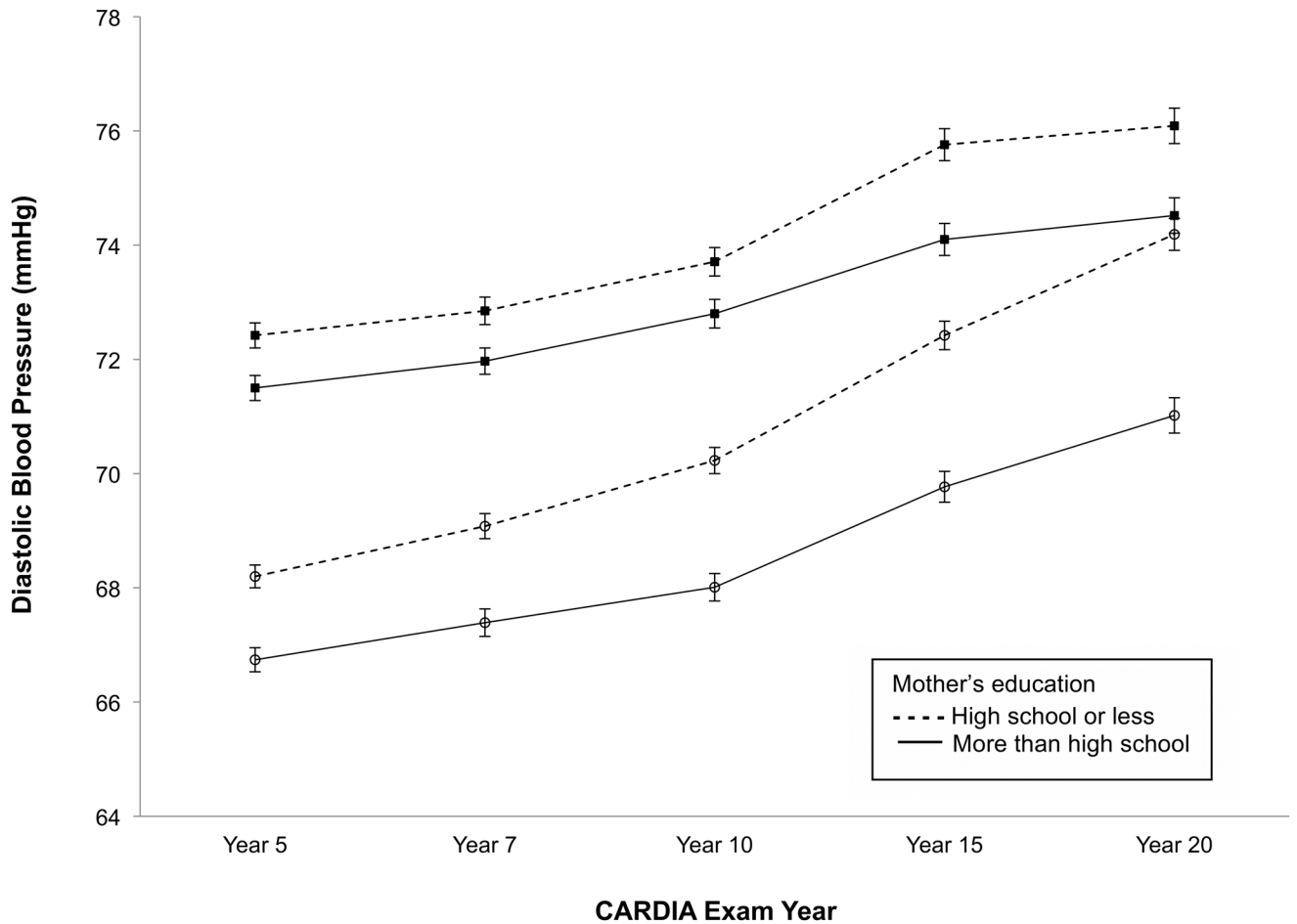
TABLE 1

Participant characteristics at baseline (CARDIA Year 5) separately by sex[1]

	Men (*n*=1838)	Women (*n*=2239)	$P_{\text{sex diff}}$
Adult predictors			
Age (yrs)	29.94 (3.56)	29.98 (3.68)	0.70
SBP (mmHg)	111.85 (11.13)	104.40 (10.76)	0.001
DBP (mmHg)	71.75 (9.60)	67.25 (9.66)	0.001
Own (adult) education (yrs)	14.0 (12.0, 16.0)	15.0 (13.0, 16.0)	0.40
% African American	44.9 (826)	48.7 (1091)	0.02
% married	40.0 (736)	41.0 (918)	0.63
BMI (kg/m^2)	25.1 (22.9, 28.1)	24.2 (21.5, 29.0)	0.42
Total cholesterol (mg/dL)	175.4 (154.0, 200.2)	173.8 (153.4, 194.8)	0.003
% taking antihypertensives	1.3 (24)	1.8 (40)	0.22
% using OC/HRT (women)		27.2 (610)	
Childhood predictors			
Mother's education (yrs)	13.17 (2.70)	12.89 (2.85)	0.002
Father's education (yrs)	13.25 (3.56)	13.07 (3.72)	0.16

[1] values expressed as mean (SD), median (IQR), or % (*n*)

Mother's education, men, *n*=1798; women, *n*=2206.

Father's education: men, *n*=1646; women, *n*=1931.

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	−.68	(.07)***	−.36	(.07)***			.02
Effect on slope	−.03	(.01)***	−.02	(.01)*			.11
Father's education							
Effect on intercept	−.41	(.05)***	−.13	(.05)**	−.08	(.05)	.53
Effect on slope	−.02	(.004)***	−.005	(.005)	−.002	(.005)	.02
Mother's education							
Effect on intercept	−.33	(.06)***	−.16	(.06)**	−.09	(.06)	.50
Effect on slope	−.02	(.01)***	−.006	(.01)	−.003	(.01)	.001

*
$P<.05$.

**
$P<.01$.

***
$P<.001$.

Model 1 includes terms for time, education, and time-x-education.

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-x-own adult education.

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	−.27	(.06)***	−.13	(.06)*			.004
Effect on slope	−.03	(.01)***	−.02	(.01)**			.15
Father's education							
Effect on intercept	−.30	(.04)***	−.06	(.04)	−.03	(.04)	.33
Effect on slope	−.01	(.004)***	−.005	(.004)	−.003	(.004)	.51
Mother's education							
Effect on intercept	−.24	(.05)***	−.06	(.05)	−.03	(.05)	.67
Effect on slope	−.02	(.004)***	−.01	(.005)	−.005	(.005)	.01

*
P<.05.

**
P<.01.

***
P<.001.

Model 1 includes terms for time, education, and time-x-education.

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-x-own adult education.

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	−.25	(.10)*	−.48	(.10)***	−.11	(.10)	−.16	(.08)*
Effect on slope	−.007	(.01)	−.02	(.01)*	−.007	(.01)	−.02	(.01)**
Father's education								
Effect on intercept	−.11	(.08)	−.05	(.07)				
Effect on slope	.004	(.01)	−.005	(.01)				
Mother's education								
Effect on intercept	−.12	(.09)	−.07	(.08)	−.06	(.08)	−.03	(.07)
Effect on slope	.02	(.01)†	−.02	(.01)*	.005	(.01)	−.01	(.01)*

*
$P<.05$.

**
$P<.01$.

***
$P<.001$.

†
$P<.10$.

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 x time.