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Parental Education is Related to C-Reactive Protein among Female Middle Aged Community Volunteers

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Abstract

Growing evidence suggests that socioeconomic attributes of both childhood and adulthood confer risk for cardiovascular morbidity and mortality. In this study, we examine the association of both parental and individual educational attainment with C-reactive protein (CRP), an inflammatory mediator relevant to cardiovascular pathophysiology, in a midlife community sample. Subjects were 811 men and women (394 men/417 women; 87 % European-American/13 % African American), 30–54 years of age. Plasma concentrations of CRP were determined from blood samples obtained at a single session following an overnight fast. Regression analyses adjusting for age and race showed both parental education and individual education to be associated inversely with CRP in women, but not men. The relationship of parental education with CRP in women persisted on multivariable adjustment for both lifestyle risk factors (smoking, alcohol consumption, sleep, exercise, body mass index) and individual SES. Independent of reported personal educational attainment, mid-life adult women whose parents achieved fewer years of educational attainment exhibit higher levels of circulating CRP than women with higher parental education. This association may help explain the increased risk of atherosclerotic cardiovascular morbidity and mortality conferred by low childhood socioeconomic status.

Keywords

C-Reactive Protein; Parental Education

Introduction

Variation in socioeconomic status (SES) is inversely related to cardiovascular mortality (Salonen, 1982;Seigel et al., 1987; Keil et al., 1992), incident coronary heart disease (Rose and Marmot, 1981; Liu et al., 1982; Diez-Roux et al.,1995), and cardiovascular disease risk factors, including cigarette smoking (Zang and Wynder, 1998), generalized obesity (Sobal and Stunkard, 1989), and physical activity (Evenson et al., 2002). In general, these associations hold for both men and women, though the strength of the relationships may vary between sexes. Among women, stronger inverse associations between SES and incident coronary heart disease (Vogels et al., 1999; Thurston et al., 2005), coronary mortality (Chandola, 1998; Pekkanen et al., 1995), and obesity (Sobal and Stunkard, 1989; Langenberg et al. 2003; Zhang and Wang, 2004) have been documented in some, but not all (Marmot et al, 1997; Frank et al., 2003; Diez-Roux et al., 1995) investigations.

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Although most literature has focused primarily on adult SES, growing evidence indicates that lower childhood SES, independently of adult social standing, is associated with increased risk of incident coronary heart disease (CHD) outcomes, including myocardial infarction (Notkola et al., 1985; Kaplan and Salonen, 1990; Gliksman et al., 1995; Wannamathee et al., 1996; Wamala et al., 2001; Davey Smith et al., 2002;Lawlor et al., 2004), and several CHD risk factors (Poulton et al., 2002; Blane et al., 1996; Ebrahim et al., 2004). Here, also, sex differences have been documented, with childhood SES predicting serum triglycerides (Parker et al., 2004), HDL cholesterol (Brunner et al., 1999), and metabolic syndrome (Lehman et al., 2005) in women, but not men.

Inflammation is now recognized as a risk factor for the development and exacerbation of atherosclerotic cardiovascular disease (Ross, 1999). C-reactive protein (CRP) is a frequently cited inflammatory marker shown to predict future cardiovascular morbidity and mortality (Danesh et al., 2000; Danesh et al., 2004; Lagrand et al., 1999). Growing evidence also suggests that adult SES, whether indicated by occupation (Owen et al., 2003), employment status (employed/unemployed; Danesh et al, 1999), education (Panagiotakos et al., 2004; Wu et al., 2002); composites of income and education (Jousilahti et al., 2003), or community-level SES (Petersen et al., 2008) is inversely related to CRP.

Extending the literature on SES associations with inflammation, more recent attention has turned to the role of childhood influences on inflammatory processes in adulthood. In this regard, it is suggested that the early-childhood environment can regulate immune development in ways that influence health in adulthood, even when adult SES is taken into account (e.g., Lynch, Kaplan, & Salonen, 1997). Thus, early life variation in exposure to environmental factors that covary inversely with SES and modulate immune function, such as air pollutants, viral infections, maternal smoking, psychological stress and allergen exposure (Donovan and Finn, 1999) may have far-reaching effects. Childhood SES may also affect adult inflammation through more proximal behavioral mechanisms. For example, lower SES families engage in poorer health practices, such as smoking, physical inactivity, poorer dietary choices (Marmot et al., 1991), that have been associated with higher systemic inflammation (Bruunsgaard, 2005; Frohlich et al., 2003). Lifestyle practices are often established early in life, with lower childhood SES predicting poorer health practices in adulthood (Politt et al., 2005). Thus, in examining possible associations between childhood SES and adult inflammation, it is important to determine whether contemporaneous factors such as adult lifestyle choices or remaining in one's social class of childhood explain observed effects or whether there is something unique about childhood SES that influences later inflammatory processes.

To date, findings from the few studies examining childhood SES in relation to adult CRP levels have been mixed. One study showed a positive association between father's occupation and CRP in men 45 to 59 years of age (Mendall et al, 2000), and similar findings obtained for a composite of father's occupation and education in younger adults (Pollitt et al., 2007). A third investigation failed to find parental education associated with CRP (Gimeno et al., 2008), whereas a fourth (Taylor et al., 2006) found that parental education was indirectly associated with adult CRP levels through a pathway encompassing a history of familial adversity, later psychological functioning, and body mass. The model tested in that study did not adjust for adult subjects' own educational attainments, however, and did not specifically address potential sex differences in the magnitude of association between early life SES and CRP. The purpose of the present study, then, was to further explore the relationship of parental education (as one frequently studied component of childhood SES) with CRP concentrations, adjusting for participants' own education, among a diverse community sample of mid-life men and women, and to examine whether these relationships might vary by sex.

Methods

Participants

This investigation was based on data derived from 1047 adults (48% male; 17% African-American) who participated in the University of Pittsburgh Adult Health and Behavior (AHAB) project between 2001 and 2005. The AHAB registry is a compendium of behavioral and biological measurements on mid-life community volunteers who were recruited, via mass-mail solicitation, from Southwestern Pennsylvania (principally Allegheny County). Exclusions from AHAB participation included age <30 or >54 years; a reported history of atherosclerotic cardiovascular disease, chronic kidney or liver disease, cancer treatment in the preceding year, and major neurological disorders, schizophrenia or other psychotic illness. Other exclusions included pregnancy and the use of insulin, glucocorticoid, antiarrhythmic, psychotropic, or prescription weight-loss medications. Data collection occurred over multiple laboratory sessions, and informed consent was obtained in accordance with approved protocol and guidelines of the University of Pittsburgh Institutional Review Board.

Because AHAB exclusions did not include common acute illnesses, such as recent colds or allergies, data of participants having circulating CRP levels >10 mg/L were not included in the current analyses (Pearson et al., 2003). Overall, 185 subjects were excluded due to elevated CRP. In order to examine the effect of both fathers' and mothers' education on CRP in later life, analyses were limited to two-parent households, which resulted in the exclusion of 78 additional subjects. This yielded a final sample of 811 European-American (EA) and African American (AA) individuals, a sample which included 394 men (37 AA) and 417 women (67 AA). These subjects did not differ in age or sex from AHAB participants who were excluded from the present analyses due to elevated inflammatory markers or childhood household status.

Procedure

Prior to arrival at the laboratory, participants were asked to fast for 8 hours and avoid exercise for 12 hours, alcohol for 24 hours, and nicotine for 1 hour. All sessions were scheduled in the morning. On arrival to the laboratory, the project nurse completed a medical history and medication use interview, obtained measurements of height and weight for the determination of body mass index (BMI; kg/m^2), and drew a 40 cc blood sample in citrate treated tubes. A portion of the blood sample was spun down and plasma collected and stored at −80°C until batched analysis of CRP levels.

Measures

Education: ***Individual Education:*** Participant's were asked to report their years of education and their highest level of academic attainment. Individual education variables were distributed normally. Subjects were well-educated on average, with a mean of 15.6 (SD=2.6) years of schooling. Nonetheless, levels of educational attainment varied appreciably among study participants, with 0.5% lacking a high school diploma, 17.5% having completed high school or technical training only, 23% with some college, 36% with a Bachelors degree, 17.5% with Masters degree, and 5.5% with PhD or doctoral-level professional degree.

Parental Education: Participants reported both their mothers' [M (SD) = 12.4 (2.5)] and fathers' [M (SD) = 12.7 (3.3)] years of education completed by the time the participant was age 18 (range: 1 – 24 years). Consistent with other studies (Karlamanga et al., 2005; Taylor et al., 2006), analyses were performed examining the higher of the parents' education in order to reflect the maximal health-relevant socioeconomic benefits afforded to a particular family. Highest household parental education ranged from 3 – 24 years [M (SD) = 13.5 (2.9)]. Parental education variables were normally distributed. In order to maintain consistent between-family

comparisons, analyses were limited to two-parent households to avoid the confounding effect of parental absence and to allow for separate analyses of mothers' and fathers' education.

CRP: CRP was measured using the BNII nephelometer from Dade Behring (Deerfield, Illinois) utilizing a particle enhanced immunonephelometric assay. In this procedure, polystyrene particles are coated with monoclonal antibodies to CRP, which, in the presence of antigen agglutinate, cause an increase in the intensity of scattered light. The increase in scattered light is proportional to the amount of CRP in the sample (Ledue et al., 1998). The assay range is 0.175–1100 mg/L. Intra-assay CVs range from 2.3–4.4% and inter-assay CVs range from 2.1–5.7%. Final CRP values were normalized by reciprocal transformation.

Additional Variables: A number of variables were assessed that might explain associations between SES and inflammation. These variables included age, sex, race, BMI, smoking status, alcohol use, sleep volume, and physical activity. Smoking status was defined by participants' self-report as being a current cigarette smoker versus all other categories of tobacco use (ex-smoker, non-smoker, other forms of tobacco use). Alcohol use was expressed as drinks/week and estimated from subject's report of the number of alcoholic drinks consumed over the previous four weeks. Sleep volume was calculated from participant's reported average hours of sleep during the 7 nights prior to study participation (hours of sleep = (average hours/week night × 5) + (average hours/weekend night × 2)). Physical activity was measured using the Paffenbarger Physical Activity Questionnaire, which estimates kilocalories expended per week (Paffenbarger et al., 1993). The distributions of physical activity scores and number of alcoholic drinks/week were normalized by square root and natural log transformations, respectively.

Statistical Analysis

Associations of education with circulating CRP were evaluated in three linear regression models. In Models IA and IB, demographic covariates (age, sex, and race) were entered on Step 1, individual (i.e., participants' own) education (IA) or parental education (IB) was entered in Step 2, and an interaction term for sex was entered in Step 3. Models IIA and IIB consisted of sex-specific analyses for circulating CRP, with covariates (age, race) on Step 1, and individual education (IIA) or parental education (IIB) entered on Step 2. In Model III, we examined whether parents' educational attainment predicted circulating CRP independent of individual education. Additionally, Model IV examined whether parental education was associated with CRP independently of BMI and health practices. Here, demographic characteristics were entered in Step 1, BMI, smoking, exercise, alcohol, and sleep in Step 2, individual education in Step 3, and parental education in Step 4 of the regression equation.

Results

Demographic and health behavior characteristics for the sample are listed in Table 1. Women drank significantly less alcohol than men, smoked less, and had lower BMI, while men engaged in more physical activity. Women had slightly lower mean years of education than men, but highest parental education did not differ by sex. Bivariate correlations describing the associations of subject characteristics with educational variables and with CRP levels are presented in Table 2. For convenience, the signs of correlations involving reciprocally transformed measurements of CRP are reversed (as in all subsequent test statistics), so that positive (and negative) coefficients are interpreted as such. Consistent with prior literature, higher CRP was associated with African-American race, higher BMI, less physical activity, and current smoking (Kasapis and Thompson, 2005;Khera et al., 2005;Mendall et al., 1996). CRP did not covary with age, sex, alcohol use, or sleep duration. Circulating levels of CRP covaried inversely with both parental and individual educational attainment.

In Models IA and IB (not shown in tables), covariates (age, sex, and race) accounted for a significant proportion of the variance in CRP levels ($\Delta R^2 = 0.011$, $F_{3, 807} = 2.87$, p= 0.036). Both individual education years ($\beta = -0.046$, $\Delta R^2 = 0.002$, $F_{4, 806} = 2.56$, p= 0.037) and highest household parental years of education ($\beta = -0.085$, $\Delta R^2 = 0.007$, $F_{4, 806} = 3.59$, p= 0.007) accounted for significant additional variance in CRP and were both found to interact significantly with sex (individual education: $\beta = -0.47$, $\Delta R^2 = 0.005$, $F_{4, 805} = 2.89$, p= 0.042; parental education: $\beta = -0.174$, $\Delta R^2 = 0.009$, $F_{4, 805} = 3.54$, p= 0.007). Sex-specific analyses were then performed (Model II) to determine whether the variation in CRP accounted for by individual and parental education differed across women and men; these analyses are reported in Table 3. Higher individual education ($\beta = -0.102$, $\Delta R^2 = .010$, $F_{3,413} = 4.67$, $p < .04$) and parental education ($\beta = -0.153$, $\Delta R^2 = .023$, $F_{3,413} = 6.49$, $p < .01$) were significant predictors of CRP among women, but no significant effects were seen in men (Table 3). In Table 4, an independent effect of women's parental education was demonstrated for CRP beyond that of individual education ($\beta = -0.133$, $\Delta R^2 = .015$, $F_{4,412} = 5.18$, $p < .01$), with individual education no longer accounting for a significant portion of the variance in CRP ($\beta = -0.057$, $\Delta R^2 = .010$, $F_{3,413} = 4.67$, $p < .27$). Thus, women in our study whose parents had fewer years of education showed higher plasma concentrations of CRP than those with higher parental educational attainment, an effect that was independent of women's own educational attainment, age, and race. Separate analyses of mothers' and fathers' education, controlling for age, race, and individual education, revealed a similar pattern of results. Both mothers' and fathers' educational attainment predicted adult CRP levels in women (mother: $\beta = -0.144$, $\Delta R^2 = .018$, $F_{4,412} = 5.48$, $p < .01$; father: $\beta = -0.116$, $\Delta R^2 = .012$, $F_{4,412} = 4.82$, $p < .03$), but not in men (data not shown in tables).

An additional analysis was then performed to determine whether lifestyle factors could account for the association of parental education with CRP in women. Parental education was entered as a predictor after demographic covariates, BMI, health practices (smoking, physical activity, alcohol use, and sleep duration), and individual education (Model IV; Table 5). After controlling for age and race, greater BMI (B = .492, $p < .01$) predicted higher CRP levels independently of demographic characteristics. Interestingly, individual SES was no longer associated with CRP when entered into the model after demographic covariates, BMI and health behaviors (B=−.053, $p > .10$). Parental education, on the other hand, remained a significant, independent predictor of CRP (B = −.084, $p < .05$) with age, race, BMI, smoking status, alcohol use, sleep volume, and physical activity in the model.

To examine the possibility that the parental education associations reported here might have emerged only in the context of the data transformation required to satisfy parametric assumptions of normality, box-plots of *untransformed* CRP measurements are reported separately for men and women in Figure 1. The values depicted here are partitioned by quartiles of the distribution of subjects' parental education (adjusted for correlated variation in age and race). Median values among women comprising the lowest quartile of parental education were greater than those of the highest quartile, and non-parametric statistic (Mann-Whitney U) corroborated end-quartile differences ($Z = -2.8$, $p < 0.006$). The Spearman rank-order correlations of parental education with untransformed CRP were likewise significant in women ($rho = -0.189$, $p < 0.001$).

Discussion

The present study shows indices of childhood and adult socioeconomic standing to be associated inversely with a circulating marker of inflammation in a relatively healthy, mid-life community sample. Substantial evidence documents that childhood socioeconomic attributes confer risk for coronary heart disease outcomes (Kaplan and Salonen, 1990; Gliksman et al., 1995; Wannamathee et al.,1996; Wamala et al., 2001; Davey Smith et al., 2002;Lawlor et al.,

2004) and risk factors (Blane et al., 1996; Ebrahim et al., 2004; Poulton et al., 2002). Consistent with these findings, our study shows that, when compared with women whose parents were more educated, women with lower parental education have higher levels of circulating CRP, an independent risk factor for cardiovascular events (Danesh et al., 2000; Danesh et al., 2004; Lagrand et al., 1999; Ridker et al., 1997; Thompson et al., 1995). This association was not seen for men in this sample. Additionally, associations between women's parental education and CRP were independent of age, race, and individual-level SES. Further, participants with lower parental education had higher CRP even after accounting for lifestyle-related risk factors, including BMI, smoking, alcohol consumption, physical activity and sleep. The current results are consistent with evidence that levels of CRP covary inversely with childhood SES (Gimeno et al., 2008; Mendall et al., 2000; Pollitt et al., 2007), though this is the first study to report sex-specific associations. These findings raise the possibility that relationships between childhood SES and increased vulnerability to cardiovascular disease could be mediated, in part, through inflammatory pathways, and that these associations may differ by gender.

The mechanisms by which socioeconomic attributes of childhood affect systemic inflammation are unclear. Childhood socioeconomic conditions are associated with behavioral factors such as smoking, diet, and physical activity (Brunner et al., 1996; Lynch et al., 1997; Winkleby et al., 1992), lifestyle factors which are associated with inflammatory processes (Bruunsgaard, 2005; Frohlich et al., 2003). In our study, however, childhood SES continued to predict CRP levels after controlling for several health practices. Lower parental education may reflect material conditions associated with chronic stress, which may play a role in the etiology of inflammation through endocrine pathways involving the hypothalamic-pituitary adrenal axis or activation of the sympathetic nervous system (Maier and Watkins, 1998; Segerstrom and Miller, 2004). Childhood SES may also contribute to adult health through poor maternal socioeconomic circumstances that may result in dysregulation of a range of metabolic and endocrine systems during fetal growth, as well as material deprivation of the child in early life (Osmond et al., 1993).

Regardless of the mechanisms, our findings were consistent with several studies documenting gender differences in the effects of childhood socioeconomic circumstances on adult health. Although not entirely consistent (Langenberg et al., 2003), substantial evidence demonstrates stronger effects of childhood social class on adult cardiovascular risk factors in women than in men, as seen in relation to smoking (Power et al., 2005), diabetes (Maty et al., 2008), depression (Sadowski et al. 1999), and composite risk profiles (Karlamanga et al., 2005). Our results support other findings that childhood social status influences health outcomes in adulthood, independent of adult socioeconomic indicators, and that these effects may differ by gender. Though this is the first study to report sex-specific associations between parental education and CRP, others have also found levels of CRP to covary inversely with childhood SES (Mendall et al., 2000; Pollitt et al., 2007, Taylor et al., 2006). In any case, the lack of a relationship between childhood SES and CRP in men in our sample warrants further exploration.

An examination of possible mechanisms linking parental education and adult CRP may shed some light on the observed sex difference in our findings. There are numerous ways in which parental education might affect cardiovascular risk factors in later life, and one such mechanism may be the sustained influence of early socioeconomic circumstances on health behaviors. Lower education may predict poor dietary habits and less physical activity, two factors that are related to CRP. It is possible that childhood SES is related to body mass and physical activity differently in males versus females. Kinra et al. (2000) showed that the relationship between socioeconomic "deprivation" and obesity was stronger in girls versus boys, and that this relationship grew stronger with age in girls only. Gender differences in physical activity

are well-established, with boys generally being more active than girls (Sallis et al., 2000; Inchley et al., 2005). In their study of Scottish adolescents, Inchley et al. (2005) found that girls from the highest SES groups were less active than boys from the lowest SES groups, suggesting an additive effect of gender and SES that places girls from low SES backgrounds at particular risk of low physical activity. Although our analyses demonstrated that parental education continued to predict CRP in women, but not men, after controlling for both BMI and physical activity, it is possible that further investigation of sex differences in health behaviors related to parental education may advance understanding of this relationship. Another hypothesized link between childhood SES and adult health is that of psychosocial influences, with lower parental education indicating a more "risky" family environment that might lead to adverse alterations in biological systems. Social, behavioral and biological stimuli perceived as "stressful" may play a role in inflammatory processes, as psychosocial stress has been associated with factors related to CRP, including abdominal fat (Lapidus et al., 1989; Wing et al., 1991; Raikkonen et al., 1994) and insulin resistance (Raikkonen et al., 1994; Black, 2003). Perhaps men in our sample were less influenced by risky childhood environmental circumstances than were women, decreasing the association of childhood environment and later inflammatory processes. To our knowledge, however, no data exists which demonstrates sex differences in the psychological effects of lower parental education. Finally, parental education may contribute to adult health through pathways that do not involve behavioral or psychosocial factors. Several studies have suggested that cardiovascular and metabolic risk are related inversely to birth weight (Reynolds et al., 2005), and the association between size at birth and mortality in adult life may be due to socioeconomic factors (Leon et al., 1996). Reduced size at birth may be a marker for poor maternal socioeconomic circumstances that may result in both dysregulation of developing biological systems and inadequate medical or nutritional resources for the child (Osmond et al., 1993). Although research examining sex differences with regard to these associations in humans has been mixed (Phillips et al., 2000; Reynolds et al., 2005), some animal studies have found female rats to be more sensitive than males to activation of the HPA axis (Weinstock et al., 1992), suggesting that relationships between birth size and cardiovascular risk may not be the same in men and women. In terms of the sex differences found in the present study, perhaps these perinatal complications have more serious and long-lasting biological effects on daughters than sons.

In addition to examining childhood SES, the current study also contributes to an understanding of relationships between individual SES and an inflammatory mediator. Consistent with previous findings (Panagiotakos et al., 2004; Wu et al., 2002), individuals' educational attainment was associated with levels of CRP, though only in women. We found no significant relationship between individual education and levels of CRP in men after adjusting for demographic characteristics. To our knowledge, no other studies have reported sex-specific associations of education and CRP though our results support previous findings showing stronger relationships of educational level and coronary risk in women (Loucks et al., 2007; Thurston et al., 2005) as compared to men.

These findings should be interpreted in the light of several study limitations. First, the cross-sectional design of this study precludes causal interpretation. Alternative explanations for our results include the possibility that CRP and education are independently related to a third factor such as personality, cognitive ability, or general health. Another limitation is the single assessment of CRP. Although evidence suggests that levels of this inflammatory mediator are relatively stable over extended periods (Rao et al., 1994), multiple assessments over time may provide a more reliable indicator of chronic interindividual variability. Future investigations should include longitudinal assessments, beginning in early life, to evaluate the influence of more proximal socioeconomic characteristics, such as chronic stress and access to health-promoting resources, on inflammatory mediators to better elucidate how education may affect individuals' health. Additionally, the retrospective nature of reported childhood conditions

may involve recall bias, although this is fairly unlikely using educational variables. Although education is a widely used index of SES, and is related to income and occupation, these indices do not fully overlap (Krieger et al., 1997). The use of education as a sole index of both childhood and current SES may have neglected other important dimensions of SES. Also, the range of educational attainment was somewhat limited in comparison with other studies, as the sample as a whole averaged over 15 years of schooling. It is possible that a clearer relationship of individual SES with inflammatory mediators would be seen across a broader spectrum of socioeconomic variation, including a greater representation of individuals from the lowest strata of socioeconomic position.

Nonetheless, our findings provide evidence that both parental and individual education are associated, in a sex-specific manner, with a marker of inflammation thought to play a role in the pathogenesis of cardiovascular and other inflammatory diseases. Furthermore, the relationship of childhood social standing with adulthood CRP is independent of demographic characteristics, measured health practices, and individual educational attainment. Thus, inflammatory processes may mediate independent relationships between childhood SES and vulnerability to cardiovascular disease, particularly in women. Further investigation of this potential pathway is warranted.

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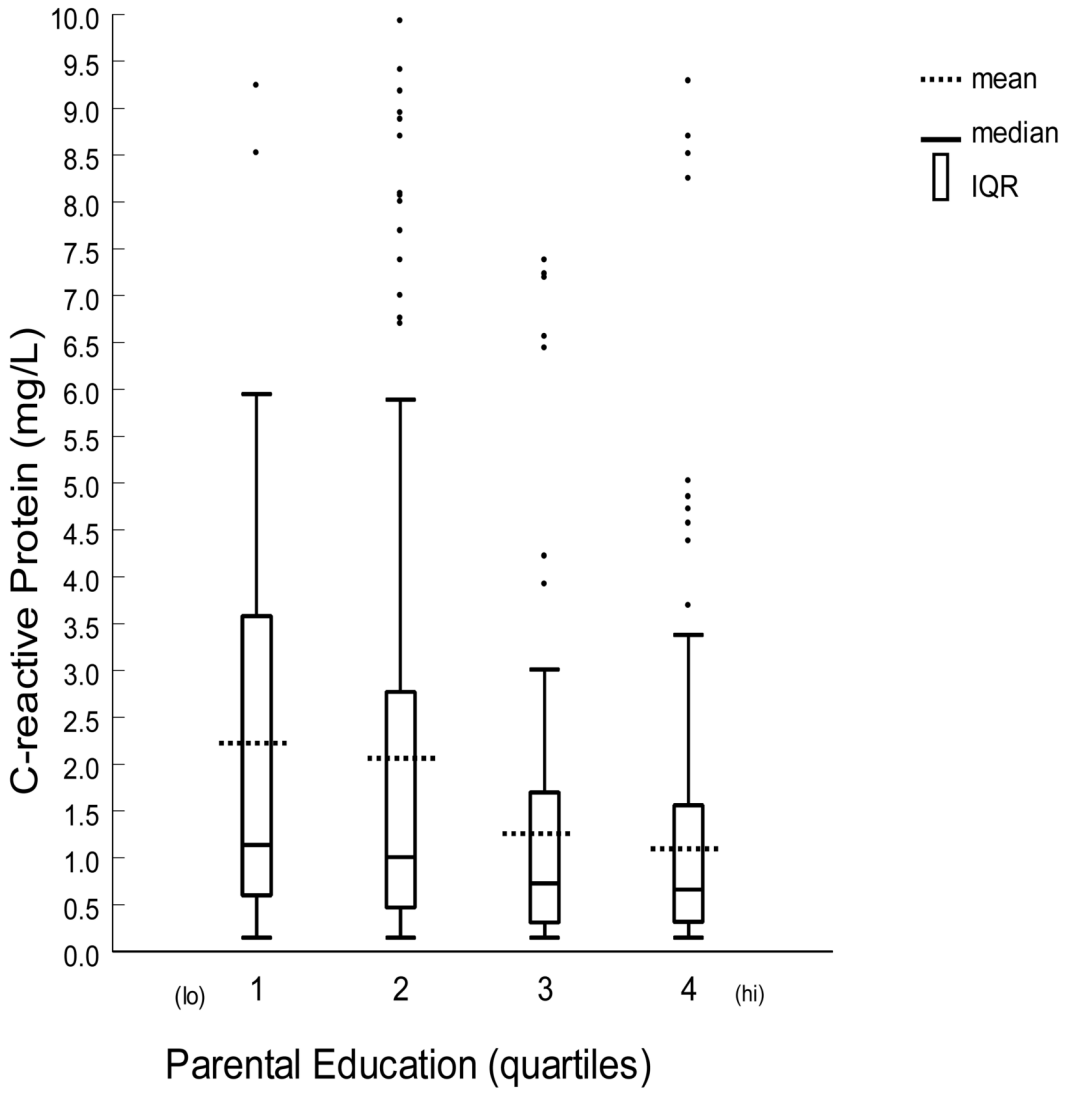

Figure 1. Distributions of untransformed values of CRP for quartiles of parental education for women (n=417)

Parental Education Years by quartile: 1 = No high school diploma, 2 = H.S. Graduate, 3 = Some college 4 = College graduate or higher

Table 1

Demographic characteristics of sample and for male and female participants separately.

Characteristic	Total Sample n = 811 M (SD)	Men n = 394 M (SD)	Women n = 417 M (SD)	Test Statistic
Age (years)	44.8 (6.7)	44.6 (7.0)	44.9 (6.6)	$F_{1,810} = 0.49$
Race (% black)	13%	9.5%	16%	$\chi^2=8.83^{**}$
BMI (kg/m^2)	27.3 (5.4)	27.8 (4.7)	26.4 (5.9)	$F_{1,810} = 23.4^{**}$
Smoking (% current)	16%	18%	13%	$\chi^2=5.8^{*}$
Exercise (kilocals)	2415 (1777)	2577(1773)	2287 (1769)	$F_{1,810} = 13.1^{**}$
Alcohol (drinks/week)	3.98 (7.5)	5.91 (9.4)	2.20 (4.4)	$F_{1, 810} = 59.1^{**}$
Sleep (hours/week)	47.9 (7.0)	47.3 (6.9)	48.8 (6.9)	$F_{1, 810} = 12.3^{**}$
CRP	1.65 (1.9)	1.49 (1.7)	1.79 (2.1)	$F_{1, 810} = 0.06$
Individual Ed (yrs)	15.6 (2.6)	15.9 (2.6)	15.4 (2.6)	$F_{1, 810} = 4.54^{*}$
Parental Ed (yrs)	13.5 (2.9)	13.5 (2.9)	13.4 (2.9)	$F_{1, 810} = 0.15$

**P<.01,

*P<.05,

$^{+}$P<.10

Race coded 0 = White, 1 = Black. BMI = Body Mass Index; Smoking Status (0=not current, 1=current).

Mean comparisons conducted on the transformed CRP, alcohol, and exercise variables.

Table 2

Bivariate correlations with covariates, CRP, and Parental and Individual Education (n = 811).

Characteristic	CRP r	Parental Ed. r	Individual Ed. r
Age	−.009	− .197**	− .060
Gender	.025†	−.018†	−.060+†
Race	.099**†	−.118**†	−.188**†
BMI (kg/m^2)	.458**	−.065*	−.071*
Smoking	.070*†	−.076*†	−.172**†
Exercise (kilocalories)	−.122**	.084*	.112**
Alcohol (drinks/week)	−.032	− .010	− .081
Sleep (hours/week)	−.042	.044	.050
Individual Ed (yrs)	−.077*	.334**	---
Parental Ed (yrs)	−.099**	---	---

** P<.01,

* P<.05,

+ P<.10,

† = point biserial correlation.

Race coded 0 = White, 1 = Black. BMI = body mass index; Smoking Status (0=not current, 1=current).

Correlations conducted on transformed CRP, alcohol, and exercise variables, but signs were reversed in the table for ease of interpretation.

Table 3

Hierarchical linear regressions showing the contributions of age, race, and individual and parental education to the prediction of reciprocally transformed CRP in men and women. Signs have been reversed for ease of interpretation.

Model IIA					
Males (394)	***β***	**b (SE)**	**Females (417)**	***β***	**b (SE)**
Step 1:			**Step 1:**		
Age	.017	.001 (.002)	Age	−.027	−.001 (.002)
Race	.033	.023 (.035)	Race	.135	.086 (.031)
Step 2:			**Step 2:**		
Individual Ed (yrs)	.021	.002 (.004)	Individual Ed (yrs)	−.102	−.009 (.004)
Model IIB					
Step 2:			**Step 2:**		
Parental Ed (yrs)	.004	.000 (.004)	Parental Ed (yrs)	−.153	−.012 (.004)

Table 4

Hierarchical linear regression showing the contributions parental education, adjusted for individual education, to the prediction of reciprocally transformed CRP in women. Signs have been reversed for ease of interpretation.

Model III		
Females (417)	***β***	**b (SE)**
Step 1: See Table 3		
Step 2:		
Individual Ed (yrs)	−.057	−.005 (.005)
Step 3:		
Parental Ed (yrs)	−.133	−.011 (.004)

Table 5

Contribution of standard covariates, health behaviors, and individual and parental education to the prediction of reciprocally transformed CRP in women. Signs have been reversed for ease of interpretation.

Model IV		
Females (417)	***β***	**b (SE)**
Step 1:		
Age	−.047	−.002 (.002)
Race	.018	.012 (.028)
Step 2:		
BMI	.523	.021 (.002)
Alcohol	−.036	−.007 (.008)
Smoking	.041	.029 (.031)
Sleep	.057	.002 (.001)
Exercise	−.012	−.003 (.012)
Step 3:		
Individual Ed (yrs)	−.009	−.001 (.004)
Step 4:		
Parental Ed (yrs)	−.090	−.007 (.004)