Association of Childhood Socioeconomic Status With Subsequent Coronary Heart Disease in Physicians

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**Background:** Adult socioeconomic status (SES) is an independent risk factor for the development of coronary heart disease (CHD), but whether low childhood SES has an effect in adults who have achieved high SES is unknown.

**Methods:** We examined the risk of CHD and mortality associated with low childhood SES in 1131 male medical students from The Johns Hopkins Precursors Study, a prospective cohort of graduates of The Johns Hopkins University School of Medicine from 1948 to 1964 with a median follow-up of 40 years.

**Results:** Of 1131 subjects, 216 (19.1%) were from low-SES families. Medical students from low-SES families were slightly older at graduation (26.8 vs 26.2 years; \( P = .004 \)) and gained more weight over time (\( P = .01 \)). Low childhood SES conferred a 2.40-fold increased hazard of developing CHD on or before age 50 years (95% confidence interval, 1.21-4.74) but not at older ages. The impact of low SES on early CHD was not reduced by adjusting for other CHD risk factors, including body mass index, cholesterol level, amount of exercise, depression, coffee drinking, smoking, hypertension, diabetes mellitus, and parental CHD history. Low childhood SES did not confer an increased risk of all-cause mortality.

**Conclusions:** Low childhood SES is associated with an increased incidence of CHD before age 50 years among men with high adulthood SES. This risk is not mediated by traditional risk factors for CHD. These findings highlight the importance of childhood events on the development of CHD early in adulthood and the persistent effects of low SES.

Arch Intern Med. 2006;166:2356-2361
A longitudinal cohort study of 1337 former medical students at The Johns Hopkins University School of Medicine in the classes of 1948 to 1964. Excluded from this analysis were the small number of women (n=121), men of non-European ancestry (n=36), those who did not answer the questions about parental occupation (n=56), and those who were unavailable for follow-up (n=13), leaving 1131 white male medical students for analysis of incident CHD (some individuals met >1 exclusion criterion).

DATA COLLECTION

At baseline during medical school, each participant provided a detailed medical history and underwent physical examination, including measurement of blood pressure, height, and weight. After medical school, follow-up data were collected using annual mailed questionnaires. In general, yearly response rates exceeded 70% and, during any 5-year interval, at least 85% of participants responded at least once. In addition, ongoing mortality surveillance is conducted by review of alumni records and obituaries and by periodic National Death Index searches. A committee of internists (including M.J.K. and D.E.F.) reviews copies of the death certificates, medical records, and self-reports to assign disease outcomes and assess cause of death.

MEASURE OF CHILDHOOD SES

Childhood SES was defined on the basis of the subject's father's occupation as reported from a 10-item checklist on a medical school questionnaire, derived from the occupations listed on the 1950 census of the population. A subject was classified as having a low childhood SES if he reported that his father was a farmer, laborer, service worker, clerical worker, sales worker, machine operator, or craftsman. A subject was classified as having a high childhood SES if he reported that his father was a professional (such as an accountant, banker, scientist, engineer, or lawyer), manager, or physician. In sensitivity analyses, alternate definitions of childhood SES were based on the mother's, as well as the father's, occupation. Socioeconomic status was also categorized as a 3-level variable: low (farmer, laborer, service worker, clerical worker, sales worker, machine operator, or craftsman), and high (professional, physicians, and managers).

COVARIATES

Information about other risk factors for CHD was also collected at baseline and during follow-up using annual questionnaires and medical records. Starting with the class of 1949, nonfasting serum cholesterol level was measured during medical school. Physical activity was assessed during medical school and follow-up using the question, "How much physical training have you had in the past month?" Possible responses were "none," "little," "moderate," and "much." Parental history of premature CHD was defined as development of CHD before age 55 years in a participant's father or before age 65 years in his mother. Prevalence of CHD in parents was assessed at baseline and throughout follow-up.

Body mass index (BMI) was defined as weight in kilograms divided by the square of height in meters. Number of cigarettes smoked per day was categorized as follows: 0, 1 to 10, 11 to 20, 21 to 39, and 40 or more. Participants indicated their current coffee intake based on 8 possible responses ranging from 0 to 7 or more cups a day. Hypertension was defined as a blood pressure of 160/105 mm Hg or greater on 1 annual questionnaire, 140/90 mm Hg on at least 2 readings at least 1 week apart, or pharmacologic treatment of hypertension. Type 2 diabetes mellitus was defined as report of pharmacologically treated diabetes on a single questionnaire, report of nonpharmacologically treated diabetes on 2 or more questionnaires, physician diagnosis of diabetes in medical records, or report of a fasting plasma glucose level of at least 140 mg/dL (7.8 mmol/L) or a nonfasting plasma glucose level of at least 200 mg/dL (11.1 mmol/L). Incidence of clinical depression was measured on the mailed surveys with direct questions concerning the occurrence of depression and associated treatment.

OUTCOME MEASURES

The primary dependent variable for this analysis was the incidence of CHD occurring on or before age 50 years. Secondary dependent variables were all CHD (incident events, fatal and nonfatal) occurring at any age, CHD mortality, and total mortality. Coronary heart disease was defined as myocardial infarction, sudden death, angina pectoris, chronic ischemic heart disease, and other coronary disease requiring coronary bypass surgery or percutaneous coronary intervention.

STATISTICAL ANALYSIS

Follow-up began at graduation from medical school and continued through December 31, 2001. Baseline characteristics for physicians from low- and high-SES families were summarized and compared using means and t tests for continuous variables and percentages and χ² tests for categorical variables. Kaplan-Meier methods and the log-rank test were used to compare the time to event for incident CHD and mortality between physicians with low and high childhood SES. Proportional hazards models were used to assess the impact of possible CHD risk factors on the relationship between childhood SES and CHD. These factors were serum cholesterol level and exercise during medical school and the presence of parental history of premature CHD and of hypertension, diabetes mellitus, depression, smoking, coffee drinking, and BMI as time-dependent covariates. Follow-up was truncated at age 50 years in models used to assess the effects of covariates on incidence at or before age 50 years. The relatively few CHD events at and before age 50 years limited our ability to adjust for multiple variables simultaneously. Thus, bivariate models were used to determine whether the effect of childhood SES on CHD was mediated through other CHD risk factors. The propor tional- ity of hazards was confirmed with log-log plots and by examining the Schoenfeld residuals. A 2-tailed P < .05 was considered statistically significant. All statistical analyses were carried out with commercially available software (Stata Release 8.0; StatCorp, College Station, Tex).

RESULTS

BASELINE CHARACTERISTICS

The baseline characteristics of the 1131 men in this study assessed during medical school are given in Table 1. Of 1131 subjects, 216 (19.1%) were from low-SES families. Medical students from low-SES families were slightly older than those from high-SES families (39.8 years v 39.4 years; P = .004). Significant differences included higher systolic blood pressure (140.6 mm Hg v 139.5 mm Hg; P = .002), higher diastolic blood pressure (81.5 mm Hg v 80.4 mm Hg; P = .002), and higher body mass index (28.1 26.3; P < .001). The proportion of men who were current smokers was significantly higher among those from low-SES families (60.4% vs 51.5%; P = .005). The prevalence of parental history of premature CHD was higher among those from low-SES families (22.2% vs 17.7%; P = .04). The proportion of men who reported having a parental history of hypertension was not significantly different between the 2 groups (31.0% vs 28.2%; P = .22). The prevalence of parental history of diabetes mellitus was also not significantly different between the 2 groups (12.7% vs 10.6%; P = .25). The proportion of men who reported a parental history of depression was significantly higher among those from low-SES families (15.9% vs 10.7%; P = .003). The prevalence of parental history of suicide or suicide attempt was not significantly different between the 2 groups (4.5% vs 4.8%; P = .82). The prevalence of parental history of other coronary disease requiring coronary bypass surgery or percutaneous coronary intervention was not significantly different between the 2 groups (2.2% vs 2.6%; P = .67). The prevalence of parental history of myocardial infarction was not significantly different between the 2 groups (11.9% vs 10.7%; P = .48). The prevalence of parental history of sudden death was not significantly different between the 2 groups (2.6% vs 2.3%; P = .76). The proportion of men who reported a history of substance use disorder was not significantly different between the 2 groups (4.5% vs 3.6%; P = .48). The proportion of men who reported a history of alcohol use disorder was not significantly different between the 2 groups (9.8% vs 7.9%; P = .23). The proportion of men who reported a history of drug use disorder was not significantly different between the 2 groups (1.3% vs 0.9%; P = .54). The proportion of men who reported a history of anxiety disorder was not significantly different between the 2 groups (9.2% vs 7.8%; P = .40). The proportion of men who reported a history of mood disorder was not significantly different between the 2 groups (13.0% vs 9.1%; P = .13). The proportion of men who reported a history of personality disorder was not significantly different between the 2 groups (2.2% vs 2.1%; P = .91). The proportion of men who reported a history of behavioral disorder was not significantly different between the 2 groups (2.2% vs 2.1%; P = .91).

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though this difference was not significant. There was no
difference in the age at onset of diabetes mellitus, with a
mean age of 57 years in both groups.

**CHILLOHD ASES AND RISK OF CHD**

Median follow-up time in this analysis was 40 years, yielding
43,160 person-years of observation. During this period,
217 men experienced CHD events. The incidence of
CHD from age 40 to age 75 years was consistently greater
in men from lower childhood SES backgrounds than in
those from higher SES families (Figure). During the en-
tire follow-up, differences in incidence between the groups
did not achieve a conventional level of statistical signifi-
cance ($P=.11$; Figure), but the incidence of CHD on or
before age 50 years was significantly greater in physi-
cians with low compared with high childhood SES
($P=.01$). Incidence of CHD, fatal CHD, and total mortality by 50
and 70 years are given in Table 3.

In univariate proportional hazards analysis, the un-
adjusted relative risk of developing CHD on or before
age 50 years associated with low childhood SES was 2.40 (95% confidence interval [CI], 1.21-4.74; Table 4). In bivariate
Cox proportional hazards models, the risk of develop-
ing early CHD associated with low childhood SES was
not reduced after adjusting for serum cholesterol level

### Table 1. Baseline Characteristics According to Childhood SES in 1131 White Male Medical School Graduates in The Johns Hopkins Precursors Study*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Low SES (n = 216)</th>
<th>High SES (n = 915)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Graduation age, mean (SD), y</td>
<td>26.8 (2.7)</td>
<td>26.2 (2.3)†</td>
</tr>
<tr>
<td>BMI, mean (SD)</td>
<td>23.0 (2.7)</td>
<td>23.1 (2.5)</td>
</tr>
<tr>
<td>Serum cholesterol, mean (SD), mg/dL</td>
<td>193 (31.1)</td>
<td>192 (28.7)</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>113 (52.3)</td>
<td>422 (46.1)</td>
</tr>
<tr>
<td>Coffee, mean (SD), cups/d</td>
<td>2.4 (2.0)</td>
<td>2.2 (1.8)</td>
</tr>
<tr>
<td>Physical training in the past month</td>
<td>No</td>
<td>Little</td>
</tr>
<tr>
<td></td>
<td>89 (50.6)</td>
<td>251 (31.8)</td>
</tr>
<tr>
<td>Moderate/much</td>
<td>32 (18.2)</td>
<td>147 (18.6)</td>
</tr>
<tr>
<td>Parental premature CHD</td>
<td>28 (11.6)</td>
<td>131 (14.3)</td>
</tr>
</tbody>
</table>

*Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); CHD, coronary heart disease; SES, socioeconomic status.
†SI conversion factor: To convert cholesterol to millimoles per liter, multiply by 0.0259.
*Data are presented as number (percentage) unless otherwise indicated. Denominators may vary because of missing data.

### Table 2. BMI and Cigarette Smoking During Follow-up by Childhood SES in 1131 White Male Medical School Graduates in The Johns Hopkins Precursors Study*

<table>
<thead>
<tr>
<th>Age, y</th>
<th>BMI, Mean (SD) Low SES</th>
<th>BMI, Mean (SD) High SES</th>
<th>Cigarette Smoking, No. (%) Low SES</th>
<th>Cigarette Smoking, No. (%) High SES</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>23.0 (2.7)</td>
<td>23.1 (2.5)</td>
<td>94 (56.0)</td>
<td>389 (49.2)</td>
</tr>
<tr>
<td>30-39</td>
<td>24.0 (2.7)</td>
<td>23.8 (2.6)</td>
<td>95 (47.0)</td>
<td>357 (41.1)</td>
</tr>
<tr>
<td>40-49</td>
<td>24.7 (2.8)</td>
<td>24.1 (2.7)†</td>
<td>51 (25.9)</td>
<td>192 (23.6)</td>
</tr>
<tr>
<td>50-59</td>
<td>25.0 (3.3)</td>
<td>24.5 (3.0)†</td>
<td>33 (19.0)</td>
<td>107 (14.4)</td>
</tr>
<tr>
<td>60-69</td>
<td>25.7 (3.5)</td>
<td>25.0 (3.4)†</td>
<td>18 (11.9)</td>
<td>50 (7.7)</td>
</tr>
</tbody>
</table>

*Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); SES, socioeconomic status.
†The length of follow-up ranged from 34 to 53 years. The BMI and smoking history data per decade were based on the last BMI measurement and smoking status reported in that age decade. Denominators may vary because of missing data.

### Table 3. Kaplan-Meier Analysis of All CHD, CHD Mortality, and Total Mortality by Ages 50 and 70 Years in The Johns Hopkins Precursors Study

<table>
<thead>
<tr>
<th>Variable</th>
<th>Incidence by Childhood SES, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>By Age 50 y</td>
</tr>
<tr>
<td></td>
<td>Low SES</td>
</tr>
<tr>
<td>Incident CHD</td>
<td>6.2</td>
</tr>
<tr>
<td>Fatal CHD</td>
<td>1.4</td>
</tr>
<tr>
<td>Total mortality</td>
<td>3.8</td>
</tr>
</tbody>
</table>

*Abbreviations: CHD, coronary heart disease; SES, socioeconomic status.
†By log rank test, comparing low vs high childhood SES.

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**CHANGES IN CARDIOVASCULAR DISEASE RISK FACTORS OVER TIME**

Body mass index increased over time in physicians from both low-SES and high-SES families (Table 2). Physicians from low-SES families, however, had a significantly
higher mean BMI at ages 40 to 49, 50 to 59, and 60 to 69
years. While the rates of cigarette smoking decreased in
both groups over time, physicians from low-SES families
had higher smoking rates in each decade, although the dif-
ferences were not statistically significant.

The mean age of developing hypertension and dia-
betes mellitus was similar in the 2 groups. Physicians from
low-SES families developed hypertension 1 year earlier
than did physicians from high-SES families, with a mean
(SD) age at onset of 54.1 (13.3) vs 55.0 (13.0) years, al-
though this difference was not significant. There was no
during medical school, amount of exercise during medical school, amount of cigarette smoking or coffee drinking during follow-up, BMI during follow-up, parental history of premature CHD, or development of diabetes mellitus, hypertension, or depression during follow-up (Table 4).

CHILDC childhood SES AND MORTALITY

During a median follow-up of 40 years, 52 deaths related to CHD and 224 total deaths occurred. When the entire follow-up was considered, participants with low childhood SES demonstrated a trend toward increased CHD mortality (hazard ratio [HR], 2.0; 95% CI, 0.99-3.90) compared with physicians with high childhood SES, but no such association was seen for all-cause mortality (HR, 1.08; 95% CI, 0.78-1.48). There were few events by age 50 years (6 deaths due to CHD and 42 total deaths). When only these events were considered, there was the suggestion of an association of low SES with CHD death (HR, 4.27; 95% CI, 0.86-21.17), but not with all-cause mortality (HR, 0.99; 95% CI, 0.46-2.14).

SENSITIVITY ANALYSIS

When information on the mother’s occupation was used to reclassify 19 of the participants with low childhood SES as high-SES subjects, our results were unchanged. In addition, analyses using 3 levels of childhood SES also showed a higher risk of premature CHD in the lowest SES group (data not shown).

COMMENT

The present study found that low childhood SES is a risk factor for incident CHD before age 50 years in men with uniformly high adulthood SES. We noted a trend toward increased CHD mortality in physicians of low childhood SES, but no difference in all-cause mortality. This increased risk exists despite physicians’ high level of SES as adults, their medical knowledge, and their access to high-quality health care. The higher risk of early CHD was not mediated by BMI, cholesterol level, exercise, depression, coffee drinking, smoking, hypertension, diabetes mellitus, or parental history of CHD.

Several conceptual models have been proposed to explain how lower childhood and adulthood SES may lead to increased risk for cardiovascular disease. The latent effects model proposes that adverse early life experiences permanently affect the individual in a way that is not influenced by adult experiences. The pathway model suggests that early life experiences affect adult health by influencing adult behavior and risk factors. The cumulative model hypothesizes that health is determined by the cumulative number of years that an individual spends in either a low- or high-SES category. Individuals with low childhood SES in our study did have higher BMI and were more likely to smoke cigarettes than were individuals with high childhood SES, supporting the pathway model. Low childhood SES was associated with higher CHD risk only before the age of 50 years. After that age, individuals with low SES in childhood in this cohort had spent proportionately more of their lifetime at a high SES than at a low SES.

Low childhood SES may confer an increased risk of CHD on or before age 50 years but not later in life for several reasons. First, other CHD risk factors are more prevalent later in life; thus, the proportionate effect of low childhood SES may be reduced. A similar relationship has been noted in The Johns Hopkins Precursors Study cohort with other risk factors. Second, the current study may have been underpowered to detect a small risk of CHD at older ages. For example, in the Nurses’ Health Study of 117,006 participants, the age-adjusted risk of total cardiovascular disease events was 1.13 (95% CI, 1.02-1.24) for nurses with low vs high childhood SES, similar to the risk seen in the present study for incident CHD over the lifespan, 1.29. Likewise, the lack of statistical significance for the 2-fold increase in the risk of CHD mortality associated with low childhood SES likely reflects lack of statistical power.

Three other studies have examined the risks of CHD in individuals who have moved from lower SES to higher SES. Two of them, a case-control study of former Harvard University students and the Nurses’ Health Study, found a higher risk in persons with low childhood SES. A study of Finnish men born in the late 1950s found that adulthood SES had a greater effect on mortality than did childhood SES, although childhood SES had a persistent effect on mortality from cardiovascular disease.

Besides these studies, which specifically examined the move from low childhood SES to high adulthood SES, many other studies have investigated the relationship between childhood SES and cardiovascular events. However, most of these studies were conducted outside the United States.

Table 4. Risk of CHD Associated With Low Childhood SES at Different Age Thresholds in 1131 White Male Medical School Graduates in The Johns Hopkins Precursors Study

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Age ≤50 y</th>
<th>All Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of events, low SES/high SES</td>
<td>13/23</td>
<td>51/166</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>2.40 (1.21-4.74)</td>
<td>1.29 (0.94-1.77)</td>
</tr>
<tr>
<td>Adjusted for</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI†</td>
<td>2.39 (1.21-4.73)</td>
<td>1.27 (0.93-1.74)</td>
</tr>
<tr>
<td>Cholesterol level†</td>
<td>2.31 (1.14-4.66)</td>
<td>1.28 (0.91-1.80)</td>
</tr>
<tr>
<td>Exercise†</td>
<td>3.21 (1.53-6.71)</td>
<td>1.46 (1.03-2.08)</td>
</tr>
<tr>
<td>Depression†</td>
<td>2.41 (1.22-4.75)</td>
<td>1.30 (0.95-1.77)</td>
</tr>
<tr>
<td>Coffee drinking†</td>
<td>2.38 (1.21-4.70)</td>
<td>1.29 (0.94-1.76)</td>
</tr>
<tr>
<td>Smoking†</td>
<td>2.24 (1.11-4.51)</td>
<td>1.25 (0.91-1.72)</td>
</tr>
<tr>
<td>Hypertension†</td>
<td>2.39 (1.21-4.72)</td>
<td>1.26 (0.92-1.72)</td>
</tr>
<tr>
<td>Diabetes mellitus†</td>
<td>2.43 (1.23-4.80)</td>
<td>1.29 (0.94-1.76)</td>
</tr>
<tr>
<td>Parental premature CHD</td>
<td>2.41 (1.22-4.76)</td>
<td>1.31 (0.96-1.79)</td>
</tr>
<tr>
<td>Adjusted for all variables</td>
<td>2.62 (1.18-5.85)</td>
<td>1.27 (0.88-1.84)</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; CHD, coronary heart disease; SES, socioeconomic status.
*Data are presented as hazard ratio (95% confidence interval) unless otherwise indicated.
†Assessed during medical school.
‡Assessed during follow-up.
possible confounders occurring later than age 40 years.10,28
Nevertheless, low childhood SES was associated with a
higher risk of cardiovascular events in all of the studies,
even the effects of adjustment for established CHD
risk factors and adulthood SES varied.13,16,18 Thus, com-
pared with all studies of childhood SES and CHD, the
present study offers a unique perspective of early CHD
in a well-defined cohort of medical school graduates.

The present study adds to the body of information on
childhood SES and CHD in several ways. First, data on
childhood SES were collected earlier in adulthood, be-
fore clinical events had occurred, minimizing possible re-
call bias. Second, all of the participants graduated from
medical school and thus had uniformly high adulthood
SES, thereby eliminating confounding by changes in adul-
thood SES. Finally, unlike the other studies, we examined
the relationship between childhood SES and early CHD.

Although different life-course models help to concep-
tualize the relationship between childhood SES and the
development of CHD in adulthood, the underlying bio-
llogical factors are less clear. The association between lower
SES in adulthood and CHD risk has been ascribed to a
higher prevalence of CHD risk factors in adults with low
SES, including hypertension,39 the metabolic syn-
drome,40 and unhealthy behaviors, including smoking,
high-fat diets, and low levels of physical activity.41 Ev-
dence also suggests that adults with low SES have differ-
cental activation of biological pathways that are impic-
cated in the development of CHD. Compared with adults
of high SES, those with lower SES have higher levels of
cortisol,42 higher levels of C-reactive protein,43 greater plate-
let activation,44 and more atherogenic lipid profiles.45 More
data are needed on the persistence of the relationships
between low SES and these biological variables.

Some limitations of our study deserve mention. Our
exposure definition, parental occupation, is based on self-
report, which can be subject to error. If persons with low
childhood SES statistically reported higher levels, as
defined by parental occupation, then the true associa-
tion between low SES and CHD would most likely be
diluted. In addition, these findings should be generalized
with caution to groups other than male physicians of Eu-
ropean ancestry. Furthermore, our conclusions are based
on a relatively small number of events before age 50 years.
Another limitation is the use of paternal occupation as a
measure of childhood SES. Socioeconomic status is a mul-
tidimensional construct, and other studies have used par-
ents’ level of education, occupation, and/or income as mea-
sures.46,47 Income may be the best marker of childhood
SES,48-50 but occupation is used in about a quarter of stud-
ies.46 Nevertheless, The Johns Hopkins Precursors Study
cohort offers study strengths. The strengths of the present study
include a long follow-up period and an excellent response
rate from participants. The cohort design also allows for ac-
curate measurement of confounders that vary over time and
for precise measurement of exposure status before disease
onset without the risk of recall bias or reverse causality.
In addition, self-reports of CHD risk factors and clinical out-
comes have been validated in this cohort.31

In conclusion, these results indicate that, even for in-
dividuals with uniformly high SES in adulthood, the pres-
ence of low SES in childhood confers an increased risk
of developing CHD before age 50 years. This increased
risk is not mediated by established CHD risk factors. These
findings highlight the importance of childhood events in
the development of CHD in adulthood. More impor-
tantly, the data illustrate the difficulty in eliminating health
status disparities between low-SES and high-SES popu-
lations: even an intervention that allowed low-SES indi-
viduals to assume the lifestyle of physicians did not to-
tally eliminate disparities in early CHD outcomes.

Accepted for Publication: August 29, 2006.

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and Klag. Acquisition of data: Kittleson, Wang, Ford, and
Klag. Analysis and interpretation of data: Kittleson, Meoni,
Wang, Chu, Ford, and Klag. Drafting of the manuscript:
Kittleson. Critical revision of the manuscript for impor-
tant intellectual content: Kittleson, Meoni, Wang, Chu,
Ford, and Klag. Statistical analysis: Kittleson, Meoni,
Administrative, technical, and material support: Ford and
Klag. Study supervision: Ford and Klag.

Financial Disclosure: None reported.

Funding/Support: This study was supported by grants
K24 DK02856, R01 AG01760, and T32 HL007227-30
from the National Institutes of Health.

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