Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature

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Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature

George A. Kaplan, PhD, and Julian E. Keil, DrPH

An association between lower socioeconomic status* (SES) and poorer health has been observed for hundreds of years. This report summarizes the evidence for an important association between socioeconomic status and cardiovascular diseases. This report is divided into sections. The first two sections provide background on cardiovascular disease and its risk factors and the conceptualization and measurement of SES. The two major sections that follow review the evidence supporting an association between SES and all-cause mortality and between SES and cardiovascular disease. The latter section includes evidence on incidence, prevalence, and mortality from, as well as on trends in, cardiovascular disease.

To attempt to explain the inverse relation between SES and cardiovascular disease, the role of psychosocial factors is examined briefly and, in much greater detail, the role of standard biologic cardiovascular risk factors. Based on these data, which indicate for the most part an inverse relation between measures of SES and risk factor levels, and also on a number of prospective studies, the relatively new body of evidence indicating that SES is an independent risk factor for cardiovascular disease is examined, along with the causal criteria used in evaluating SES as a risk factor. That SES may be an independent risk factor simply admits to the current lack of knowledge about the behavioral, social, psychological, and biologic pathways by which SES affects cardiovascular disease. Although the available evidence indicates that SES is independent of other known risk factors, future studies undoubtedly will clarify the environmental and biologic pathways involved. Finally, the effect of SES in major cardiovascular intervention trials is considered, then the implications of all the data reviewed earlier. Basic recommendations for using and building on existing information about SES are also provided.

*The term "socioeconomic status" covers a wide range of measures, sometimes referred to as "social class," but it includes measures of education, income, occupation, living conditions, income inequality, and many other socioeconomic aspects of life. "Socioeconomic status" is used here for simplification.

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The evidence appears to support the argument that SES is an important factor in the etiology and progression of cardiovascular disease. Although the evidence is relatively consistent, different readers will undoubtedly draw different conclusions about steps for reducing disparities in the socioeconomic burden of cardiovascular disease. It is hoped that this report will help stimulate discussion about this topic.

Although this review attempts to present a comprehensive view of the relation between SES and cardiovascular disease, because of the volume of literature, it is impossible to comment critically on every article. The original articles and reviews on the variously defined association between SES and cardiovascular or coronary disease and their risk factors were located through MEDLINE searches. The authors also used extensive personal files compiled over the past 20 years. The review of the literature was limited to publications from the United States, Canada, the United Kingdom, Germany, and the Nordic countries. There are variations in the quality of these articles, but the consistency of the results is quite impressive, and most of the articles in the reading list are from peer-reviewed journals. For example, 21 (11%) were from the *American Journal of Epidemiology;* 18 (10%) from the *American Journal of Public Health;* 9 (5%) from the *Journal of the American Medical Association;* 20 (10%) from other well-known epidemiology journals; 16 (9%) from *Lancet* or the *British Medical Journal;* and 22 (11%) from clinical journals such as the *American Heart Journal* and *Circulation.* An additional 11 (5%) were from government publications.

The purposes of writing this review of SES and cardiovascular disease were several: To provide a summary documentation of the effects of SES on cardiovascular disease morbidity and mortality; to provide insights into the conceptualization, measurement, and components of SES; to emphasize a relatively new body of evidence that SES may, in addition to exerting effects through commonly accepted risk factors, function independently of these factors; to identify the implications of the existing data; and to provide basic recommendations for using and building on existing information about SES.

Cardiovascular Disease and Its Risk Factors

The observations of Virchow[1] in 1856 concerning changes in the arterial wall in atherosclerosis paved the

way for intensive study of the pathology of cardiovascular disease. This study led to the theory that lipid materials deposited in the intima of medium-sized and larger arteries leads to atheromatous deposits and ulceration of the intimal surface in contact with the circulating blood. Ancel Keys,[2] one of the pioneers in cardiovascular disease epidemiology, advanced the idea that atherosclerotic disease (particularly coronary heart disease) was not an inevitable result of aging but rather was related to environmental factors.

Beginning in the 1900s there was a decline in mortality from infectious disease, attributable initially to improvements in sanitation and attention to personal hygiene and then to the discovery and use of vaccines and various antibiotics. Concomitant with this change was the gradual increase in mortality due to the degenerative diseases, including heart disease. Until the 1940s and 1950s, the primary cardiovascular concerns of the medical profession were the effects of rheumatic fever and syphilis on the heart and the influence of high blood pressure on stroke.

Dawber[3] has charted the organization and reorganization of the health activities of the US government, beginning with the reorganization of the federal bureaucracy by Franklin Roosevelt. The focus for heart disease research as a national priority began in 1949 with the creation of the National Heart Institute of the National Institutes of Health.

One of the cardinal resources of the new National Heart Institute was the Framingham Heart Study, conceived in the late 1940s and implemented in 1950 in Framingham, Mass. Many studies have replicated the results of the Framingham study, confirming that the primary risk factors for cardiovascular disease are cigarette smoking, hypertension, elevated serum cholesterol level, sedentary lifestyle, and diabetes. However, these risk factors influence only about 50% of overt cases of coronary heart disease.[4] Numerous investigators have studied other possible risk factors, such as hemostatic factors, obesity, hostility, stress, noise, and coping styles. Based on a large body of evidence, we believe SES should be added to the list of potential risk factors for coronary disease.

For decades, analyses of vital statistics and other data have demonstrated an inverse relation between mortality and SES. The relations of SES to cardiovascular disease, other risk factors for cardiovascular disease, and changes in cardiovascular risk factors will be the focus of this commentary. The evidence for such relations is abundant.

Conceptualization and Measurement of Socioeconomic Status

A variety of approaches to the conceptualization and measurement of SES (social class, social status, etc) have been taken, reflecting different theoretical orientations on the one hand and the exigencies of conducting research on the other. The complex issues involved in the conceptualization and measurement of socioeconomic position in epidemiological studies have been reviewed by several authors.[5-7] The debate on the conceptualization of SES has a long history as part of sociological theory, the intricacies of which are not germane to this discussion. However, approaches have generally reflected the orientations of either Weber[8] or Marx.[9] The Weberian approach, which has heavily influenced American sociology, sees social stratification as organized around the independent tripartite entities of class (economic interest), status, and power. This approach has tended to use summary measures of SES that include those indicating the more subjective issue of status as well as more objective measures such as income or education. Indeed, there has been a tendency to emphasize the aspect of status in contemporary work, which shares this historical base. The Marxist approach is, to oversimplify, based on analysis of the opposing interests of those who differ with respect to ownership of the means of production. Groups differing with respect to their place in the production process are seen as locked in inevitable conflict. This approach has led to a focus on issues of power, control, and ownership.[10]

For the most part, epidemiologic studies have not been explicitly driven by these opposing conceptions of SES, but it is useful to keep these orientations in mind in a review of the literature. Although the Weberian and Marxist approaches have had some influences on studies of the association between socioeconomic factors and health, these studies are in general best characterized by their theoretical eclecticism.

With this background in mind, the most commonly used measures, indexes, and ecological measures of social class are reviewed below.

Education

Education is the most widely used measure of SES in epidemiologic studies. Of the studies of chronic disease published in the *American Journal of Epidemiology* in 1982 and 1985 in which measures of SES were used, education was used by 45% as a surrogate measure of SES.[7] There are a variety of reasons for this choice. Questions to respondents about education have relatively low nonresponse rates and are not complex, and education is highly valued by the well-educated researcher who is designing the study. In addition, because level of education is usually fixed after young adulthood, it is unlikely that it is influenced by poor health among adults. Although education is a useful measure for all of these reasons, it does have limitations. For example, there are large birth cohort differences in level of education, so that the social, behavioral, and psychological correlates of a given level of education may vary depending on a subject's age. The value of education as a measure of SES may also differ for specific subgroups. For example, for African-Americans and women, the link between education and income is weaker than it is for Caucasian men. Finally, although illness in adulthood cannot influence level of education completed before adulthood, it is not implausible that poor health in childhood could influence amount or quality of education received.[11]

Income

Measures of income are obviously an important marker of SES. Income provides access to goods and services, including quality education and medical care, that may protect against disease. However, lower income may reflect the influence of poorer health. In addition, the measurement of income level is complex: Individual or family income can be measured, family income can be adjusted for family size, income can be

compared to poverty levels, sources of income other than wages can be included, noncash benefits such as food stamps or Medicare can be included, and income levels may vary over time. It may also be important to measure wealth, which includes total assets. Income was reported in only 15% of the articles published in the *American Journal of Epidemiology* that included measures of social class.[7] Failure to incorporate measures of income may be due to perceived sensitivity of income-related questions. Nonresponse by subjects to income-related questions averages 9% to 10%. However, many studies have lower rates of nonresponse, suggesting that respondents need to be reassured about the confidentiality of the data they provide.

Occupation

Health outcomes for specific occupations have been examined in numerous studies, but the use of occupation as a measure of SES generally involves some categorization. These categorizations are based on issues of status and roles, power, prestige, lifestyle, job characteristics, income and education, traditions, beliefs, and values.[5] Used in this way, classification of occupation represents something quite different from the specific job characteristics and exposures usually associated with it. Two examples illustrate this form of occupational classification. The US Bureau of the Census has categorized occupations since 1897,[5] and a reasonably consistent set of categories has been in use since 1910. This scale, based on putative social rank, divides occupations into the following 12 ordered categories: professional; technical and kindred workers; managers and administrators (other than farm); sales workers; clerical and kindred workers; craftsmen and kindred workers; operatives (except transport); transport equipment operatives; laborers (except farm); farmers and farm managers; service workers (excluding household); and private household workers. These categories are roughly comparable to those of the Registrar General of Great Britain, with some discrepancies in ordering.[12] This categorization, which has been in use in one form or another since 1911, currently divides the population into six classes presumptively based on education and culture: I, professionals (eg, doctors, lawyers, executives); II, managerial and lower professionals (eg, sales managers, teachers); IIIN, nonmanual skilled (eg, clerks, shop assistants); IIIM, manual skilled (eg, machinists); IV, partly skilled (eg, postmen); V, unskilled (eg, laborers, porters).[12] These can be grouped into nonmanual (I-IIIN) and manual occupations (IIIM-V). A similar grouping has been used within an occupation, with civil service workers being characterized as administrative, professional-executive, clerical, or other.[13]

Occupation is an important status characteristic in modern societies, and numerous studies have indicated that people readily rank occupations in terms of prestige and status. In the survey of the use of social class measures in articles published in the *American Journal of Epidemiology*, occupation was used as a measure in 22% of those articles that had measures of SES.[7] However, numerous difficulties are associated with the use of occupational measures. For one, considerable information should be collected about occupational history so the classification will reflect more than current occupation, which could easily reflect the impact of disease. A more critical difficulty stems from the breadth of the occupational groupings used in most systems. For example, the chief executive officer of a large multinational corporation and a proprietor of a small family business would be the same rank in some systems, or a skilled manual worker might have an income that considerably exceeds that of a university professor. In fact, Karasek et al[14] have argued that classifications based on characteristics such as decision latitude, time pressure, intellectual discretion, and other job-related characteristics provide a better way of grouping occupations with respect to SES. Other difficulties stem from the need for accurate rankings, rapid changes in the existence and status of new occupations, and difficulties in accommodating those who do not work or who are housekeepers. Finally, SES scales based on prestige rankings can be faulted for their inherent subjectivity.

Employment Status

It is also possible to characterize people in terms of current employment status. Although this would not seem difficult, there are a number of interpretations of such a characterization. The most important of these relates to analyses of the association between employment status and health; it is critically important to distinguish between those who are able to work but cannot find employment and those who are unable to work for health reasons. It may also be important to gather information on underemployment and history of job loss. Classification of employment status for students or homemakers is also problematic.

Indexes of Social Class

A large number of indexes that combine various individual measures of SES are also available. Excellent discussions of the major indexes can be found in Morgenstern,[6] Susser et al,[5] and Liberatos et al.[7] These indexes include measures of education, income, occupational prestige, and neighborhood quality. These measures have been used widely, but they suffer from all the difficulties associated with their components, as well as from additional difficulties stemming from the construction of the indexes: for example, the weighting of components. Indeed, in four reviews of SES and health issues, the use of such indexes has not been recommended.[5-7,15]

Measures of Living Conditions

Data can be collected about the material conditions under which people live. For example, ownership of a house, automobile, television, dishwasher, or other material goods has been used in some analyses.[16,17] Such measures will be highly correlated with income and education, but may also indicate important lifestyle differences.

Area-Based Measures

A large number of studies have used measures of SES, both individual and composite, that involve socioeconomic characteristics of the areas in which subjects live. For example, census tracts, block groups, counties, or other geographic units can be characterized with respect to income, education, occupation, crowding,

condition of housing, value of homes or rental prices, or other measures.[18-23] These indicators of SES can be used individually or combined into composite measures. Such measures are often chosen when data on SES is not available for individual subjects. Although use of these measures is subject to the aggregation bias or "ecological fallacy" (that of assuming all individuals in an area have the same or similar characteristics), they may also tap characteristics of the areas in which people live that cannot be measured individually. In one study the distribution of occupational level in areas was associated with the timing of the decline in cardiovascular mortality for those areas.[24] In another, residence in a poverty area was associated with increased risk of death from all causes, even when individual SES was taken into account.[18]

Life Span Measures of Social Class

It is sometimes useful to describe subjects' developmental history with respect to SES. Some studies, for example, have examined childhood SES as well as changes from childhood to adult SES with respect to risk factors and disease.[17,25-27] In these studies researchers have used both individual measures such as education or occupation as well as composite measures involving individual or area characteristics. The collection of this information is subject to all the problems indicated above. In addition, the high correlation, in some societies, between childhood and adult SES lead to analytic problems arising from collinearity.

Measures of Income Inequality

Measures of income inequality can be applied to a society or group to describe the relative equity in the distribution of wealth or income.[28,29] One way of doing this is to ask to what extent the cumulative distribution of wealth in a society or group is matched by the cumulative proportion of the population; for example, what percentage of the group's income is earned by the lowest-earning 40% or 60% of the population. Although attractive with respect to characterizing inequalities in the distribution of income or wealth, such measures are ecological and suffer from the problems in interpretation referred to above.

SES and All-Cause Mortality

There is a considerable body of evidence for an association between socioeconomic factors and mortality from all causes, and this material has been extensively reviewed by a number of authors.[5,15,30-33] Rather than duplicate these reviews, the consistency of this association across measures of socioeconomic level, geographic place, and time is highlighted.

Associations between risk of all-cause mortality and education level, income level, occupational group, composite indexes of these measures, poverty status, unemployment, living conditions or standard of living, and other measures have all been demonstrated using both individual and aggregate data.

Education

Fig 1 is a bar chart of the results of analyses of mortality and educational level from the US National Longitudinal Mortality Study.[34] The National Death Index[35] was used to ascertain the 1979-1985 mortality of

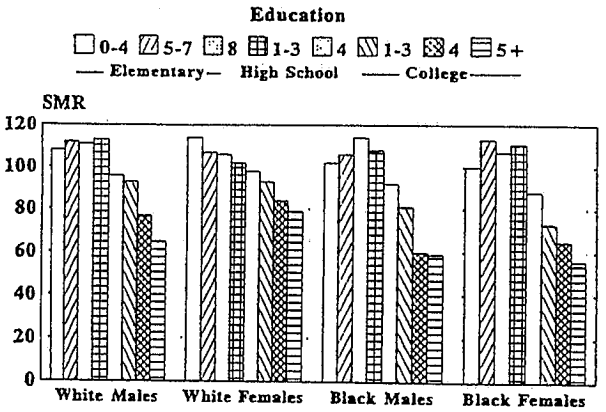

FIG 1. *All-cause mortality by education in subjects 25 years old and older. Adapted from Rogot et al.*[34]

a sample of 1.3 million people interviewed as part of the Current Population Survey and the US Census. For those aged 25 years or older, there was a steady drop in the standardized mortality ratio as educational level increased. For both white and African-American men and women, those who had at least 4 years of high school education had substantially lower death rates than those with less. For white men and women, those with 0 to 4 years of education had approximately 66% and 44% higher standardized mortality ratios, respectively, than those with 5 or more years of college. For African-American men and women, the corresponding ratios were 73% and 78% higher.

Similar results from a study done nearly 20 years earlier by Kitagawa and Hauser[36] are presented in Fig 2. These analyses were based on a sample of 340 000 1960 death certificates that were matched with educational level. The results showed strong mortality gradients associated with level of education for those aged 25 to 64 years. For white men and women, those with 0 to 4 years of education had 64% and 205% higher standardized mortality ratios, respectively, than those with 4 or more years of college.

More recently, analyses of the mortality of participants in the first National Health and Nutrition Examination Survey also showed a strong association between educational level and risk of death (Fig 3).[37] Men aged 45 to 64 with 0 to 7 years of education had 1.96 times the

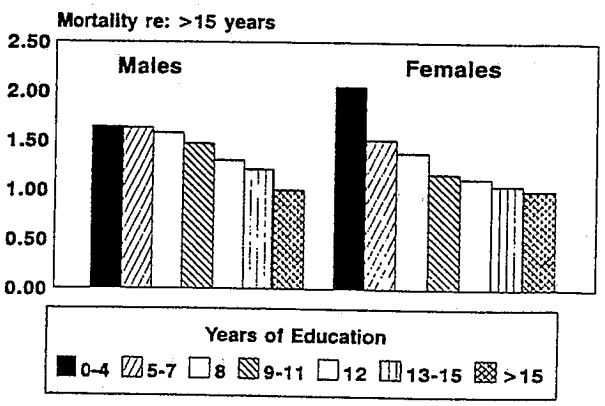

FIG 2. *All-cause mortality by education in white males and females 25 to 64 years old. Adapted from Kitagawa and Hauser.*[36]

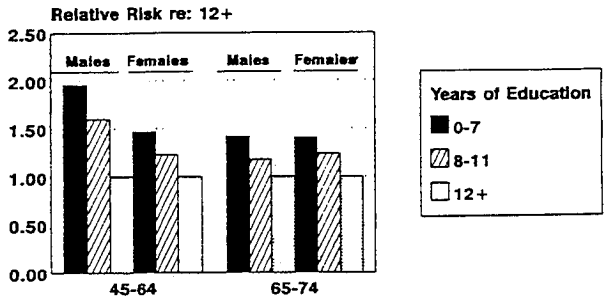

FIG 3. *All-cause mortality by education in men and women 45 to 74 years old. Adapted from Feldman et al.*[37]

risk of death (95% confidence interval, 1.40-2.77) than those with 12 or more years of education, and those with 8 to 11 years of education had 1.6 times the risk (95% confidence interval, 1.19-2.16) as derived from the Cox proportional hazards models. For white women, the corresponding relative risks were 1.47 (95% confidence interval, 0.83-2.61) and 1.23 (95% confidence interval, 0.83-1.85).

Within the United States, analyses of data collected as part of the Alameda County Study,[15] the Washington County Study,[38] the Charleston Heart Study[39,40] (see Fig 4[39]), the Systolic Hypertension in the Elderly Program pilot,[41] three studies in Chicago,[42] and the Hypertension Detection and Follow-up Program[43] have also demonstrated a strong association between educational level and risk of death. Similar results have been found in studies conducted in Norway,[44] the Netherlands,[45] England and Wales,[16] and Finland.[46]

Income

Measures of income are also strongly associated with risk of death from all causes. Figure 5 is a bar chart of the results from the National Longitudinal Mortality Study,[34] (Rogot et al, 1992) which indicate a strong inverse association between total family income and risk of death. White men with incomes of less than $5000 had a standardized mortality ratio 1.8 times greater than that of men with incomes of $50 000 or more, and for white women the ratio was 1.3. Even greater differences were seen for African-Americans. African-American men with incomes less than $5000 had a standardized mortality ratio twice that of men with incomes

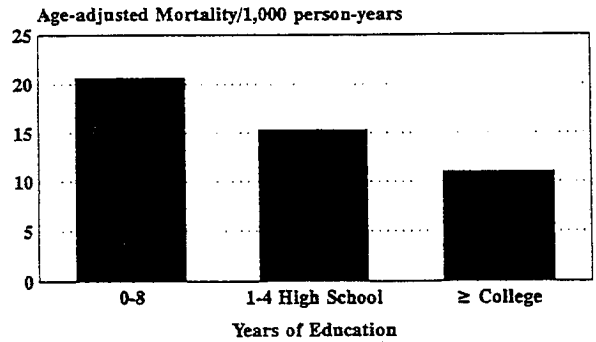

FIG 4. *All-cause mortality by education in subjects 35 to 64 years old in the Charleston Heart Study. Adapted from Keil et al (unpublished data).*

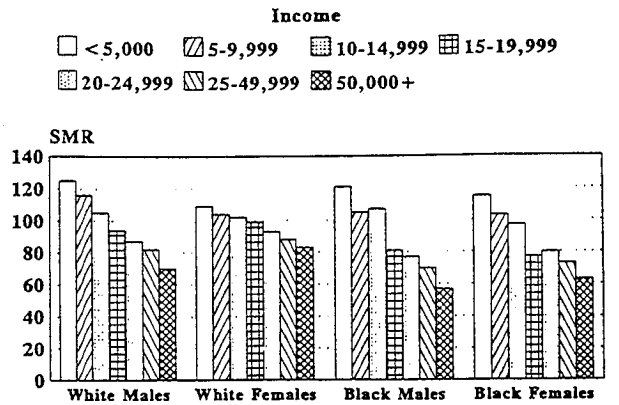

FIG 5. *All-cause mortality by income in subjects 25 years old and older. Adapted from Rogot et al.*[34]

greater than $50 000, and for African-American women the ratio was 1.8.

Kitagawa and Hauser[36] found similar relations. For men and women 25 to 64 years of age, there was a consistently inverse relation between total family income and standardized mortality ratio; men and women earning less than $2000 (in 1959 dollars) had a standardized mortality ratio 79% and 40% higher, respectively, than those making $10 000 or more.

The results from the Alameda County Study (Fig 6), which had a population-based cohort in a typical urban county, show that this association persists throughout a 19-year follow-up period.[15] Total family income was adjusted for family size and compared with federal standards to generate four categories: very adequate, adequate, marginal, and inadequate. Based on an age- and sex-adjusted Cox proportional hazards regression model, those in the "inadequate" category had 2.23 times the risk of death of those in the "very adequate" category ($P<.00001$).

Occupation

The association between occupational group and all-cause mortality has been examined in many studies. The National Longitudinal Mortality Study provides representative findings that vary considerably by gender and race. White men aged 25 years or older who were service workers, laborers, and transportation workers had standardized mortality ratios of 115 or greater, and

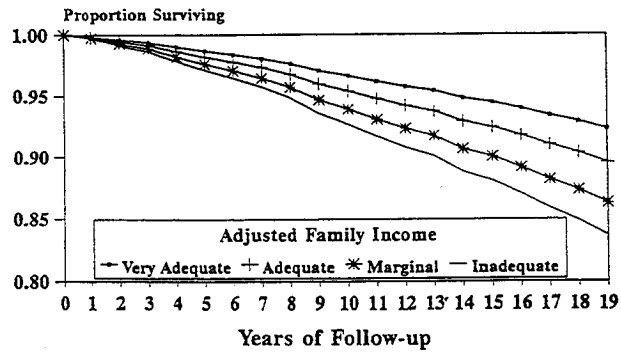

FIG 6. *Nineteen-year survival by income adequacy, adjusted for age and sex, Alameda County Study. Adapted from Haan et al.*[15]

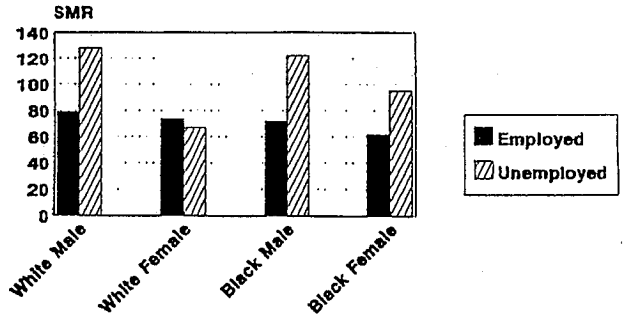

FIG 7. *All-cause mortality by employment status of subjects 25 to 64 years old. Adapted from Rogot et al.*[34]

those in professional-technical occupations had a ratio of 80. Black men aged 25 years or older who were salesmen, service workers, private household workers, and transportation workers had standardized mortality ratios of 112 or greater. White women who were private household workers, laborers, or transportation workers had standardized mortality ratios of 110-120, and white women who were farm laborers had a ratio of 66. Black women who were private household workers had a standardized mortality ratio of 117, and black saleswomen had a ratio of 44, although this was based on a very small number of deaths. These results are similar to the earlier findings reported by Kitagawa and Hauser.[36] For white men 25 to 64 years of age, those who were service workers (including household workers) had a standardized mortality ratio of 137, compared with a ratio of 80 for those who were professional or technical workers.

Longitudinal analyses of the participants in the Charleston Heart Study produced similar findings.[40] The 1960-1988 age-adjusted mortality rate for white men aged 35 to 74 who were classified as craftsmen, foremen, operatives, protective service workers, or laborers was 18.43/1000 person-years, compared with 12.96/1000 person-years for those classified as professionals or proprietors. The age-adjusted mortality rates for clerical workers and salesmen were intermediate.

By far the greatest number of studies of the association between occupational categories and all-cause mortality have been carried out using measures identical or similar to those used by the Registrar General of Great Britain. An inverse relation between occupational class and all-cause mortality has been consistently demonstrated.[47] For example, for 15- to 64-year-old men in England and Wales in 1970-1972, the standardized mortality ratios increased with decreasing social class: 77 for Class I; 81 for Class II; 99 for Class IIIN; 106 for Class IIIM; 114 for Class IV; and 137 for Class V. This association has also been seen in longitudinal studies[16] and within subgroups of, for example, civil servants.[48] Studies in Finland,[49] New Zealand,[50] Sweden,[51] Norway,[52] and other countries have found similar relations.

Employment Status

Being unemployed has consistently been shown to be associated with increased risk of death, as illustrated by results from the National Longitudinal Mortality Study comparing standardized mortality ratios for employed and unemployed 25- to 64-year-olds (Fig 7).[34] Those who were able to work included those who were looking for work, waiting to be called back, or beginning a new job and excluded those who were unable to work, retired, or out of the work force in order to care for home or children or attend school. For white men and women, the standardized mortality ratios for those unemployed, compared with those employed, are 1.6-fold and 0.9-fold greater, respectively. For black men and women the corresponding elevations are 1.7-fold and 1.5-fold. The association between unemployment and increased risk of death was also demonstrated by Kitagawa and Hauser[36] and in studies from Finland[46] and the United Kingdom.[53]

Living Conditions

In some analyses, information about living conditions has been added to that supplied by more traditional measures of SES. In a longitudinal study based on a 1% sample of the population in England and Wales, housing tenure and car ownership were associated with variations in mortality risk within an SES stratum. Even men at the highest level of SES who rented had a standardized mortality ratio 18% higher than those who owned their own homes.[16] In the Whitehall Study, a longitudinal study of British civil servants, car ownership was used as a proxy for level of material conditions. Not owning a car was associated with a 49%-higher mortality risk, and when there was adjustment for social class there was still a 28% higher risk.[54] Researchers in earlier studies in the United States also found measures of living conditions to be important predictors of mortality.[38,55]

Other Measures

Measures of income inequality,[28] deprivation,[56,57] and various indexes of SES[23,58,59] have also been associated with all-cause mortality, both in prospective cohort studies and in cross-sectional ecological studies.

Consistency of Association Over Time

A number of investigators have examined the consistency over time of the association between measures of SES and risk of death from all causes. Feldman et al[37] compared the magnitude of the association between education and all-cause mortality using the 1960 data reported by Kitagawa and Hauser[36] and the National Health and Nutrition Examination Survey Epidemiologic Follow-up Study, which was conducted from 1971 through 1984. In these analyses, which were restricted to those 55 to 84 years old, the trends varied considerably by gender. For men there was a substantial increase in the association between level of education and risk of death. The difference in annual death rates between those with 0 to 7 years of education and those with 13 or more years increased from 2 per 1000 in 1960 to 28.1 per 1000 in 1971-1984. For women, the decline in all-cause mortality rates was experienced relatively equally by those at all educational levels, so that a rate approximately 50% higher among those with a low level compared with a high level of education was maintained. Using an area-based measure, Yeracaris,[23] comparing data from 1960 and 1970, also found increases in socioeconomic disparities in mortality for men.

Comparison of the Kitagawa and Hauser[36] data with the National Longitudinal Mortality Study data[37] (Fig 8) suggests that the relation between occupational

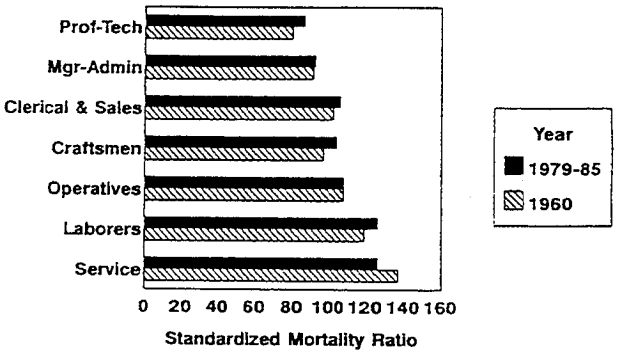

FIG 8. *Occupation and all-cause mortality in the United States in 1979-1985 and in 1960. Adapted from Rogot et al[34] and Kitagawa and Hauser.[36]*

groupings and mortality has remained relatively constant over the last three decades, during a time of considerable declines in mortality rates. However, there are difficulties in comparing occupational groupings and their constituent jobs over such a long period of time. Increasing disparities in the association between SES and death rates from all causes have been found by several investigators who examined data from England and Wales.[60-63]

Socioeconomic Status and Cardiovascular Disease

A large number of studies have examined the relation between SES and cardiovascular mortality, primarily coronary heart disease mortality. The authors reviewed articles dating back to 1956 including ecological studies, cross-sectional or prevalence studies, prospective cohort studies, and special occupational or other studies such as the Systolic Hypertension in the Elderly Program. Most of these papers were from the United States, but many were from Canada, the United Kingdom, and Finland. This assortment of studies used education, occupation, housing tenure, or income or combinations of these as measures of SES. The articles are not necessarily representative of all SES–cardiovascular mortality studies but are a good cross-section of the studies and provide a fair appraisal of the study results. This review has not focused on minorities or the role of SES in minority health differentials but includes information about African-Americans from the Charleston and Evans County Heart Studies.

Socioeconomic Status and Coronary Mortality

Some of the early work in this area was conducted by Abraham M. Lilienfeld.[55] From 1949 through 1951 Lilienfeld identified 14 504 deaths certified as heart disease that had been recorded in Baltimore, Md. Nearly 21% of these were in the nonwhite population. Census tracts in Baltimore were ranked according to the median monthly rental as determined in the 1950 census and then divided into fifths so that 20% of the city's population fell into each fifth. The census tracts comprised neighborhoods of 3000 to 6000 persons who were relatively homogeneous in terms of median monthly rental, occupational status, and home ownership. Each heart disease death was assigned to a socioeconomic fifth on the basis of its allocation to a given census tract. Mortality rates for arteriosclerotic heart disease (including coronary artery disease) showed no significant differences by SES grouping. For mortality from hypertensive disease (with mention of heart disease) the lowest SES group had the highest rates and there tended to be a decrease in rates with an increase in SES.

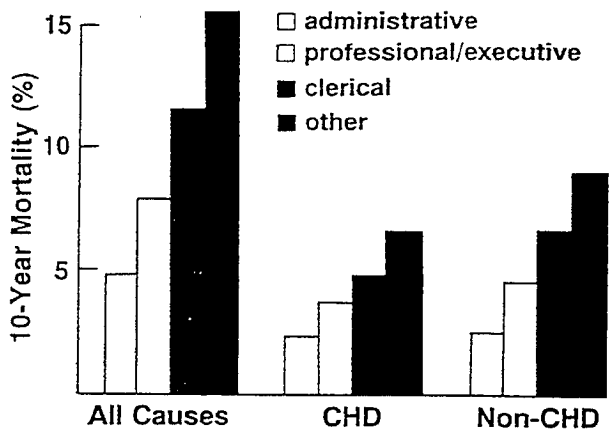

FIG 9. *Age-adjusted percent of men dying in 10 years from all causes, from coronary heart disease, and from non–coronary heart diseases. Subjects were British civil servants. Adapted from Marmot et al.[48]*

In 1971 Cassel et al[64] examined the incidence of coronary heart disease and its association with SES in the Evans County Georgia Heart Study. Cassel reported that, in contrast to earlier results that showed excess prevalence of coronary heart disease in the high-SES group, the 7-year age-adjusted incidence of coronary heart disease in white men was virtually the same in higher- and lower-SES groups (84 per 1000 in the highest group compared with 81 per 1000 in men of low SES). The authors postulated that the excess prevalence previously found in the high-SES group was a function of prior high incidence in this class. When the incidence rates were stratified by age (35 to 54 years and 55 years and older), it was evident that, in the older men, incidence rates were higher among men of high SES and lower in men of lower SES. The opposite association occurred in the younger men; men of high SES had lower incidence rates than men of low SES, suggesting an age-related SES crossover effect. Cassel et al also reported that over 7 years, incidence of coronary heart disease among nonfarming white men tended to be lowest among the professionals and highest among laborers and unemployed workers. The rates for workers in the lower-SES categories were approximately twice those of the professional workers in this rural Georgia community.

One of the outstanding studies of SES and coronary heart disease, the Whitehall Study, was reported by Rose and Marmot.[13] The subjects were 17 530 civil servants aged 40 to 64 at baseline in 1968. The age-adjusted prevalence of angina pectoris was 53% higher for men in the lowest employment grade than for those in the top administrative grade, and ischemic-type electrocardiogram abnormalities were 72% higher in the lower than in the top grades. At follow-up the 10-year coronary mortality rate was 3.6 times higher in the lowest than in the top grades (Fig 9).

In three Chicago epidemiologic studies, as reported by Liu et al,[42] there was an inverse relation between

TABLE 1. Coronary Heart Disease Mortality Rates,* 1960 through 1988 in White and Black Men

Socioeconomic Status/Race Strata	n	Age-Adjusted Rate	(95% Confidence Interval)
Low Socioeconomic Status			
White men (n=151)	33	7.1	(2.2–11.9)
Black men (n=258)	36	4.0	(2.2–5.9)
High Socioeconomic Status			
White men (n=115)	22	3.5	(2.0–5.0)
Black men (n=77)	8	2.4	(0.7–4.1)

*Age-adjusted rate per 1000 person-years.
From Keil et al.[40]

education and long-term risk of coronary heart disease, cardiovascular disease, and all-cause mortality.

In the Systolic Hypertension in the Elderly Program pilot project in 1981 to 1985, Siegel et al[41] reported that low education was a significant predictor (relative risk, 1.9) for myocardial infarction or sudden death in univariate analyses.

In the early 1960s a cohort of 101 black men of high SES was recruited into the Charleston Heart Study.[39] After 14 years of follow-up these men had acute myocardial infarction and coronary heart disease rates half those of other black men in the study who were almost entirely of lower SES. None of the high-SES black men experienced angina pectoris, acute myocardial infarction, or sudden death during the observation period.

Keil et al[40] found in the Charleston Heart Study during 1960 through 1988 that the age-adjusted rates per 1000 person-years of coronary heart disease mortality among white men of low SES was 7.1, compared with 3.5 among high-SES white men. In black men the rates were 4.0 and 2.4, respectively (Table 1).

In 1982 Salonen[46] studied the association of SES with the risk of death from ischemic heart disease, cerebral stroke, and cancer and other diseases in a random sample of 3644 men aged 35 to 59 from eastern Finland. After controlling for age, smoking, blood pressure, serum cholesterol level, and marital status, low education and low income were associated with an excessive risk of death from ischemic heart disease and any disease.

Several Canadian studies have indicated somewhat different effects of SES on mortality than US or other international studies. Hirdes et al[65] found that education was not a significant predictor of mortality in a 20-year follow-up of a cohort of 2000 Ontario men. However, income was a substantial ($P<.001$) predictor of mortality. They reported that the relative risk of mortality for high income compared with low income was 0.41 (95% confidence intervals, 0.23–0.72). In the Quebec Cardiovascular Disease Study 4576 Quebec men aged 35 to 44 who were free of coronary disease in 1974 were followed up through 1985 for first incidence of coronary events (angina pectoris, nonfatal myocardial infarction, or coronary heart disease death). Systolic blood pressure, diastolic blood pressure, cholesterol level, and smoking, but not education, were related to incidence of first event. The investigators observed that blood pressure, cholesterol level, and smoking accounted for two thirds of the attributable risk of first coronary event.[67] Possible explanations for the lack of effect of education are lack of statistical power, the possibility that education only reflects the standard risk factors, or the possibility that the range of education may have been too narrow. In another report from the Quebec Cardiovascular Study, Dagenais et al[68] reported that serum cholesterol and education levels were not significantly associated with total or coronary artery disease mortality. Although it was not statistically significant, an inverse relation between educational level and total mortality was found, whereas an increase was observed in coronary artery disease mortality in men with the highest education.

The prevalence of cardiovascular disease was studied in relation to the educational level of 1560 male industrial employees in Ireland by Mulcahy et al.[68] The higher educational groups had lower levels of coronary risk factors (smoking, diastolic blood pressure, weight, and plasma cholesterol level) and a lower level of coronary morbidity.

In 1968 Hinkle et al[69] found that risk of heart disease was influenced more by education and background than by occupation. They reported the results of a 5-year prospective survey of the relation between occupation/education and coronary disease which was carried out among 270 000 men employed by the Bell System throughout the continental United States. These early findings indicated that men who attained the highest level of management as a group did not have a higher risk of coronary heart disease than men who remained at lower levels. Men who entered the organization with a college degree had a lower incidence and death rate from coronary disease and less associated disability at every age in every part of the country and in all departments.

Heller et al[70] found in England and Wales that the SES gradient for ischemic heart disease was much stronger and steeper for women than for men. There was an excess of ischemic heart disease in men in categories I and II but an excess in women in categories IV and V. The male to female standardized mortality ratios, by category, were as follows: I, 1.5; II, 1.2; IV, 0.9; and V, 0.8.

The US National Longitudinal Mortality Study provided the basis for the more recent work of Rogot et al,[34] which, as indicated previously, showed a strong inverse relation of lower all-cause mortality with higher education and higher income. The study also examined the relation of SES and coronary disease. For all race-gender groups there was an inverse relation of ischemic heart disease mortality with education (Fig 10). This gradient was strongest in white women, in whom the standardized mortality ratio in those with 0 to 4 years of education was 122, compared with 56 among those with 5 or more years of college.

In the Scottish Heart Study, Woodward et al[71] found in a random population sample of 10 359 men and women aged 40 to 59 that housing tenure (owner or renter) was the most discriminating measure of SES in predicting risk of coronary heart disease (physician-diagnosed angina pectoris or myocardial infarction, Rose angina pectoris or Rose myocardial infarction, or electrocardiographic evidence of myocardial infarction

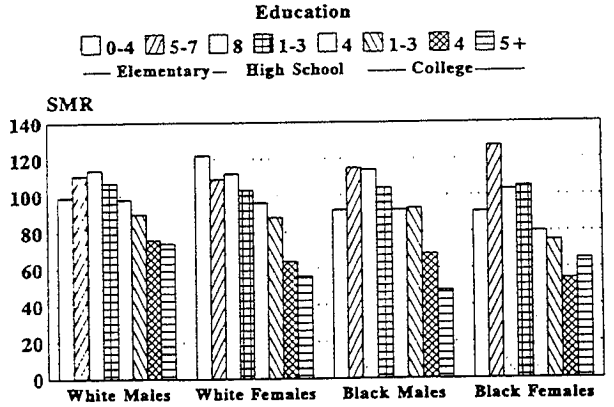

FIG 10. *Ischemic heart disease mortality (International Classification of Diseases 410-414) mortality by education in subjects 25 years old and older. Adapted from Rogot et al.*[34]

or ischemia). After adjustment for coronary heart disease risk factors, housing tenure was highly significant ($P<.001$); the odds ratio was 1.48 in men and 1.45 in women. Four measures of SES (level of education, years of education, housing tenure, and occupational status) were examined and for each measure the least advantaged had a significantly higher prevalence of coronary heart disease. When relations among the social factors themselves were investigated, housing tenure was found to remove the significant effects of education and occupation in men and education in women. The authors report that since World War II private rentals have almost disappeared because of controls on private landlords and incentives for tenants to own. This has resulted in a polarization between owner-occupied and municipal housing, some of the latter good but much of it in deteriorating estates with social problems. The most important confounders in men were smoking and fibrinogen level; in women, body mass index, high-density lipoprotein level, and triglyceride level were found to be important.

Occupation and Coronary Disease

Haynes and Feinleib[72] explored the occupational and behavioral risk factors for coronary heart disease in women in the Framingham Heart Study. Respondents were 45 to 64 years of age at baseline (1965-1967) and were followed for the development of coronary heart disease over the next 8 years. Working women had higher (but not significantly higher) incidence of coronary heart disease than housewives (7.8% and 5.4%, respectively). The 8-year incidence of coronary heart disease was 5.4% for housewives, 6.4% for working women currently employed, 7.8% for working women ever employed, and 12.8% for men. Among working women, clerical workers who had children and were married to blue-collar workers were at the highest risk of developing coronary disease (21.3%). Thus, the influence of the work environment may be carried over to the home.

Eaker et al[73] addressed the 20-year incidence of myocardial infarction or coronary death among women in the Framingham Study. The significant predictor of the 20-year incidence among employed women was perceived financial status; among homemakers, significant predictors were symptoms of tension and anxiety, loneliness during the day, difficulty falling asleep, infrequent vacations, the effects of house work on health, and belief that one is prone to heart disease. Among the employed women there was an inverse relation between education and incidence of myocardial infarction or coronary death; rates were lower in women who held white-collar jobs than in blue-collar workers. Similarly, women whose husbands had white-collar jobs had slightly lower rates than women whose husbands held blue-collar jobs.

In the British Regional Heart Study, 7735 middle-aged men were followed up for 6 years, during which time 336 men experienced major ischemic heart disease events (fatal or nonfatal myocardial infarction or sudden cardiac death). There were substantial social differences in risk of ischemic heart disease in British men, with the manual-work SES groups having higher ischemic heart disease prevalence and attack rates. Cigarette smoking accounted for most of these differences. Hypertension made only a small contribution to the excess ischemic heart disease in the manual workers. Marked differences in cigarette smoking contributed substantially to the increased risk of ischemic heart disease in manual workers, who also had higher blood pressures, were more obese, and engaged in much less leisure-time physical activity. After smoking and blood pressure were accounted for, there remained an SES gradient in ischemic heart disease calculated to be in excess of 24% ($P=0.08$) in the manual workers.[74]

Psychosocial Factors as Potential Mediators of Social Class and Coronary Disease

A number of psychosocial factors may mediate the relation between SES and coronary disease, including social isolation or social support, coping styles, behavior, job strain or stress, and anger or hostility. Not all of these were reviewed because, for the most part, their status as cardiovascular risk factors is still uncertain. However, one of the newer hypotheses about occupation was thought important enough to include, because occupational status is a part of SES and an emerging literature indicates a connection between occupational characteristics and cardiovascular disease.

Karasek et al[75] suggested that psychological work load and decision latitude on the job may be related to coronary risk. Initially using data from the Department of Labor Quality of Employment Survey, and then from the Health Examination Surveys, 1960 through 1961, and the Health and Nutrition Examination Survey, 1971 through 1975, Karasek and coworkers showed that low decision latitude was associated with increased prevalence of myocardial infarction in both of the latter studies, but the psychological work load and physical exertion were significantly associated with myocardial infarction only in the Health and Nutrition Examination Survey. As a follow-up to the two-dimensional model of Karasek et al for measuring psychosocial work conditions, Falk et al[76] reported that a high relative mortality risk of 1.7 was found among men, born in 1914 and living in Malmo, Sweden, who were exposed to job strain. The combination of job strain and seven different measures of a weak social network and social support was associated with further increased relative risks ranging from 2.1 to 4.6. The authors also found that

social network and social support outside the workplace seemed to buffer the negative health effects of job strain.

Karasek et al[14] also tested the hypotheses of demanding jobs and low decision latitudes as risk factors in a random sample of 1461 Swedish working men. The prospective development of coronary disease symptoms and signs was analyzed using a multivariable logistic regression technique. A case-control study was used to analyze all coronary and cerebrovascular deaths during a 6-year follow-up. The results showed that a hectic and psychologically demanding job increased the risk of developing coronary heart disease symptoms and signs (odds ratio, 1.29; $P<.025$) and premature coronary heart disease or cardiovascular disease death (relative risk, 4.0, $P<.01$). Low intellectual discretion predicted the development of coronary heart disease symptoms and signs (odds ratio, 1.4, $P<.01$), and low personal scheduling freedom among the majority of workers increased the risk of coronary heart disease or cardiovascular disease death (relative risk, 6.6; $P<.0002$). These associations existed after controlling for age, education, smoking, and overweight.

Haan[77] examined the influence of job strain on the incidence of ischemic heart disease in a group of metal industry workers in Finland. Baseline data were collected in 1973 from 902 employees of a metal fabrication company, followed up in 1978 and again in 1983. Extensive questions about job conditions, health status, and a variety of other psychosocial factors were included in the survey. The medical examination included a physical, blood pressure measurement, anthropometric measurements, and assay of serum cholesterol. The measure of job strain had three components: physical strain, variety, and control. Gender-specific age-adjusted ischemic heart disease rates had a positive dose relation with job strain. The age-adjusted rate for women exposed to high strain was 83 per 1000 compared with 48 per 1000 for women exposed to low strain. For men under high strain the rate was 173 per 1000 compared with 90 per 1000 for men under low strain. Thus, the relative risk for high strain compared with low strain was similar in men and women and the risk associated with male compared with female gender was the same for both high-strain and low-strain occupations. The adjusted odds ratios for fatal ischemic heart disease associated with job strain were 4 to 6 after adjusting for blood pressure, relative weight, cholesterol, smoking, and alcohol consumption. This research strongly suggests that the association between job strain and coronary disease is not accounted for by differences in age, sex, cholesterol level, blood pressure, relative weight, or alcohol consumption, and provides more evidence that SES or its components may act as independent risk factors for coronary disease.

The Honolulu Heart Study investigators also tested the hypothesis that men in high-strain occupations have an increased risk of developing coronary heart disease. During an 18-year follow-up study (1965 through 1983) of a cohort of 8006 persons of Japanese ancestry in Hawaii, they found no significant associations between the incidence of coronary heart disease and the individual components of high psychological demands and low job control or for the high-strain interaction of these two characteristics. In fact, there were trends of association opposite to that predicted by the job-strain model that were of borderline significance in multivariate analyses.[78]

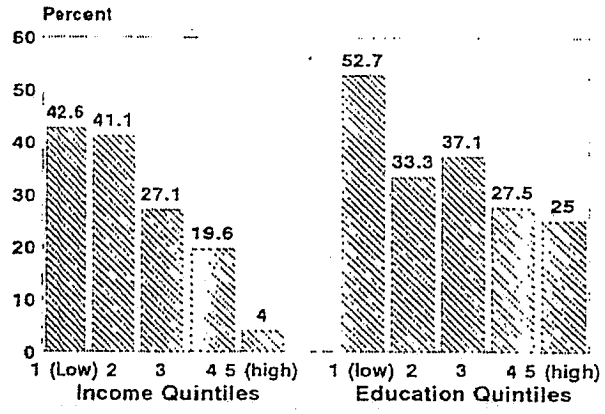

FIG 11. *Percent of state economic areas with late onset (after 1968) of decline in cardiovascular mortality. From Wing et al.*[24]

Associations of Socioeconomic Status With the Decline in Cardiovascular Disease Mortality

Decline in coronary disease mortality. Feldman et al[37] demonstrated that secular trends in mortality declines were associated with educational level. These authors chose educational attainment to measure SES because it remains relatively constant throughout adulthood. These investigators utilized 1960 data from the Matched Record Study and 1971-1984 data from the First National Health and Nutrition Examination Survey and the Epidemiologic Followup Study. They observed that, although there was little difference in mortality by educational level among middle-aged and older men in 1960, rates among men had declined more rapidly for the more educated than the less educated since that time. Much of this decline was in deaths from cardiovascular disease. This trend resulted in substantial increases in educational differentials in mortality from 1971 to 1984. Among women, however, death rates declined at about the same rate regardless of educational attainment. It was therefore suggested that trends in educational differentials for heart disease mortality in men were responsible for much of the change in all causes of death.

Marmot and McDowall[79] were among the first to point out that there may be widening social inequalities in the decline in coronary mortality. After analysis of vital statistics data from Scotland, Wales, and England, they found that among men in every region of Britain, coronary heart disease mortality declined in nonmanual workers, but only in Wales had there been an appreciable decline in coronary heart disease mortality in manual workers. This decline in mortality during 1970 through 1983 was found to be greater in nonmanual workers than in manual workers and was true for all cause of mortality, lung cancer, and cardiovascular disease. In 1988 (for men[24]) and 1992 (for women[22]), Wing et al provided evidence that the course of the coronary heart disease epidemic in the United States is strongly influenced by socioeconomic development (Fig 11). They determined that areas with the poorest socioenvironmental conditions were two to ten times more

likely to experience late onset of the decline in coronary mortality than areas with the highest levels.

Using data from the US Veterans' Study for 1954 to 1979, Rogot and Hrubec[80] found that the declines in coronary heart disease mortality were greater among those of professional status, persons with high SES scores, and nonsmokers.

Decline in stroke mortality. Stroke mortality began to decline in the early 1900s. It has been postulated that acceleration of the decline in the early 1970s was attributable to increased control of hypertension by use of antihypertensive medications. Using data from three successive national health surveys, Casper et al[81] assessed the decline in stroke mortality in light of the prevalence of hypertension, controlled hypertension trends, and sociodemographic variables, and reported that rates of decline since 1972 were modestly but consistently related to improvements in socioeconomic indicators of education and income.

Jacobs et al,[83] commenting on the work of Casper et al,[81] conclude that the changes in stroke mortality in the United States since 1960 have followed blood pressure changes in the population, influenced to some extent by the use of antihypertensive medications. In addition, other lifestyle factors probably influenced the decline of stroke mortality both directly (decreased use of cigarettes) and indirectly (the effects of decreased sodium and alcohol intake on blood pressure). These authors also believe that improved treatment after stroke, as well as medical care changes that have influenced coronary disease, may have also played a role in the reduced stroke mortality rate.

Survival and Recovery

Although there have been relatively few studies on the effect of SES on survival and recovery from chronic cardiovascular disease and acute events, most provide evidence for an association between SES and survival. Williams et al[83] studied the prognosis of 1368 men and women with angiographically documented coronary artery disease. After statistical adjustment for a severity score based on left ventricular ejection fraction, myocardial damage index, conduction disturbances, mitral regurgitation, history of myocardial infarction and congestive heart failure, number of diseased vessels, and other important prognostic factors, strong associations between survival and family income or the number of people dependent on that income remained. Over an average follow-up of 5 years, those with annual incomes below $10 000 were almost twice as likely to die as those with incomes of $40 000 or more (Fig 12). Ruberman et al,[84] analyzing myocardial infarction survival in men who participated in the Beta-Blocker Heart Attack Trial, found that educational level was associated with survival. In addition, in those who survive acute coronary events, SES appears to be related to postevent functioning as measured by return to work.[85-90]

Trends in the Association Between Social Class and Cardiovascular Disease

The inverse relation between SES and cardiovascular disease is now commonly accepted, but it has not always been the case that cardiovascular rates were higher in the poor. William Osler described angina pectoris as being an affliction of the better-off.[5] Studies conducted in the 1930s and 1940s in the United States and the United Kingdom confirmed higher rates of coronary heart disease in men from higher-SES groups.[91] Between the 1940s and 1960s, there was a reversal in this pattern for men. By 1961 mortality from coronary disease in the United Kingdom was more common in the lower occupational classes than in the higher.[92] There were similar findings in the Evans County Study.[93] It is important to note that these trends are specific for men; for women there has always been an inverse relation between SES and cardiovascular mortality. The reasons for these trends in cardiovascular mortality, and for the strikingly different patterns by gender, are complex and not completely understood.[15,61,92,93]

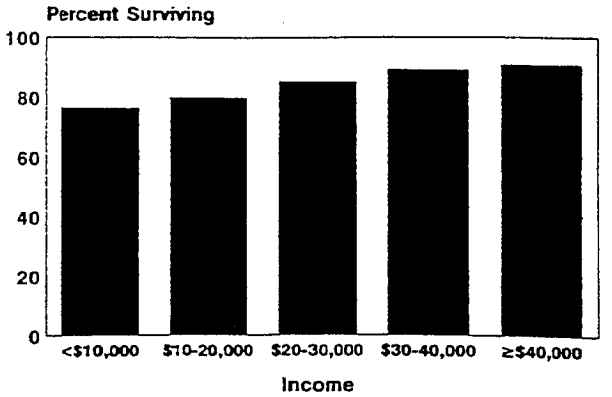

FIG 12. *Five-year survival of patients with coronary artery disease, adjusted for prognostic factors. From Williams et al.*[83]

There is some agreement that the inverse relation between SES and cardiovascular disease has been widening,[23,61,79,94,95] reflecting to some extent the failure of the declines in coronary mortality of the last few decades to reach all segments of society. In analyses of the onset of the decline in cardiovascular mortality, state economic areas with the poorest socioenvironmental conditions (in terms of income, education, and occupation) were two to ten times more likely to have a later onset of the decline in cardiovascular mortality.[24] Similar analyses by Wing et al have indicated that the occupational structure of areas is also related to the timing of secular trends in cardiovascular mortality.

Thus, there is evidence, at least from the United Kingdom and the United States, that there has been a change in the relation between SES and coronary disease from the early 1900s to the latter part of this century.

Socioeconomic Status, Early Life Events, and Coronary Disease

A number of studies suggest that poor living conditions in childhood and adolescence increase the risk of arteriosclerotic heart disease later in life. Forsdahl[25] was the first to suggest that great poverty in childhood and adolescence followed by prosperity is a risk factor for arteriosclerotic heart disease. Based on observations from the Whitehall Study reflecting an inverse relation between height and mortality, Marmot et al[48] suggested that factors operating from early life may influence adult death rates. Notkola et al[96] also tested the hypothesis that bad socioeconomic conditions in childhood increase the probability of coronary heart disease in adulthood. Their findings were based on the data of the

East-West Study in Finland, which was part of the Seven Countries Study. Socioeconomic conditions in childhood were measured by the size of the father's farm; if the father was not alive, similar information for the mother was used. The mortality and morbidity of the cohorts were followed from 1959 to 1974, and risk factors were measured and medical examinations completed in 1959, 1964, 1969, and 1974. The parents of those included in the sample were traced by using parish registers from 1900 to 1919; more than 90% were found and the parents' socioeconomic position during the subjects' childhood was determined. The relative risks of coronary death, myocardial infarction, and ischemic heart disease were systematically increased for those born landless in east Finland. Variables partly explaining the increased risk were body height and smoking.

Kaplan and Salonen[17] conducted a population-based study in Kuopio, Finland, to examine the association between conditions in childhood and ischemic heart disease in 2679 men aged 42, 48, 54, and 60. In this prevalence study, which included extensive examination testing and recall of childhood conditions, low socioeconomic status in childhood was associated with significantly higher prevalence of ischemia during exercise. Compared with those in the highest tertile of childhood socioeconomic conditions, the age-adjusted odds ratio for the subjects in the lowest tertile was 1.44 and for those in the middle tertile, 1.35. The relation was not appreciably weakened by adjustment for years of cigarette smoking or the average number of cigarettes smoked, the ratio of HDL to LDL cholesterol, fibrinogen level, serum selenium concentration, or adult height. The authors suggest that ischemic heart disease develops earlier in those who had a lower SES during childhood.

Barker and Osmand[97] demonstrated a close geographic relation in England and Wales between mortality for ischemic heart disease from 1968 through 1978 and infant mortality rates from 1921 through 1925. The positive correlations between ischemic heart disease mortality and infant mortality rates were remarkably consistent in both sexes, all age groups, and in the different geographical areas studied (Fig 13). The authors suggested that poor nutrition in early life increases susceptibility to the effects of an affluent diet in later life.

The above reports are among the key initial reports. Elford et al[98,99] have completed extensive critical reviews of the influence of early life experiences on cardiovascular disease. Their initial review examined 15 longitudinal and 4 case-control studies on the subject and their second reviewed 10 ecological studies. In their first review, Elford et al[98] found inconsistencies between and within the studies. Their critique suggests that the role of confounding factors was inadequately handled, there were nonspecific relations of varying degrees of strength, and there were no consistent dose-response relations, indicating that the confounding role of persisting social and economic disadvantage throughout life presents epidemiologists with difficult methodological problems. Elford et al believe that epidemiological studies published thus far have failed to support a causal relation between experiences early in life and adult cardiovascular disease.

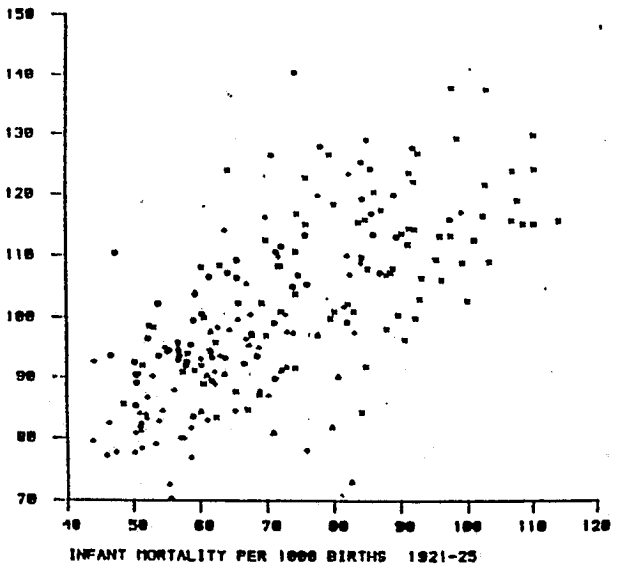

FIG 13. *Standardized mortality ratios for coronary heart disease in men 35 to 74 years old from 1968 to 1978, related to infant mortality in 1921 to 1925 in 212 areas of England and Wales. From Barker and Osmond.*[97]

In their second review, Elford et al[99] pointed out that the ecological studies they reviewed have all described strong dose-related relations between present-day adult cardiovascular mortality and an index of early life experience. The authors' concerns about the ecological studies are the same as for the longitudinal and case-control studies because they believe that the role of confounding was inadequately treated in most of the articles. They conclude that the relations found in the ecological studies should be interpreted with caution and the hypotheses generated by these studies should be tested in studies based on individuals rather than on groups.

Barker and Martyn[100] have presented data relating weight at 1 year to ischemic heart disease rates in adulthood in the same men. There is a relation between greater weight at 1 year and decreasing ischemic heart disease rates in later life, according to analysis of data from 6500 men born in eight districts of Hertfordshire, England, between 1911 and 1930. In addition, although the data are not conclusive, there appears to be an inverse relation in men aged 59 to 70 between weight at 1 year and fibrinogen levels in adulthood. The authors also found an inverse relation of impaired glucose tolerance in these men with weight at 1 year. There was also an inverse relation between systolic blood pressure in the older men with birth weight. Finally, there was a direct relation between systolic blood pressure and placenta weight in men and women aged 46 to 56.

Barker and Martyn[100] provide three explanations for their findings; first, that birth weight is merely a marker for adverse environmental influences that activate in later life; secondly, that genetic influences that first show themselves in early life as gross failures are revealed in adult life through the occurrence of degenerative diseases; finally, that the relation between retarded growth in early life and risk of adult disease is due to long-term effects on physiology and metabolism imposed by an adverse environment during critical

periods of development. They state that their conclusions do not imply that the environment in adult life is unimportant but that it may explain why the known adult risk factors predict cardiovascular disease in individuals so poorly.

The findings by Barker and Martyn[100] and Kaplan and Salonen[17] justify continued study in this area that could lead to primary prevention at its best.

Socioeconomic Status and Cardiovascular Disease Risk Factors

There is substantial evidence for an inverse relation between socioeconomic status and almost all the cardiovascular disease risk factors, with the possible exception of cholesterol level. Probably most of the evidence for an inverse relation between SES and risk factors has to do with hypertension; there is consistent and substantial evidence that low SES is related to both the prevalence and incidence of hypertension. There seems to be an inverse relation between SES and cigarette smoking, obesity, and some hemostatic factors such as fibrinogen, diabetes, and physical activity. The literature has mostly addressed the influence of socioeconomic indicators on individual risk factors, but some studies have examined multiple risk factors.

Studies of Socioeconomic Status and Multiple Risk Factors

As part of the Canadian Health and the Canada Fitness Survey, adults aged 20 through 69 were examined between 1978 and 1981 to estimate the prevalence of cardiovascular risk factors.[101] Level of education was used as a measure of SES. The prevalence of cigarette smoking, overweight, obesity, elevated diastolic blood pressure, physical inactivity, excessive alcohol consumption, elevated serum cholesterol, and diabetes mellitus and (in women) the simultaneous use of oral contraceptives and cigarettes tended to be higher among men and women with a lower level of education. Jacobsen et al[102] examined the relation between level of education, lifestyle variables, and major risk factors for coronary disease in 12 368 Norwegian men and women. The subjects with the highest level of education tended to be less overweight, smoked less, were more physically active in leisure time, and had food habits assumed to be less atherogenic, (drinking less coffee, using soft margarine and low-fat milk, and eating fruits and vegetables daily) than persons with low levels of education.

Matthews et al[103] investigated the association between educational attainment and biologic and behavioral risk factors for coronary disease in a community sample of 2138 middle-aged women in Allegheny County, Pa. (eligibility criteria reduced the number of participants to 541). The less education the women reported, the more atherogenic their risk profile, including higher systolic blood pressure; higher LDL, cholesterol, apolipoprotein B, triglycerides, and 2-hour fasting glucose levels; higher body mass index; and lower high-density lipoprotein cholesterol level. They were more likely to be smokers, to take little physical exercise, and to consume alcohol less than 1 day a week. Similar associations between educational attainment and risk factors were reported by the 1588 nonparticipants.

The data from the First National Health Examination Survey of the German Cardiovascular Prevention Study, taken from 1984 to 1986, indicate that for most cardiovascular risk factors there is a clear negative association between prevalence and length of school education.[104]

Using data from a sample of 3349 people aged 25 to 74 who participated in cross-sectional surveys conducted by the Stanford Five-City Project between 1979 and 1986, the investigators examined six risk factors: knowledge about health, cigarette smoking, hypertension, serum cholesterol level, body mass index, and height. They found highly significant relations between educational level and the six risk factors in the direction of higher risk among those with lower education ($P<.01$ for all relations.[105]).

In a rural area of New York the effects of socioeconomic indicators on risk factors for cardiovascular disease were investigated by Gold and Franks.[106] After adjustment for age and sex, at least one and typically two of the three dichotomized indexes (manual labor, failure to graduate from high school, and poverty) were associated with an increased prevalence of smoking, obesity, frequent use of salt, high cholesterol consumption, low level of leisure activity, and social isolation.

Hypertension

Numerous studies indicate that there is an inverse relation between SES and high blood pressure. James[107] reviewed psychosocial precursors of hypertension. He focused on changes in mean blood pressure of Third-World populations undergoing modernization and on psychosocial correlates of elevated blood pressure and low SES in black populations in the United States and provided a number of examples to document changes in blood pressure with the process of acculturation. This process and the experience of low SES may have common roots in that both may encompass the physical stresses of a new or low SES environment, and the stresses (eg, catecholamine secretion) that accompany psychological stress and the coping abilities and hostilities manifested in a hostile environment.

The Hypertension and Detection Follow-up Program was one of the largest community-based hypertension treatment programs undertaken in the United States. Standardized blood pressure measurements, a medical history, and socioeconomic information were obtained from 158 906 adults in 14 communities. Using prevalence data from this study, investigators sought answers to two questions: Is there a relation between SES and the prevalence of hypertension? Do differences in SES account for the racial differences in prevalence of hypertension? Data from the 14 communities were used to examine the relation of education to the well-documented racial differences in prevalence of hypertension. Education was found to be inversely related to hypertension for each race and sex group. Educational differences did not fully account for the observed difference between blacks and whites in hypertension prevalence, because even at the higher educational levels the adjusted prevalence of hypertension remained nearly twice as high in blacks as in whites.[108]

In 1976 Dyer et al[109] described the relation of education to actual blood pressure among 27 033 white and black men and women, aged 25 through 64, in the Chicago Heart Association Detection Project in Industry. A statistically significant inverse relation between educational level and high blood pressure was present

in all groups of whites and could not be accounted for by differences in age, relative weight, and heart rate. Among black men, a significant inverse relation between educational level and blood pressure was found in the younger subjects; for the older men there was an inverse relation, although because of the small number of subjects in this group, it did not achieve statistical significance. For black women there was no clear relation.

In a rural Swedish county, 7986 people aged 25 through 75 in 16 municipalities participated in a health examination and survey. Casual measurements of blood pressure were made after a 5-minute rest period. There was significant variation in mean blood pressure between subjects in different socioeconomic groups and occupations. These differences associated with educational level were more pronounced for women than for men. Workers, especially men and all subjects with less formal education, had the highest mean blood pressure. There were significant differences between socioeconomic groups even after adjustment for age, gender, weight index, smoking, and treatment of hypertension.[110]

Investigators conducted a study in Dakar, Senegal, of employees of an oil company, a print shop, a cotton mill, a tobacco factory, a canning factory, and a hotel. Employees were examined during the company's annual medical examination or during a special survey carried out for the study. This cross-sectional study indicated that there was a significant inverse relation of blood pressure with occupational category and educational level.[111]

Most studies of the relation between SES and blood pressure have been cross-sectional. Keil et al[112,113] examined the 14-year incidence of hypertension in black men and women in the Charleston Heart Study; high and low SES strata were based on education and occupation for the men and on education only for the women. For both men and women the incidence of hypertension was significantly higher in study participants with lower educational level or lower SES.

Sorel et al[114] have suggested that secular trends in increasing levels of education and treatment of hypertension may have changed the nature of the frequently reported inverse relation between SES, as measured by education, and blood pressure. Their study population consisted of persons aged 25 through 74 who were examined in the Second National Health and Nutrition Examination Survey or the Mexican-American portion of the Hispanic Health and Nutrition Examination Survey. After adjustment for body mass index and age, there was not a consistent association between education and blood pressure except for systolic pressure in whites (a negative association) and in black women (a negative association) and for diastolic pressure (a positive association) in Mexican-American women. The authors indicate that selection bias in the Second National Health and Nutrition Examination Survey II and Hispanic Health and Nutrition Examination Survey may explain the disparity between their findings and those in the earlier literature. They also suggest two other explanations: First, difference in the association of education and blood pressure by gender and ethnicity raise questions of the validity of education as an indicator of SES across population subgroups, and second, the documented inverse relation between education and blood pressure may be little more than an artifact of the study design and methods of earlier studies.

Smoking

Although it has not always been the case, there is now an inverse relation between smoking prevalence and SES in most industrialized countries. Using data from the National Health Interview Survey, Pierce et al[115] found a strong inverse relation between level of education and current smoking. In 1987 the prevalence of smoking among those who had not graduated from high school was more than twice that among college graduates (35.7% and 16.3%, respectively). Quitting rates among current smokers were also higher for those with greater education. When the data were examined for the years from 1974 to 1985 it was found that although smoking prevalence had decreased in all educational strata, the decrease had been greater among those with more education: smoking prevalence decreased from 35.3% to 34.2% among those with less than a high school education and from 28.5% to 18.4% among those with a college education or more.[116] Thus, the association between educational level and smoking prevalence actually increased over this period, with those in the lowest education category having a smoking prevalence 27% higher than those in the highest category in 1974 and 86% higher in 1985. Other analyses of the data indicate that this trend has been present at least since 1965.[117]

Blue-collar workers have higher rates of smoking than do white-collar workers.[117-125] For example, in 1986 white-collar men had rates of smoking 13.7% lower than blue collar (nonservice) men (26.4% and 40.1%, respectively).[117] For women the difference was 5.9% (33.9% and 28.0%, respectively). This difference was relatively similar to that observed in 1970 for men (14.2%) but had increased markedly for women (1.6% in 1970).

Smoking prevalence is also related to employment status,[117] income,[122,126] and other measures of SES,[48,74,119,126-128] with lower rates of smoking associated with employment and higher SES.

Cholesterol

There are apparently few published papers that specifically focus on the relation between SES and total cholesterol level; most have focused on the relation with high-density lipoprotein cholesterol level.

In the Rancho Bernardo Study, Kritz-Silverstein et al[129] examined the relation between employment status and heart disease risk factors, including lipid levels, lipoprotein levels, blood pressure, and fasting and postchallenge plasma glucose and insulin levels in 242 women aged 40 to 59 who were followed up between 1972-1974 and 1984-1987. Approximately 47% were employed, primarily in managerial positions; the unemployed women were homemakers. Employed women smoked fewer cigarettes, drank less alcohol, and exercised more than unemployed women, but these differences were not statistically significant. After adjusting for covariates, employed women were found to have significantly lower total cholesterol and fasting plasma glucose levels than unemployed women. The authors concluded that middle-aged women in managerial positions are healthier than unemployed women.

In the Beaver County Lipid Study, which had a 9-year follow-up, 561 young men and women (mean age, 22 years) were initially screened for hypercholesterolemia.[130] Among men, total cholesterol level was 187.7 mg/dL in those with a college education and 181.7 mg/dL in those with less than a college education. Women with more than a college education had slightly lower total cholesterol levels than those with less than a college education. Educational achievement was positively related to high-density lipoprotein cholesterol level in men and women ($P<.01$), and was an independent predictor of high-density lipoprotein cholesterol level in women but not in men.

Baker et al[131] used data from the Caerphilly (n=2445) and Speedwell (n=2347) Heart Disease Studies to examine the relation between the number of known risk factors for ischemic heart disease and SES. Their overall conclusions were that lipid levels and obesity are unlikely to play any part in explaining social differences in ischemic heart disease. They did find marked differences in the hemostatic-related variables from the various SES groups, and the patterns of these were similar in both studies.

Data from the Lipid Research Clinics Program Prevalence Study (1980) indicated that being in a high educational stratum was one of the characteristics associated with higher high-density lipoprotein cholesterol concentration.[132]

In the Second National Health and Nutrition Examination Survey (1976 through 1980), Linn et al[133] reported that for whites high-density lipoprotein cholesterol levels were highest in the highest category of earnings, whereas blacks generally had lower levels of high-density lipoprotein cholesterol with increased earnings. For the poverty index used (total income of the household as the numerator and total income necessary to maintain a family [adjusted by the size of the household, gender and head of the family, and place of residence] as the denominator), ratios of less than 1 were described as below poverty and ratios of 1 or more as at or above poverty. The investigators examined high-density lipoprotein levels by quartile of the poverty index, and found that among whites high-density lipoprotein levels generally increased with higher levels of the poverty index. The mean high-density lipoprotein level of black women was stable for all categories of the poverty index; the pattern for black men was not consistent.

Freedman et al,[134] using data from the Second National Health and Nutrition Examination Survey, focused their examination of high-density lipoprotein cholesterol level on the influence of education and race. They reported that the associations between education and high-density lipoprotein cholesterol levels varied from negative in black men to nearly nonexistent in white men and black women to positive in white women. They found that among adults with less than 9 years of education, mean high-density lipoprotein levels were 6 to 10 mg/dL higher among blacks, but the difference by race was less than 1 mg/dL among adults with at least 16 years of education. There were indications that 20% to 40% of the differences could be accounted for by obesity, alcohol consumption, and other characteristics. The authors conclude that consideration should be given to behavioral characteristics associated with the interaction between race and educational achievement.

TABLE 2. Plasma Fibrinogen (g/l) by Four Socioeconomic Measures in the Kuopio Ischemic Heart Disease Risk Factor Study

Socioeconomic Measure	Mean	95% Confidence Interval
Income Quintile		
Lowest	3.19	3.14-3.25
2	3.03	2.98-3.09
3	2.99	2.94-3.05
4	3.01	2.96-3.06
Highest	2.89	2.83-2.94
Occupation		
Farmer	3.06	3.00-3.12
Blue-collar	3.06	3.02-3.10
White-collar	2.95	2.91-2.99
Education		
Less than elementary	3.06	2.98-3.15
Elementary	3.05	3.02-3.09
Middle school	2.99	2.95-3.03
High school or higher	2.84	2.75-2.93
Material Possessions Quartile		
Lowest	3.12	3.06-3.19
Second	3.02	2.98-3.05
Third	2.99	2.94-3.04
Highest	2.96	2.91-3.02

From Wilson et al (1993).[27]

Hemostatic Factors

Hemostatic factors, which are increasingly being considered in the natural history of ischemic heart disease and stroke,[135-138] are strongly associated with SES. This appears to be particularly true for fibrinogen. In the Kuopio Ischemic Heart Disease Risk Factor Study in eastern Finland, there was a strong and significant inverse relation between fibrinogen and four measures of SES (income, occupation, education, material possessions). Subjects in the lowest SES group had the highest level of plasma fibrinogen, and those in the highest group had the lowest level (Table 2).[27] These relations persisted after adjustment for smoking, alcohol consumption, body mass index, physical fitness, coffee consumption, plasma lipids (low-density lipoprotein, high-density lipoprotein), and prior history of ischemic heart disease, hypertension, or stroke. Similarly, Markowe et al[139] found that employment grade was related to plasma fibrinogen, but levels of factors II, VII, VIII, and X, fibrinolytic activity, antithrombin II level, and platelet aggregation were not. Other studies have also found evidence for a relation between plasma fibrinogen and SES.[140-143]

Diabetes

There have been few studies on the association between measures of SES and incidence, prevalence, or

mortality from diabetes. What data there are suggest an important association, although it may reflect the importance of other factors such as race or ethnicity, diet, physical activity, and obesity, and may be different for insulin-dependent and non–insulin-dependent diabetes. In the National Longitudinal Mortality Study data there is a strong relation between income level and death with diabetes as the underlying cause for white men and women. For men aged 25 years or older, those with an eighth-grade education or less had a standardized mortality ratio of 109 and those with any college education had a standardized mortality ratio of 89. For white women, there was a stronger association: those with an eighth-grade education or less had a standardized mortality ratio of 151 and those with any college education had a standardized mortality ratio of 61. Data from the National Health Interview Study on the prevalence of diabetes show a similar relation of diabetes with income. The prevalence in people with family incomes of less than $10 000 was 136.1 per 1000, almost twice that in people with family incomes of $35 000 or more (68.9 per 1000).[144] Data from the Israeli Ischemic Heart Disease Project[145] and the San Antonio Heart Study[146] also indicated that there was a relation between diabetes and SES. In the Israeli study, 5-year incidence of diabetes in men aged 40 or older was inversely related with level of education. Incidence rates, adjusted for age and area of birth, were 63% higher in those with an elementary school education compared to those with more than a high school education, and rates in those with a high school education were intermediate. Incidence in professionals, technicians, and teachers was lower than in either administrators or laborers. In the San Antonio study, prevalence of non–insulin-dependent diabetes was higher in Mexican-Americans who lived in a barrio than in those who lived in the suburbs and was intermediate for those in an intermediate area.

Obesity

In 1974[147] and again in 1977,[148] Oken and Rimm et al found that among a population of 59 566 female volunteers in the 'Take Off Pounds Sensibly' program in the United States and Canada, there was an inverse relation between SES and obesity. In the more recent reexamination of these data by age, it was found that SES was related to obesity primarily because of its effect on weight change during the child-bearing years. The difference in adult weight (adjusted for height) between the lowest and highest socioeconomic groups was 17.5 pounds. Stern[146] and Hazuda[149] reported that in Mexican-American men increased acculturation was accompanied by a significant linear decline in the incidence of both obesity and diabetes and that SES had no significant effect on either outcome. In Mexican-American women increased acculturation and increased SES were accompanied by statistically significant linear declines in the incidence of both obesity and diabetes. However, the effect of acculturation in women was stronger than that of SES. The results suggest that culturally mediated factors exert a more pervasive influence on obesity and diabetes in Mexican-Americans than do socioeconomically mediated factors. The authors point out that the influence of SES in women cannot be ignored, particularly with regard to obesity.

Helmert et al[150] used data from the First and Second National Health Surveys in Germany to evaluate social inequities in the incidence of hypertension, hypercholesterolemia, cigarette smoking, obesity, and predicted cardiovascular disease mortality. They found that throughout the regions of study SES gradients were strongest for obesity and weakest for hypercholesterolemia.

Body mass index, as a measure of obesity, was examined in the Charleston Heart Study cohort by Stevens et al[151] to determine its influence on mortality. It was predictive of all-cause and coronary heart disease mortality in black men but not in white men. There was a significant ($P<.05$) correlation between body mass index and education in black men who were reexamined in 1963 but not in those who were originally seen in 1960. Low educational level appeared to significantly enhance the predictiveness of the anthropometric measurements (chest, mid-arm and abdominal circumference) for mortality in black men.

Kumanyika[152] provided an epidemiological review of obesity in black women and in the process explored the influence of socioeconomic factors on obesity. In addition to the work of Oken and Rimm et al,[147,148] data from Garn et al[153] and the National Center for Health Statistics[154] were cited to provide additional evidence that the prevalence of obesity among women varies inversely with SES. Work by Garn et al[155,156] is also cited to indicate that female children in low-income families are of similar or slightly less fatness than female children in high-income families; however, low-income women are more likely to be obese after adolescence than those of high income. Kumanyika also pointed out that even after controlling for SES there were still differences in obesity between black and white women. In the First National Health and Nutrition Examination Survey, in each income class black women had age-adjusted mean weights 4.1 to 7.3 kg higher than those of white women.

Physical Activity

Levels of physical activity have consistently been shown to vary by SES, although not always in the same direction for leisure and occupational physical activity. In the 1985 National Health Interview Survey there was a consistent direct relation between physical activity levels and both education and income.[157] The prevalence of physical inactivity in Canadian 20- to 69-year-olds was also related to education level.[101] Rate of inactivity in those with an elementary school education was more than twice the rate in those with more than a secondary education. Matthews et al[103] found that middle-aged women who had an advanced degree expended 56% more energy per week in nonoccupational physical activities than women with a high school education or less (1699.1 kcal/wk compared with 1090.3 kcal/wk), and energy expenditure increased monotonically with educational level. In the German Cardiovascular Prevention Study, the percentage of subjects not physically active for at least 1 hour per week decreased with increasing social class measured by an index that included income, education, and occupation. Those in the lowest social class had increased risk of being inactive four and a half to five times that of subjects in the highest class.[126] Using an SES measure that combined education and income, Holme et al[158] found similar

direct relations between social class and level of both occupational and leisure-time physical activity, but Salonen et al[159] found that lower-SES men in Finland had higher levels of occupational physical activity. Finally, 9-year declines in physical activity were studied in 4025 people in the Alameda County Study.[160] There was a monotonic inverse relation between amount of decline in leisure-time physical activity and level of either education or income.

Socioeconomic Status and Medical Care

That there are strong inverse relations between SES and causes of death that are "not amenable to medical care"[47] suggests that inadequate access to medical care and poorer quality of care do not fully explain the effect of SES on health. Nevertheless, there are indications that access to and quality of primary, secondary, and tertiary care are poorer for people in lower-SES groups, and these variations in access and quality may to some extent be involved in their comparatively poor health. For example, data from the 1985 National Health Interview Survey indicate that the percentage of people who have not had their blood pressure checked in the past year decreases with increasing education and income (1988).[161] For 30- to 44-year-old men, those who had less than 12 years of education were 60% more likely not to have had their blood pressure checked during the last year, and for women the figure was 67%. Men and women with annual family incomes of less than $10 000 were more than twice as likely not to have had their blood pressure checked than those with family incomes of $50 000 or more.

Similarly, Woolhandler and Himmelstein,[162] analyzing 1983 National Health Interview Survey data, found that for middle-aged women residence in a poor area was associated with a 30% higher prevalence of not being screened for hypertension during the last 2 years. They also report that not having any health insurance coverage was associated with a 60% increased risk of not being screened for hypertension during the last 2 years.

A number of studies indicate that having no health insurance is associated with less access to care and poorer quality of care. Woolhandler and Himmelstein[162] found that poorer women who had insurance reported 9.5 ambulatory care visits per year, and those without insurance reported 4.8 per year. In analyses of data collected from a national probability sample of households, it was found that subjects who were less than 65 years old and uninsured had 60% of the visit rate of those with private insurance when there was adjustment for need.[163,164] However, even in a population with health insurance, Brook et al[165] found that lower income was associated with lower quality of care for hypertension in terms of both process and outcome.

Treatment modalities also vary by SES. Wenneker et al[166] reviewed the almost 38 000 hospitalizations for circulatory disorders and chest pain in Massachusetts in 1985. Rates of angiography, bypass grafts, and angioplasty increased with patients' SES. For example, angioplasty was 32% more frequent in those who lived in areas that were in the top tertile of the income distribution compared with those living in areas in the bottom tertile. Insurance status was also strongly associated with utilization of procedures. The rate of angiography was 80% higher, of bypass grafts 40% higher, and of angioplasty 28% higher for those who were privately insured than in those who were uninsured. Rates for patients with Medicaid were intermediate.

These studies, as well as those indicating poorer access and quality of care for African-American patients[167-170] in studies in which SES information was not usually available, strongly suggest that SES is importantly related to access to and quality of preventive care, ambulatory care, and high-technology procedures.

Is Socioeconomic Status an Independent Cardiovascular Risk Factor?

The answer to this question is not simple as it raises a number of methodological and conceptual questions. Because there have been no trials in which people were randomly allocated to a particular SES group, we must rely on observational studies. The usual approach is to examine in a multivariate fashion the net association between SES and a cardiovascular outcome after statistical adjustment for a variety of accepted risk factors for that outcome. From a methodological viewpoint, the results of such an analysis are not without problems. Essentially, this is an analysis of confounding; that is, the association between cardiovascular disease and the SES measure of interest may be confounded by some other risk factor. It is a simple matter to examine the changes in the strength of association between SES and the outcome with and without adjustment for a risk factor or set of risk factors, but the results of such an analysis are determined by more than the "true" state of affairs. Of particular importance is the fact that all of the associations of interest—SES and outcome, risk factor and outcome, and SES and risk factor—are influenced by measurement error. Because these errors can result in a "regression dilution bias" for each of these three associations, the solution to the problem of confounding is not clear. The observed pattern of confounding might reflect either an underestimate of the confounding effect due to greater measurement error in the confounder or an overestimate due to greater measurement error in the SES measure.

In addition, if changes in risk factors are related to SES, the resultant misclassification of exposure may weaken the association between the risk factor and the outcome, thereby weakening its contribution to the association of SES with that outcome.

Even more important, although decreases in the strength of the association between a measure of SES and a cardiovascular outcome with adjustment for accepted risk factors might indicate that SES is not an independent predictor, there is another possible interpretation. The same observation would be consistent with the role of SES as an antecedent, causal variable that is involved in the adoption and maintenance of these risk factors. In other words, if SES is an important factor in determining the levels of other risk factors, it remains an important independent risk factor, despite the statistical evidence for confounding.

Our review of the literature included a number of studies relevant to the general question of confounding, although the larger issue of independence of risk factors is not addressed in these studies. The best known of these studies is the Whitehall Study,[48] in which a substantial gradient in 10-year risk of cardiovascular

events was negatively related to employment grade in British civil servants. Although prevalence of smoking, high blood pressure, physical inactivity, obesity, and other risk factors were higher in the lower employment grades, these differences did not explain the increased risk. Men in the lowest grade had 2.7 times the 10-year risk of coronary heart disease death of those in the highest grade (Fig 9). When a multiple logistic model was used to adjust for smoking, systolic blood pressure, plasma cholesterol level, blood glucose level, and height, the relative risk was reduced by less than a fourth, to 2.1. Therefore, a considerable relation between SES and coronary heart disease risk remained after adjustment for accepted risk factors.

In similar analyses of the National Health Examination Follow-up Study, the association between educational level and 10-year risk of a coronary event was examined. With adjustment for age, systolic blood pressure, serum cholesterol level, cigarette smoking, diabetes, body mass index, and alcohol, the strength of the association between educational level and risk of coronary event was reduced by 19% for men and 30% for women, remaining statistically significant for both groups.[171]

Studies from Finland,[46] Norway,[158] Sweden,[172] and the Netherlands[45] have reported similar findings. For example, the age-adjusted association between low occupational class and nonfatal myocardial infarction in men in Gothenberg[172] was reduced by only 17% after adjustment for systolic blood pressure, serum cholesterol level, body mass index, heart rate, smoking, diabetes, history of coronary heart disease, leisure-time physical activity, alcohol abuse, and other covariates.

Overall, keeping in mind the complexities discussed at the beginning of this section, it does appear that the relation between measures of SES and cardiovascular events remains substantial when accepted cardiovascular risk factors are considered simultaneously.

Criteria for a Causal Relation Between Socioeconomic Status and Coronary Disease

Socioeconomic status may be a risk factor for coronary disease independent of the other classic cardiovascular disease risk factors. There is a substantial body of evidence showing the inverse relation between SES and coronary disease or its risk factors. With the assumption that SES may be an independent risk factor, the evidence should be examined to determine whether SES meets the criteria for being a risk factor. In 1976 Kuller[4] set forth rules to be adopted in the search for new risk factors for coronary disease. The evidence for independence will be briefly discussed in light of each of Kuller's nine rules or criteria.

1. It should be shown experimentally that any new factor would increase the extent of atherosclerosis or its complications in suitable animal models.

2. Persons with coronary disease would have either a higher risk (if the factor is directly correlated with coronary disease) or lower risk of disease (if inversely correlated with the level of the risk factor) than carefully matched controls. Socioeconomic status meets this criterion because the prevalence of coronary disease has been shown to be higher in groups of low SES than in groups of higher SES.

3. Distribution of risk factors should be correlated with the incidence, prevalence, and mortality of atherosclerotic disease within and between populations. The review of the literature provides abundant evidence for a correlation of SES with the incidence, prevalence, and mortality of coronary disease in the United States, the United Kingdom, Scandinavia, Germany, and other geographic areas. Although some studies have shown a correlation of coronary disease with occupation but not education, in almost every study education and income had an inverse relationship with disease.

4. People exposed to the factor would have a higher risk of coronary disease in longitudinal studies. This criterion has been met by results of the Whitehall Study, the Alameda County Study, the Evans County Study, Georgia Heart Study, the Charleston Heart Study, and the US National Longitudinal Mortality Study.

5. There should be a time-dose relation: the higher the dose the earlier the onset of the disease. Evidence to meet this criterion is sparse except in the relatively few studies that indicated a correlation between SES in early life and coronary disease in adulthood. However, there is obverse evidence from ecological and other studies showing that declines in coronary mortality began first in areas or individuals of high SES.

6. The results of studies should be consistent from study to study, and ideally in different cultural settings. The general inverse relation of SES with mortality and cardiovascular disease risk factors has been quite consistent. However, the results are not yet clear in different ethnic groups such as blacks or Hispanics. Generally the sample size in these studies has been relatively small. Compounding this problem is that in many instances, as for example in the Charleston Heart Study, the range of SES in a random sample of the population is so narrow that it is difficult to detect an effect. In the Charleston Heart Study a special cohort of high-SES black men were compared with a random sample of black men from the community and with white men from the community. Mortality in the high-SES groups over time has consistently been less than the mortality in the random sample of either white or black men. More recent data from the Charleston Heart Study indicate that measures of SES have a strong influence longitudinally in both white and black men.

7. The relation between the risk factor and the disease should be independent of other known risk factors unless it enhances the predictive power of these risk factors. Investigations to meet these criteria have only been reported in the last 10 years and generally show a consistent independent effect of SES on coronary disease. There have not been specific analyses of the interaction of SES with coronary risk factors.

8. Evidence should be available in either humans or a suitable animal model that modification of the risk factor would result in the reversal of the progression of atherosclerosis or clinical disease. Modification of educational level (in this case, knowledge about the risk associated with coronary disease) has been studied in community intervention trials. From these studies at Stanford and in Minnesota; Pawtucket, R.I.; and selected other locations has come evidence that attempts to educate populations about coronary disease and the modification of these risk factors have had minimal success in the experimental communities compared with

control communities. These community intervention studies are subject to the same confounding as the Multiple Risk Factor Intervention Trial in the 1970s: These studies were conducted in a time of generally declining coronary mortality, decline in incidence of coronary disease,[173] and a national health education program by the American Heart Association, the National Institutes of Health, the Centers for Disease Control, and the media in general.

9. The risk factors should make sense in relation to a biological model for cardiovascular disease. There is direct evidence that SES affects behavior styles, coping styles, the endocrine system, the hemostasis system, and access to medical care. The evidence to meet this criterion is probably the most sparse, but future research must identify the psychological, physiological, and biochemical mediators of the relation between SES and disease.

Kuller[4] reported more than 15 years ago that few of the major risk factors met all of the above criteria for a relation with coronary disease. It appears that with additional examination of existing data and additional studies, these nine criteria might be met by SES.

Impact of Socioeconomic Status in Intervention Trials

The Stanford Trials

At least 11 community cardiovascular intervention trials have been conducted or are now ongoing: five in the United States, one each in Finland, Australia, Switzerland, South Africa, and two in Germany.[174] Only reports from the studies in the United States, Finland, and Germany are included in this report.

The Stanford Five-City Project was an outgrowth of the earlier work of the Stanford Heart Disease Prevention Program, for which the Stanford Three-Community Study had first been organized.[174] The investigators hypothesized that a 20% decrease in cardiovascular disease risk would lead to a significant decline in cardiovascular disease event rates in two treatment communities compared with three reference communities.

In an earlier report from the Stanford Three-Community Study, the relation of selected social factors to diet, weight, and plasma cholesterol level were studied in one control and two treatment towns before and after a 3-year bilingual mass media health education program. Spanish-speaking subjects had higher cholesterol and saturated fat intakes than English-speaking participants at baseline even after controlling for the confounding influence of SES. Over the 3 years of the education program all groups reported a 20% to 40% decrease in cholesterol and saturated fat intake. These decreases were as large in low-SES groups as in high-SES groups.[175]

In 1990 Farquhar et al[176] reported on the effects of community-wide education on cardiovascular disease risk factors. A 5-year, low-cost comprehensive program was carried out in two treatment cities (n=122 800). The program included social learning theory, a communication-behavior change model, community organization principles, and social marketing methods that resulted in 26 hours of exposure to multichannel and multifactor education. After 30 to 64 months of education, the authors reported modest but significant net reductions in community averages (compared to those in two control cities [n=197 500]) in plasma cholesterol level (2%), blood pressure (4%), resting pulse rate (3%), and smoking rate (13%). The risk factor changes resulted in estimated reductions in total mortality of 15% and in coronary disease risk of 16%. Farquhar et al provide an excellent public health perspective for their findings with a summary of the total exposures to educational messages through television and radio. They estimate that the organizational and educational program was delivered at a per capita cost of about $4 per year (excluding research costs) and suggest that this expenditure represents only a small fraction of the estimated annual per capita expenditure for cigarettes.

Winkleby et al[177] provided an 8-year follow-up report of trends in blood pressure, smoking, and cholesterol by level of education from 1979 to 1980 through 1985 to 1986 in five communities. Measurements were made through four biennial cross-sectional surveys in two treatment and two control cities. Over the study period men and women from each educational group in the treatment cities experienced significant declines in smoking prevalence and blood pressure, and there was also a significant decline in cholesterol level in men. The authors found that in general, declines were similar in the least educated group (less than high school) and the most educated group (college or postgraduate). Similar trends occurred in control cities.

North Karelia Project

The hypertension program of the North Karelia Project was aimed at lowering high blood pressure levels among the whole population. In 1972 in North Karelia and in the matched reference area a baseline survey was carried out in 6.6% of the population aged 25 through 59. The response rate was 90%. The significant findings of this study were that the decline in mean arterial blood pressure between 1972 and 1977 was significantly greater in the North Karelia target area than in the reference area. Intervention effects were noted in participants not on antihypertensive medication as well as in those on antihypertensive medication at both examinations. There was a slight but nonsignificant difference in blood pressure reduction between high and low SES groups. Although the changes were greater in higher SES groups, the blood pressure reduction was also evident in the lower SES groups. Among participants not reporting antihypertensive medication use, the age-adjusted mean reduction in mean arterial pressure was 4.3 mm Hg in those with low education and 4.7 mm Hg in those of higher education. However, among participants reporting medication use in both 1972 and 1977, the mean reduction in mean arterial pressure was 2.5 mm Hg among the less-educated subjects and 7.0 mm Hg in those with higher education.[178] The number of men on medication at both points in time was small and does not necessarily provide a good comparison of the treatment versus control groups. These latter differences may indicate differential compliance between those of lower and higher education.

The German Cardiovascular Prevention Study

The German Cardiovascular Prevention Study, a multicenter community-based trial with five regions (to be compared with the nation as a reference group), aims at

an 8% reduction of cardiovascular mortality after 7 years of intervention. Midterm results in the city of Bremen, after 3.5 years of behavior-directed intervention, were a significant net reduction of the prevalence of uncontrolled hypertension in both men and women (24% and 35%); a decrease in the prevalence of current smoking (17% in men but no decrease in women); no effect in reduction of body mass; and a significant increase in total serum cholesterol level in women of 2.8%. The multiple logistic function for cardiovascular mortality derived from the United States' First National Health and Nutrition Examination Survey was used to estimate potential mortality. The coefficients showed a potential 9.9% net reduction in cardiovascular disease mortality risk in men and a 16.5% reduction in women. The trends at midterm showed larger net reductions in mortality risk in less-educated people. These changes were due to the equivalent changes in prevalence of uncontrolled hypertension.[179]

The Minnesota Heart Survey

The Minnesota Heart Health Program is a community-based research and demonstration program designed to accelerate population-wide changes in coronary risk factors and disease. The program is being conducted in three pairs of communities in Minnesota, North Dakota, and South Dakota. Six communities are monitored for risk factor levels for a period of 10 years and disease rates are monitored for 10 years or more, starting before the education program begins and continuing for several years after intensive education ends. Three of the communities participate in an education program designed to promote community change in coronary heart disease risk factors and related behavior.[180]

In the Minnesota Heart Survey, levels of coronary heart disease risk factors and behaviors were determined in population-based samples of Minneapolis and St. Paul residents aged 25 through 74 in 1980-1982 and 1985-1987. Education was significantly inversely related to blood pressure, cigarette smoking, and body mass index in summary risk score for both men and women. Improvement in coronary heart disease risk factors over time was unrelated to education or income, suggesting that population-wide factors such as improved health knowledge, availability of health food items, hypertension treatment, and restriction on cigarette smoking are beneficial in all SES groups. Although the SES gradient in risk factors is not increasing, it remains substantial and indicates direction for future prevention efforts (RV Luepker, WD Rosamond, R Murphy, JM Sprafka, AR Folsom, PG McGovern. Personal communication, 1992).

Blackburn[181] has provided an excellent summary of community intervention programs. The first-generation community studies were well received in Finland, the United States, and elsewhere. They showed that communities were highly receptive to well-conceived programs, that changes could be implemented, and that studies were replicable. Although early reports of the intervention program's effects were encouraging, it was unclear whether the educational and motivational strategies were themselves sufficiently powerful to induce, or the evaluation strategies sufficiently sensitive to detect, a major acceleration of the already remarkable upturn in healthy behavior and downturn in cardiovascular disease risk observed in American communities and elsewhere. A major limitation in the community studies, which Blackburn emphasizes, is the relatively small number of nonrandomized communities involved. He points out that the variability found in average risk factor levels among cluster samples of communities and between the years of intervention and observation was greater than anticipated from the well-known individual variability of risk factor values. He specifically states that "low level correlations of risk factors within clusters led, when extended over many people, to very large 'design effects' which reduced power to detect program effects. Systematic and random errors in measurement reduced power further. The changing composition of American communities created by migration contributed to reduced response rates and exposure to programmes."

According to Blackburn,[181] social learning theory is a key to successful implementation of intervention programs. He also points to the advantages and successes of school-based youth-parent programs in the community, yet he says that because "communities are heterogeneous, media messages must be oriented to specific groups defined by age, sex or other demographic factors. Groups may also require special attention based on their knowledge levels about a health topic." Blackburn cites Rose's prevention paradox,[183] which implies that health promotion may penalize many for the benefit of the few and emphasizes the individual cost of the population prevention strategy.

Health education methodology is a key component of intervention and prevention effects in community intervention studies. Wallack and Winkleby[183] believe that further improvements in the health status of Americans, especially among high-risk subgroups in the population, are necessary. Lefebvre,[184] an authority on social marketing and health promotion, has written that "[s]ocial marketing is a method of empowering people to be totally involved and responsible for their well-being; a problem-solving process that may suggest new and innovative ways to attack health and social problems; is a comprehensive strategy for effecting social change on a broad scale; and it requires careful planning, research, and management to implement effectively."

Hypertension Detection and Follow-up Program

Target populations for study of intervention effects on hypertension in 14 centers geographically dispersed in the United States were identified; in 13, target populations were chosen on the basis of residential area and in one, employment rolls of entire divisions of large industries were used. Three consecutive blood pressure readings were taken with a random zero mercury manometer on all subjects aged 30 to 69, excluding only the bedridden and those confined to institutions. Subjects for whom the average of the second and third diastolic blood pressures was ≥95 mm Hg were referred to the study's local clinical center, where those whose average of the second and fourth diastolic blood pressures were ≥90 mm Hg were randomly assigned by blood pressure strata (90-104, 105-114, and >115 mm Hg) and by clinical center to the stepped-care or referred-care group.[185]

The age- and gender-adjusted 5-year all-cause mortality by educational class, race, and presence of hypertension indicated that in the referred care group there was a strong relation in both whites and blacks between low education and higher mortality rates. In the stepped-care program the 5-year mortality appeared to be only slightly related to level of education. The relative risk for all-cause 5-year mortality varied by education class and care group: for those with less than a high school education, the relative risks were 1.33 (referred care) and 1.18 (stepped care); for those with a high school education, they were 1.0 and 1.0; and for those with more than a high school education they were 0.82 and 1.0.[186]

It appears that good medical care and follow-up in the stepped-care group eliminated much of the effects of SES on 5-year mortality. However, the data indicated a slight excess risk of all-cause mortality in those with the least education.

Summary

- Despite recent declines in mortality, cardiovascular diseases are the leading cause of death in the United States today. It appears that many of the major risk factors for coronary disease have been identified. Researchers are still learning about different modifiable factors that may influence cardiovascular diseases. Socioeconomic status may provide a new focus.
- The principal measures of SES have been education, occupation, and income or combinations of these. Education has been the most frequent measure because it does not usually change (as occupation or income might) after young adulthood, information about education can be obtained easily, and it is unlikely that poor health in adulthood influences level of education. However, other measures of SES have merit, and the most informative strategy would incorporate multiple indicators of SES.
- A variety of psychosocial measures—for example, certain aspects of occupational status—may be important mediators of SES and disease. The hypothesis that high job strain may adversely affect health status has a rational basis and is supported by evidence from a limited number of studies.
- There is a considerable body of evidence for a relation between socioeconomic factors and all-cause mortality. These findings have been replicated repeatedly for 80 years across measures of socioeconomic level and in geographically diverse populations.
- During 40 years of study there has been a consistent inverse relation between cardiovascular disease, primarily coronary heart disease, and many of the indicators of SES. Evidence for this relation has been derived from prevalence, prospective, and retrospective cohort studies. Of particular importance to the hypothesis that SES is a risk factor for cardiovascular disease was the finding by several investigators that the patterns of association of SES with coronary disease had changed in men during the past 30 to 40 years and that SES has been associated with the decline of coronary mortality since the mid-1960s. However, the declines in coronary mortality of the last few decades have not affected all segments of society equally. There is some evidence that areas with the poorest socioenvironmental conditions experience later onset in the decline in cardiovascular mortality.
- A number of studies suggest that poor living conditions in childhood and adolescence contribute to increased risk of arteriosclerosis. Some of these studies have been criticized because of their ecological nature, and others for inadequate control of confounding factors. Nevertheless, the evidence is intriguing and provides a great challenge to the epidemiologist because effective research in this area could lead to very early prevention.
- There is an abundance of literature linking SES with established cardiovascular risk factors. In the few studies of multiple risk factors there has generally been an inverse relation between SES indicators and hypertension, smoking, total cholesterol level, body mass index, excess alcohol use, and sometimes diabetes. There is also evidence for an inverse relation between SES and individual risk factors. The literature is most abundant and striking for the relations of SES with hypertension and cigarette smoking. The data are more limited for hemostatic factors, diabetes, obesity, and physical activity. The evidence for a relation between cholesterol level and socioeconomic status is not consistent.
- Socioeconomic status may be an independent risk factor for cardiovascular disease. Evidence for this must be viewed with reservations because of possible confounding between SES and some other factors. In addition, assessment of this confounding may be influenced by measurement error. That SES may meet some of the criteria for causality for the association of an independent risk factor with cardiovascular disease may admit to the current lack of knowledge of how SES exerts its effects. Keeping in mind the complexities of measurement and confounding, it does appear that the association between SES and cardiovascular events is substantial when accepted cardiovascular risk factors are simultaneously considered.
- In one clinical intervention trial (the Hypertension Detection and Follow-up Program) there was a strong inverse relation between education and all-cause mortality in the referred-care group, but it was virtually eliminated in the stepped-care group, indicating that improved care and hypertension follow-up modified the effect of SES. In community intervention trials in the United States there was no evidence for a strong effect of SES on change in risk factors in either intervention or control communities. In North Karelia, Finland, reduction in blood pressure in the intervention community (compared with the control community) was greatest among high-SES participants. In contrast, in a study conducted in Bremen, Germany, risk reduction was greatest in the less-educated participants.

Implications and Recommendations

Our review indicates that there is a substantial body of evidence for a consistent relation between SES and the incidence and prevalence of cardiovascular disease, secular trends in cardiovascular mortality, survival with cardiovascular disease, the prevalence of cardiovascular risk factors, and access to high-quality care and diagnostic services for cardiovascular disease. Documentation for this relation extends over time and is from many locations. Based on this literature, several implications and recommendations for public education, cardiovas-

cular research, medical care, and public policy have emerged.

Public Education

- Successful intervention to reduce the increased cardiovascular disease risk associated with lower SES will have to be broad based, addressing not only specific cardiovascular disease risk factors but also the societal conditions that lead to the adoption and maintenance of high-risk behaviors.
- Ensure that the targeted audience is involved in developing and implementing the education program.
- Make messages simpler and appropriate to the targeted audience.
- Explore new and appropriate techniques and methods to deliver more effective messages.

Research

- There should be a focus on an understanding of the behavioral, social, biological, and physiological mediators that link SES and cardiovascular disease. Attention should be paid to aspects of study design and measurement that are critical to determining whether the association between SES and cardiovascular disease is independent of other risk factors.
- There is considerable need to better understand the links between economic policy, health care coverage, unemployment, and other economic phenomena and the prevention, incidence, and treatment of cardiovascular disease.
- Specific recommendations could best evolve from a consensus conference of leading social and cardiovascular scientists.
- Measures of SES should be included in all human research on cardiovascular disease, including cross-sectional and longitudinal studies, clinical trials, and community interventions.
- Secondary analyses of existing data sets that contain potential information on SES and cardiovascular disease should be encouraged. Such analyses could use data collected as part of clinical trials, observational studies, risk factor modification trials, or community interventions.
- Further attention should be devoted to the effect of SES on cardiovascular risk factors and pathophysiological changes throughout the lifespan, with a particular focus on clarifying the relative role of SES at various life stages, including the prenatal and early-childhood periods.
- There should be more attention to the multiple ways in which SES affects the natural history of cardiovascular disease and to the potentially different roles of education, income, occupation, and other measures of SES.
- There is a need for further study of the psychosocial and personality patterns associated with different SES groups and of their relations with cardiovascular disease.
- Chronic stress associated with work and other daily aspects of life and its role in cardiovascular disease needs further investigation.
- Further study of SES-related influences on the large geographic differences in cardiovascular disease incidence and secular trends should be encouraged.
- Pilot studies might be undertaken to determine whether modifications of SES are effective for prevention of cardiovascular disease. This may be one of the more formidable tasks to be addressed.
- There should be trials to compare the cost-effectiveness of focused and community intervention trials. Because there is a strong correlation with SES for many diseases, it appears that across-the-board interventions may have the greatest potential for improving the health of a large proportion of the population.
- The interfamilial transmission of those aspects of SES that increase the risk of cardiovascular disease should be studied. Attempts should be made to understand the protective factors that enable some people to stay healthy despite an increased risk due to lower SES.

Medical Care

- The variations by SES in access to care and quality of care for cardiovascular disease need further documentation.
- Preventive services need to be aggressively targeted at lower-SES groups and areas in an effort to reduce disparities in cardiovascular risk between different SES groups.
- Education should be developed to help health care practitioners understand the magnitude of the problem of the SES–cardiovascular disease risk relation and the factors that underlie it.

Public Policy

- Promotion of products associated with increased risk of cardiovascular disease (eg, tobacco and high-fat foods) seems to be targeted toward lower-SES groups. The American Heart Association and other organizations may want to consider policies that might reduce this practice.
- There should be a continuing commitment by the American Heart Association to periodically evaluate issues of SES related to the association's mission. Given their common interest in SES as a risk factor for poor health, voluntary health organizations such as the American Heart Association, the American Cancer Society, the American Lung Association, and others might work together to reduce the ill effects of low SES.
- Evidence for the association between socioeconomic factors and cardiovascular diseases and stroke requires a continued emphasis in the following three public policy areas:
  - *Research*—a continued public policy program is needed to ensure that federally funded research programs at the National Institutes of Health and other federal agencies include investigations into the association between socioeconomic factors and cardiovascular diseases and stroke.
  - *Health care*—the AHA's public policy recommendations for national health care reform are that all Americans have access to basic health care, that preventive health care for low-SES groups is given high priority, and that the AHA participate in the development of guidelines for appropriate patient care.
  - *Disease prevention and health promotion*—there should be a continued emphasis on federal, state, and local policy initiatives that encourage the development, expansion, and implementation of public policy—re-

lated programs to help educate all Americans about the prevention and control of cardiovascular diseases and stroke. This is particularly important in policy initiatives related to tobacco control and diet and health.

Some of these recommendations will be easy to achieve and some will be extremely difficult, requiring large-scale solutions, unspecified in nature and subject to great debate. When the evidence of a problem is so enormous, it is not surprising that the solutions would be complex. Tremendous strides have been made toward reducing the overall burden of cardiovascular disease during the last few decades; the time is now ripe to engage in a similar effort to make sure that this decline in disease is shared by all. It is our hope that this report will help to catalyze such a discussion and effort.

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