American Journal of Public Health

EDITOR
Mervyn Susser, MB, BCh, FRCP(E), DrPH

DEPUTY EDITOR
Mary E. Northridge, PhD, MPH, MT

ASSOCIATE EDITORS
Lawrence J. Fine, MS, MD, MPH, DrPH
Richard Neugebauer, PhD
Zena A. Stein, MA, MB, BCh

CONSULTING EDITOR FOR STATISTICS
Bruce Levin, PhD, MA

EDITORIAL BOARD
Lucie S. Kelly, PhD, RN, FAAN (1995), *Chair*

Hortensia Amaro, PhD (1996)
Sevgi O. Aral, PhD, MS (1995)
Heinz W. Berendes, MD, MHS (1995)
Shirley A. A. Beresford, PhD, MA, MSc (1995)
Lawrence Bergner, MD, MPH (1996)
John G. Bruhn, PhD (1997)
Manning Feinleib, MD, DrPH (1997)
Beatrix A. Hamburg, MD (1997)
Herschel S. Horowitz, DDS, MPH (1995)
Eun Sul Lee, PhD (1997)
Beatrice A. Rouse, PhD (1997)
Victor W. Sidel, MD (1996)
Ellen K. Silbergeld, PhD (1996)
Barbara Starfield, MD (1996)

STAFF
Fernando M. Treviño, PhD, MPH
Executive Editor/Executive Director

Sabine J. Beisler
Director, Publications Services

Mina M. Chung
Managing Editor

Carole Leach-Lemens
Editorial Coordinator

Anne Mattison
Production Editor

Maureen Sheridan
Advertising Manager

Ashell Alston, Charlene Bright, Marilyn Butler, Maura Leonard
Publication Assistants

CONTRIBUTING EDITORS
George A. Silver, MD, MPH
Public Health Policy Forum

Wendy K. Mariner, JD, LLM, MPH
Health Law and Ethics

Elizabeth Fee, PhD
Public Health Then and Now

Hugh H. Tilson, MD, DrPH
Notes from the Field

Gerald M. Oppenheimer, PhD, MPH
Book Corner

July 1995, Vol. 85, No. 7

Editorials

Editorial: Social Determinants of Health—Socioeconomic Status, Social Class, and Ethnicity

There is a striking consistency in the distribution of mortality and morbidity between social groups. The more advantaged groups, whether expressed in terms of income, education, social class or ethnicity, tend to have better health than the other members of their societies. The distribution is not bipolar (advantaged vs the rest) but graded, so that each change in the level of advantage or disadvantage is in general associated with a change in health.

This social patterning of health is important for a number of reasons. The size of the gap between the mortality rates of the most and least advantaged groups gives some indication of the potential for improvement in a nation's health. Identification of the groups who are at greatest risk of poor health can inform sound governance of medical services. Most interestingly, perhaps, the graded relationship between health and social position can suggest hypotheses concerning the etiology of both specific diseases and all-cause mortality. Finally, understanding the causes of social variations in health should lead to intervention strategies which can reduce them.

There is a long tradition of medical and social science interest in this area. Great progress has been made in recent decades through the linkage of data sets containing vital and socioeconomic information, the development of statistical methods for analyzing ordinal and categorical variables, and the introduction of computers. The present issue of the Journal reports two studies[1,2] that used these methods to investigate, respectively, infant mortality and all-cause mortality at ages over 25 years in the United States.

The results of both are broadly consistent with a number of other studies that have been reported recently.[3–7] All of these have found an inverse graded relationship between socioeconomic position and health. Sorlie and colleagues were also able to examine the independent effect on all-cause mortality of several indicators of socioeconomic position. These analyses suggest that employment status and family income are more powerful predictors than level of education, although health selection is clearly a problem, particularly in the case of employment status. Their additional finding, that the mortality risk of Blacks remains higher than that of Whites even after adjusting for other social variables, needs to be interpreted in the light of Singh and Yu's time trend analyses of infant mortality, which show that the size of the Black/White disparity varies across historical time.

While it is clearly fruitful to pursue multivariate and time trend analyses of linked data sets, it is also relevant to ask what these studies tell us about the social determinants of health and what new insights might be further investigated in the future. Both studies, for example, include race and ethnicity as an independent variable. However, while Sorlie and colleagues aggregated all subjects who were categorized as neither White nor Black into an "all other" group, Singh and Yu disaggregated these subjects by race and ethnic origin. The latter approach reveals that the "all other" category is

Editor's Note. See related articles by Singh and Yu (p 957) and Sorlie et al. (p 949) in this issue.

extremely heterogenous. Some groups, such as those of Japanese and Chinese origins, had lower infant mortality rates than do Whites, while others, such as those of American Indian and Puerto Rican origins, had higher rates. This suggests that future studies may benefit from a more detailed examination of race and ethnicity, the number of subjects permitting.

In relation to understanding causes, Sorlie and colleagues recognize that the various dimensions of socioeconomic status are usually strongly correlated. When regression analyses are used to estimate the relative importance of these factors to health, the results will, in consequence, be highly sensitive to misspecification of the predictor variables.[8] The authors also recognize that these socioeconomic factors encapsulate complex information about a person's life. For example, several mechanisms could account for the relationship which they found between length of education and adult mortality.[9] The material and cultural resources of the parental home are strong predictors of a child's educational attainment, so education will be a marker of conditions during childhood, and these could be determining adult health. Educational attainment is also a strong predictor of occupation and labor market position during adulthood, and these could be the major influence on adult health. Third, the level of education might affect receptivity to health education messages, with adult health determined by the likelihood of adopting health-enhancing behaviors and quitting those that are health-damaging. Fourth, personality characteristics such as time-preference or self-efficacy may independently influence both educational attainment and health behavior. Finally, poor health during childhood and adolescence could result in both low educational achievement and impaired adult health. In other words, many causal pathways are plausible, and technical issues limit the infallibility of regression analyses as the means of choosing among them.

In this situation, it is appropriate to use a variety of approaches to understanding the causes of social variations in health. Some years ago, the Black Report[10] suggested that we might benefit from viewing such variations in their wider context. Social variations in health, historical changes in health, and cross-national differences in health may be three instances of the same process by which social factors determine health. The Black Report united two of these instances, the social variation and the historical change, when it reminded us that the socioeconomic mortality gradients have long been maintained against an historical background of falling mortality rates. Singh and Yu's data illustrate this point. Although their use of different data sets cautions against precise comparison, the infant mortality rates of the most affluent and longest educated Whites in the mid-1960s are remarkably similar to the rates of the least affluent and shortest educated Blacks in the late 1980s, although between these dates the infant mortality rates of the most advantaged Whites fell even further. It would be parsimonious if one set of causes could account for both phenomena. A similar challenge is presented by contemporary developments in Europe where socioeconomic gradients within countries have been accompanied by a growing and marked divergence between the national mortality rates of Eastern and Western Europe.[11,12]

Another potentially fruitful approach gives due weight to the concept of social structure and recognizes that this is precisely what societies do: they *structure* the life experiences of their members so that advantages and disadvantages tend to cluster cross-sectionally and accumulate longitudinally. Some of the steps in this process have been demonstrated. Parental disadvantage is associated with low birthweight in the parents' offspring.[13] Low birthweight, in turn, is associated with social disadvantage during childhood and adolescence.[14] The accumulation of disadvantage and risk during the period from birth to early adulthood is associated with disease,[15] risk factors,[16] and health behavior[17] during middle age. In addition, low birthweight appears to be associated with several prevalent chronic diseases in late middle age.[18] One contested[19] explanatory hypothesis is that these associations are mediated by biological programming in utero. Much of this work is based on birth cohorts, and, as these mature, they may provide biologically plausible accounts of the social distribution of disease and death.

The experience of life and its social structuring are not limited to asbestos dust, cigarette smoke, dietary fatty acids, and atmospheric pollutants. The biological processes by which such factors could affect health may appear relatively straightforward, but the present lack of comparable knowledge in respect of psychosocial factors should not lead to their neglect. Occupations characterized by high demands and low control[20] or effort–reward imbalance[21] appear to be associated with poorer health, particularly in relation to cardiovascular disease. Similarly, the combination of stress and social isolation appears to damage health. In the study by Sorlie et al., the data on marital status and mortality, complex as associations are with so changeable a state, could reflect such psychosocial processes. The groups whose members were likely to be living alone had an increased mortality risk compared with those who were married. For most groups, this remained the case after controlling for other social factors and regardless of the reason for their domestic solitude.

A number of areas can thus be identified that are important for future studies of the social determinants of health: monitoring health in terms of the basic dimensions of its social variation, examining it in relation to its wider historical and cross-national contexts, investigating the processes by which hazards and benefits cluster cross-sectionally and accumulate longitudinally, and understanding how psychosocial factors are socially structured and the biological processes by which they affect health. Each of these strands is well under way. The next challenge may be their full integration. □

David Blane
Academic Department of Psychiatry
Charing Cross and Westminster
Medical School
London, England

References

1. Singh GK, Yu SM. Infant mortality in the United States: trends, differentials, and projections, 1950 through 2010. *Am J Public Health.* 1995;85:957–964.
2. Sorlie PD, Backlund E, Keller JB. US mortality by economic, demographic, and social characteristics: the National Longitudinal Mortality Study. *Am J Public Health.* 1995;85:949–956.
3. Goldblatt P. *Longitudinal Study: Mortality and Social Organisation.* London, England: Her Majesty's Stationery Office; 1990.
4. Pappas G, Queen S, Hadden W, Fisher G. The increasing disparity in mortality between socioeconomic groups in the United States, 1960–1986. *N Engl J Med.* 1993;329:103–109.
5. Mackenbach JP. Inequalities in health in The Netherlands according to age, gender, marital status, level of education, degree of urbanisation and region. *Eur J Public Health.* 1993;3:112–118.
6. Guralnik JM, Land KC, Blazer D, et al. Educational status and active life expectancy among older blacks and whites. *N Engl J Med.* 1993;329:110–116.
7. Lahelma E, Huuhka M, Kunst A, et al. Class, education or income? Analyzing

health inequalities among Finnish men and women. Presented at the European Society of Medical Sociology Conference; September 16–18, 1994; Vienna, Austria.
8. Measurement imprecision: ignore or investigate? *Lancet.* 1992;339:587–588. Editorial.
9. Blane D, White I, Morris JN. Education, deprivation and mortality. In: Blane D, Brunner E, Wilkinson R, eds. *Health and Society: Research for Public Health Policy in the New Century.* London, England: Routledge. In press.
10. Black D, Morris JN, Smith C, Townsend P. Inequalities in Health: Report of a Research Working Group (The Black Report). London, England: Department of Health and Social Security; 1980.
11. Bobak M, Feachem RG. Health status in the Czech and Slovak Federal Republic. *Health Pol Plann.* 1992;7:234–242.
12. Bobak M, Marmot MG. East–west health divide and potential explanations. Presented at the WHO Regional Office for Europe's European Health Policy Conference; December 5–9, 1994; Copenhagen, Denmark.
13. Butler N, Alberman E. *Perinatal Problems.* Edinburgh, Scotland: Livingstone; 1969.
14. Bartley M, Power C, Blane D, et al. Birth weight and later socioeconomic disadvantage: evidence from the 1958 British cohort study. *Br Med J.* 1994;309:1475–1478.
15. Mann SL, Wadsworth MEJ, Colley JRT. Accumulation of factors influencing respiratory illness in members of a national birth cohort and their offspring. *J Epidemiol Community Health.* 1992;46:286–292.
16. Kuh DJL, Wadsworth MEJ. Physical health status at 36 years in a British national birth cohort. *Soc Sci Med.* 1993;37:905–916.
17. Kuh DJL, Cooper C. Physical activity at 36 years: patterns and childhood predictors in a longitudinal study. *J Epidemiol Community Health.* 1992;46:114–119.
18. Barker DJP, ed. *Fetal and Infant Origins of Adult Disease.* London, England: British Medical Journal; 1992.
19. Paneth N, Susser M. Early origins of coronary heart disease (the Barker hypothesis). *Br Med J.* 1995;310:411–412. Editorial.
20. Karasek RA, Theorell T, Schwartz JE, et al. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Am J Public Health.* 1988;78:910–918.
21. Siegrist J, Peter R, Junge A, et al. Low status control, high effort at work and ischaemic heart disease: prospective evidence from blue-collar men. *Soc Sci Med.* 1993;31:1127–1134.

Editorial: Ethnicity, Socioeconomic Status, and the 50-Year US Infant Mortality Record

The article by Singh and Yu in this issue of the Journal[1] discusses important issues for public health professionals and needs to be carefully studied. The purposes of their study are "(1) to examine the long-term trends and differentials in infant, neonatal, and postneonatal mortality in the United States from 1950 through 1991 by race and ethnicity, education, and family income; (2) to examine the extent of socioeconomic differentials over time in infant mortality; (3) to examine changes in the race-specific patterns of leading causes of death over time; and (4) to assess the implications of the past and recent trends for the future course of mortality by projecting mortality rates for infants to the year 2010."

This is an impressive agenda! It is not surprising that all these objectives could not be met in the space of one journal article. The extensive amassing and presentation of data on these important matters is invaluable. It is unfortunate that there is not space for an equally desirable analysis of the important impacts and interrelations of time, ethnicity, and social factors on infant mortality and its major components. No one article can answer all needs, but the article by Singh and Yu answers a major need to lay out important data for all of us to think about.

The chief conclusions can be summarized from the Discussion section as follows: (1) despite impressive reductions in overall infant mortality, the Black/White disparity in infant mortality has not only persisted but widened; (2) substantial differences in infant mortality exist across other racial and ethnic groups; (3) inequality in infant survival widened across educational levels between 1964 and 1987; (4) the Black/White disparity in infant mortality also widened across all educational levels; (5) there is no empirical evidence of increasing inequality across income levels in infant mortality; and (6) infant mortality in the US remains higher than that in most other industrialized nations.

This seems a curiously limited set of conclusions, one mostly restricted to a statement of empirical findings. Perhaps there is a reason for this paucity, not because of any sins of the authors, but because they, as all of us do, use oversimplified indices of unfavorable pregnancy outcome as shorthand explanations for or summaries of a complex situation. This aggregation tends to obscure the complexity of the real world and, correspondingly, to fog the conclusions we might reach as to causation and prevention.

Barring occasional revolutions, science proceeds by replacing simpler truths with more complex ones, and the present situation with infant mortality may require data and analyses more complex than those in Singh and Yu. The need for greater differentiation of analytic variables is illustrated and/or underlined by several aspects of their study.

Mortality variables. The prime example here is of the need to differentiate further infant mortality into components. Figures 1 and 2 show important time trends in infant mortality, neonatal, and postneonatal mortality. The emphasis throughout the paper is on infant mortality as such, but careful examination of the two figures shows that the two parts of infant mortality behave quite differently. Moreover, although both White and Black groups show consistent long-term declines in infant mortality, there are striking (and unexplained) differences in the behavior of postneonatal mortality between Blacks and Whites. The ratio of Black to White postneonatal mortality rate *rose* during the 1950s and then declined sharply thereafter so that in recent years the ratio of Black/White postneonatal mortality declined from nearly three to just under two.

Other outcome variables. The outcomes used by Singh and Yu are restricted to mortality and cause of death; thus several other variables used as outcomes (often as a proxy for mortality) are not mentioned except by implication, e.g., "short gestation and low birthweight" is mentioned as a cause of death in Table 5. We clearly need to differentiate the dissimilar epidemiologic behavior of such constructs as low birthweight (variously defined), prematurity, intrauterine growth retardation, etc.

Ethnicity and race. The paper shows clearly that the categorization of race or

Editor's Note. See related article by Singh and Yu (p 957) in this issue.