

Geographic and Socioeconomic Variation in the Onset of Decline of Coronary Heart Disease Mortality in White Women

ABSTRACT

Background. Regional, metropolitan, and socioeconomic factors related to the onset of decline of coronary heart disease (CHD) mortality among White women are reported. Such studies are important for planning population-level interventions.

Methods. Mortality data for 1962 to 1978 were used, to estimate the year of onset of decline. Ecological analyses of socioeconomic data from the US census were used to emphasize structural and organizational aspects of changes in disease, rather than as a substitute for an individual-level design.

Results. Onset of decline of CHD mortality among White women was estimated to have occurred by 1962 in 53% of 507 state economic areas (SEAs), ranging from 79% in the Northeast to 39% in the South. Metropolitan areas experienced earlier onset of decline than did nonmetropolitan areas. Average income, education, and occupational levels were highest in early onset areas and declined across onset categories.

Conclusions. The results provide additional evidence for previously observed geographic and social patterns of CHD decline. Emphasis on structural economic factors determining the shape of the CHD epidemic curve does not detract from the medical importance of risk factors, but underscores the importance of community development to public health improvements. The results are consistent with the idea that the course of the CHD epidemic in the United States has been strongly influenced by socioeconomic development. (*Am J Public Health*. 1992;82:204-209)

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Introduction

Early literature suggested that the decline of coronary heart disease (CHD) mortality began around the mid-1960s in all sociodemographic groups in the United States.¹⁻³ However, declining CHD mortality had been noted in California and New York a decade before the national decline began,^{4,5} and a later study demonstrated evidence of substantial variation in the onset of decline among the 50 states for White women and men.⁶ Our previous work has shown that geographic variation in the onset of CHD decline among White men in the United States was related to the metropolitan and socioeconomic characteristics of areas.^{7,8} Regional, metropolitan, and socioeconomic characteristics related to the onset of decline of CHD mortality among White women aged 35 to 74 are reported here.

In this study we use populations as units of analysis; this design is typically called "aggregate" or "ecological." Ecological studies are generally considered to be inferior substitutes for more powerful individual designs, ideally a randomized experiment, in which information on both exposure and outcome is known for each person.⁹ Although ecological studies are clearly inferior when the purpose of research is to isolate "independent" risk factor-disease associations, they are essential for addressing population characteristics such as epidemic cycles or economic relations. The ecological design is chosen here not as a substitute for a "superior" individual study, but as a tool for investigating population-level determinants of disease that cannot be addressed with individual-level study designs. An understanding of these determinants can help to delineate the public health impact

of policies in areas such as economic development, housing, or agriculture, and their potential for reducing mass disease.

Methods

Individual records of underlying cause of death for all US residents for the years 1962 through 1978 were used. Because the definition of CHD was changed between the seventh (1962 to 1967) and eighth (1968 to 1978) revisions of the International Classification of Diseases (ICD), a group of seventh-revision codes believed to show good comparability with the eighth-revision definition¹⁰ were chosen: 420, 422.1, 440, 441, and 443. The standard eighth-revision definition of 410-413 was used.

CHD deaths for White women aged 35 to 74 were grouped in 5-year age categories within each state economic area (SEA) in the continental United States. SEAs are single metropolitan counties or groups of nonmetropolitan counties. Annual SEA age-specific population counts were derived by linear intercensal interpolation for the 1960s and were available from the Census Bureau for the 1970s.

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Age-adjusted rates for each SEA were computed by the direct method, using the total 1970 US population as the standard. More detailed information about data sources has been published previously.¹¹

Quadratic regression was used to model the annual age-adjusted rates for each SEA.^{6-8,12} In this model the adjusted rate is considered to be a function of an intercept, a slope due to year and a slope due to year squared. SEAs with declining rates over the entire period were considered to have experienced onset of decline in 1962 or earlier. Other SEAs were considered to have experienced onset of decline in the year of the maximum estimated rate from the quadratic regression.

The distribution of the timing of the onset of decline of CHD mortality was examined by geographic region and metropolitan status. The four standard US census region definitions were used: Northeast, South, North Central, and West.⁷ SEAs were classified as metropolitan ($n = 206$) if the component counties were part of a standard metropolitan statistical area with a central city of more than 50 000 or a total population of 100 000 or more. Other SEAs were classified as nonmetropolitan ($n = 301$). Average levels of socioeconomic characteristics from the 1960 and 1970 US censuses were used to characterize each SEA. On the basis of previous work,⁸ we considered three measures: percentage of families with annual income greater than \$10 000, percentage of the labor force employed in white-collar occupations, and percentage of the population aged 25 and above that had completed high school or more. These measures reflect many aspects of individuals and their communities, and are considered here primarily as indicators of socioeconomic development and community resources. For example, high levels of white-collar employment reflect an area's economic base in service, government, and financial industries rather than in heavy manufacturing, agricultural, or extractive industries. White-collar employment, as an indicator of occupational structure, is as much a function of the kinds of jobs provided by a community as it is of the characteristics of the workers. The ecological use of these measures stresses not only the implications of white-collar employment for white-collar workers, but the effect of the occupational structure on health-relevant living conditions—including diet, recreation, housing, working conditions, and medical care—for the entire population.

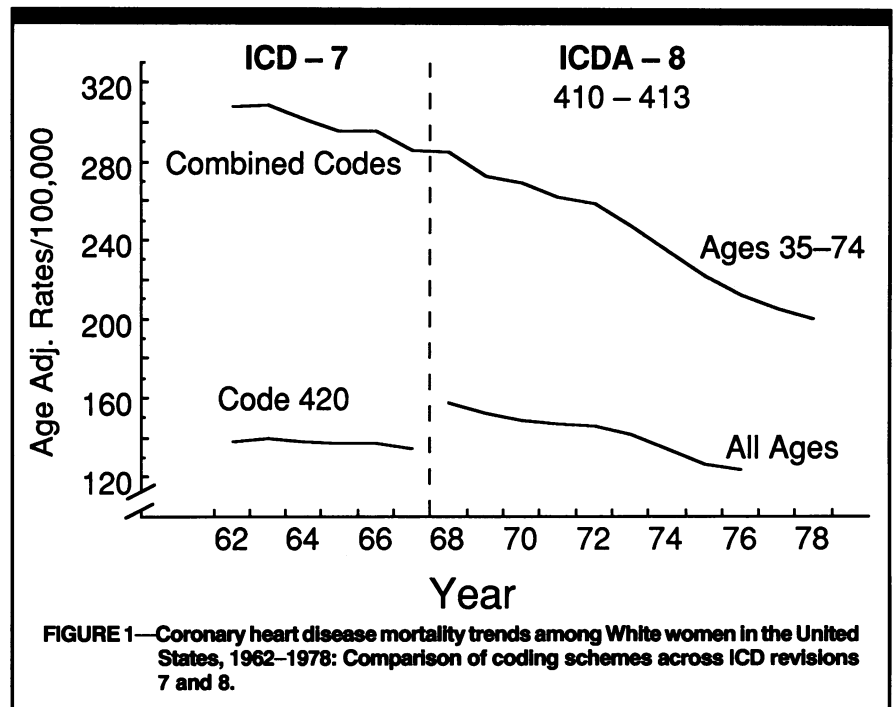


FIGURE 1—Coronary heart disease mortality trends among White women in the United States, 1962–1978: Comparison of coding schemes across ICD revisions 7 and 8.

Results

The comparability of CHD rates when two seventh-revision definitions and the standard eighth-revision definition were used is shown in Figure 1. The bottom line shows published direct-adjusted rates for all ages for ICD-7 code 420.¹⁰ The discontinuity evident in that trend is eliminated by the recoding of seventh-revision data (codes 420, 422.1, 440, 441, and 443) shown in the top line for ages 35 to 74. Similar graphs of the recoded data for individual SEAs showed no evidence of discontinuities due to geographic variation in the comparability of the CHD definitions.

Quadratic regression estimates of the CHD mortality trend within each SEA for the years 1962 to 1978 showed that in 271 (53%) of the 507 SEAs, in which 70% of White women aged 35 to 74 resided, the highest estimated CHD rate occurred in 1962. These SEAs were classified as experiencing onset of decline in 1962 or earlier. Figure 2 shows the cumulative percentage distributions of onset of decline by metropolitan status. CHD rates in 59% of metropolitan SEAs, compared with 50% of nonmetropolitan SEAs, were estimated to have begun to decline by 1962. The earlier timing of the onset of decline in metropolitan areas was maintained through most of the distribution.

Figure 3 shows cumulative percentage distributions of onset of decline by region. The Northeast region contained the largest proportion of SEAs in onset of

decline by 1962, and it remained ahead of other regions throughout the period of onset of decline. The North Central and West regions showed almost identical distributions of onset of decline; the South was later than other regions in the onset of decline. Metropolitan differentials seen for the United States were observed within each region. The more urban composition of the Northeast and the more rural composition of the South, however, did not account for the differences between these regions and the US average: 82% of metropolitan and 74% of nonmetropolitan SEAs in the Northeast were in decline by 1962, compared with 42% and 38%, respectively, in the South. Onset of decline was estimated to have occurred after 1968 in 40 SEAs, of which 35 were in the South. The Appendix gives percentages of SEAs experiencing onset of decline of CHD mortality in different time periods by metropolitan status and region.

Figure 4 shows average levels of 1970 socioeconomic indicators among SEAs grouped into four categories of onset of decline: 1962 or earlier, 1963 to 1965, 1966 to 1968, and 1969 or later. SEAs in the earliest onset-of-decline category show the highest levels of each of the three measures (percentage of families with annual income greater than \$10 000, proportion of the labor force in white-collar jobs, and proportion of the population aged 25 and above with a high school diploma). SEAs in later onset-of-decline categories show progressively lower proportions of the population

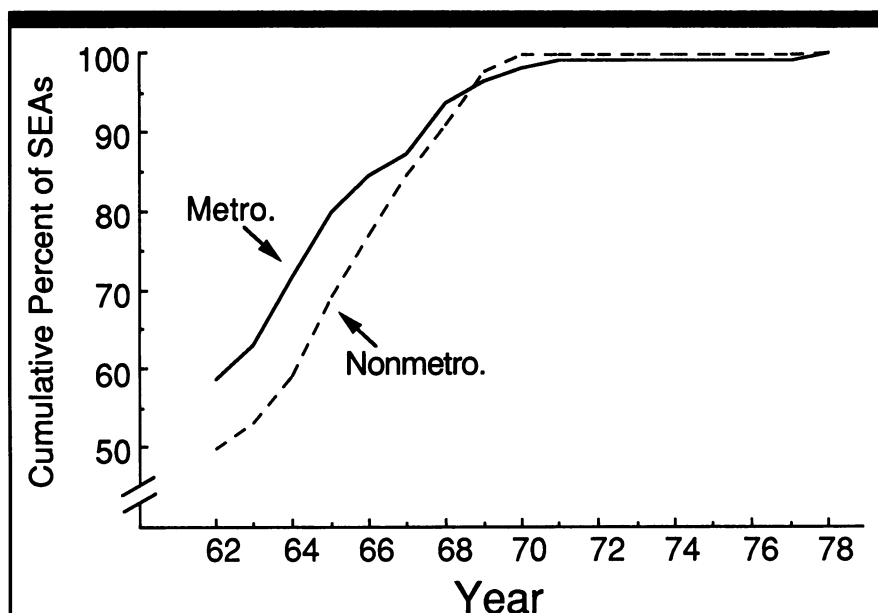


FIGURE 2—Cumulative percentage distributions of onset of decline of coronary heart disease mortality among White women aged 35–74, by metropolitan status.

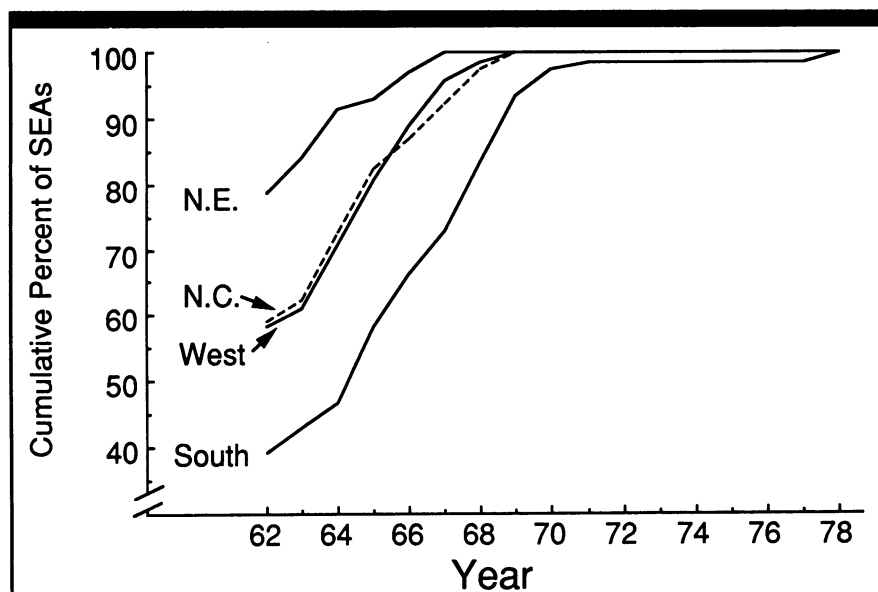


FIGURE 3—Cumulative percentage distributions of onset of decline of coronary heart disease mortality among White women aged 35–74, by region.

with high levels of these indicators. An examination of the average socioeconomic characteristics of SEAs by onset of decline separately within region and metropolitan status categories (data not presented) showed differences in the same direction but of smaller magnitude. Analyses using 1960 census indicators of socioeconomic conditions showed similar results.

The estimated time period of onset of decline of each of the 507 SEAs of the continental United States is shown on the map (Figure 5). Variability in onset of de-

cline is evident in all regions and in most states. Spatial concentrations of early onset of decline are evident in the Northeast and West, whereas concentrations of late-onset areas appear in the South, especially in the area from Oklahoma through the Mississippi Delta to Georgia and in eastern Tennessee and Kentucky.

Discussion

In general, areas in the United States experiencing earlier onset of decline of

CHD mortality among White women showed higher levels of socioeconomic indicators than areas experiencing later onset of decline. These early-onset areas tended to be metropolitan areas and areas outside the South. This pattern of onset of decline is similar to the pattern reported earlier for White men in the United States^{7,8} and also to the pattern reported for White men in the Netherlands, where data for women were not analyzed.¹² The current results provide additional quantitative evidence for the previously observed geographic patterns.

Results of such ecological studies are often interpreted as reflecting differentials occurring among individuals; for example, it may be argued that places have earlier onset of decline because there are greater proportions of individuals of higher socioeconomic status there. Some evidence supports this interpretation for CHD mortality among men^{13,14} and for changes in smoking behavior,¹⁵ an important risk factor for CHD. However, ecological studies remain a poor method of isolating individual risk factor–disease associations, owing to the many potential ambiguities of that interpretation.⁹

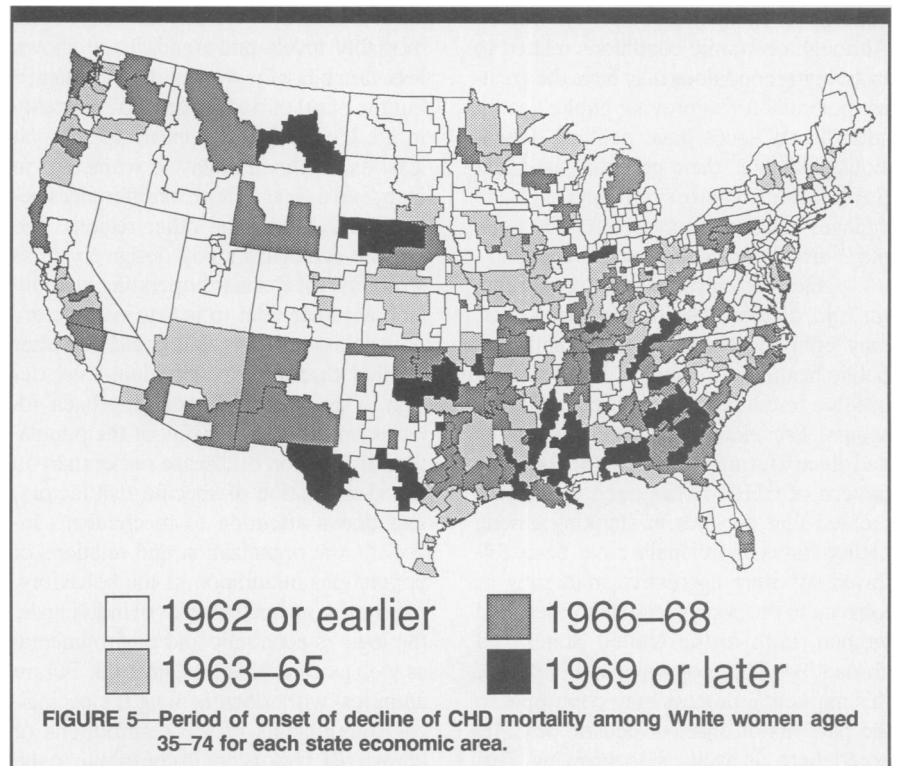
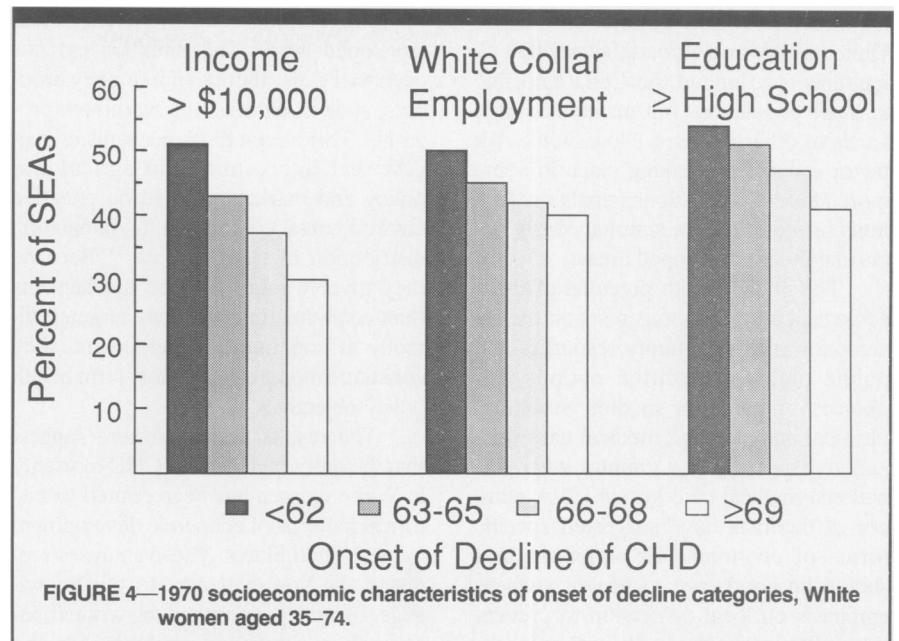
The information used to evaluate the plausibility of findings about risk factor–disease relationships is usually derived primarily from genetic, behavioral, physiological, and clinical disciplines. These epidemiological perspectives emphasize aspects of individuals rather than the organization of individuals in groups, and they focus on the roles of agents (physical, biological, or psychosocial) in the production of disease in individuals. The potential strength of the ecological approach, on the other hand, is in understanding the production of disease in populations viewed not merely as numbers of individuals, but as organized groups with structures and relationships that create health and disease. In order to realize this strength, epidemiological information on risk factors must be combined with perspectives on populations as organized systems. These perspectives are better developed in areas such as ecology, evolutionary biology, history, sociology, and political economy than in molecular and clinical disciplines. An ecological approach to epidemiology can use these perspectives to provide an alternative account of the causes of disease in populations.

Ecological studies are crucial to a public health epidemiology (as it may now be distinguished from a clinical epidemiology) because they suggest mechanisms

of disease production and possibilities for intervention that involve human populations as organized systems.^{16,17} In the case of the study of geographic variation in the onset of decline of CHD mortality, we recognize that areas experience historical economic and social development with profound implications for industrial structure, local wages, educational resources, food supplies, recreational opportunities, and ability to attract medical personnel and facilities. Studies of the mechanisms through which these processes may affect population distributions of risk factors and disease trends are part of developing the ecological view.

In the United States, the Northeast has traditionally been a rich economic area in relation to the poorer South, which has provided raw materials and cheap labor to capital centers of the Northeast.^{18,19} Despite some evidence of regional convergence of incomes during the 1960 to 1976 time period, especially for advantaged workers, a substantial South–non-South gap was still present for blue-collar and less educated workers.²⁰ Additionally, a metropolitan–nonmetropolitan gap in industrial base, occupational structure, wages, and living conditions has developed within the South.²¹ Interestingly, the metropolitan differential in the onset of decline of CHD in White women in the South appears to be smaller than the metropolitan differential in other regions (Appendix); however, the growing inequality of CHD mortality in White men and women between areas classified on the basis of occupational structure is clearer in the South than in other regions of the country.²² As we have noted previously,⁷ factors may affect the onset of decline and the rate of decline in different ways.

The delayed onset of decline of CHD mortality in the South compared with the rest of the United States (Figure 3) is consistent with the South's historical underdevelopment. This "regional colonialism"¹⁸ has been implicated in epidemic patterns of hookworm and pellagra in the early 20th century,^{23,24} and suggests continuity of structural determinants of health during the transition from "old" to "new" diseases as the major public health problems. Such historical continuity of disease causation in populations implies an alternative understanding of epidemiological transition, which has traditionally been seen as involving a parallel change in the causes of health problems and the major diseases of a particular period.²⁵ With respect to understanding and reducing poor



health conditions in the South, the major diseases and disease agents may change while the population-level relations that determine their distribution are maintained. An analogous situation could be described in the experience of epidemiologic transition in some underdeveloped countries.²⁶

When population relationships of development and underdevelopment are stable through epidemiological transition, the association between population charac-

teristics and disease can change dramatically. For example, areas that experienced earlier onset of decline of CHD mortality in the period covered in this study showed a more rapid increase of cardiovascular mortality during the 1930s.²⁷ The epidemic of CHD accompanied rising levels of affluence that brought more atherogenic diets, physical inactivity, and increased cigarette smoking. Ironically, this relative affluence resulted from hard manual labor and lower consumption

by underdeveloped populations in the United States and abroad, factors that simultaneously limited the CHD epidemic in these populations but maintained high levels of other diseases. Now, while risk factor reduction is taking place in some more affluent populations, smaller reductions or increases are simultaneously occurring in less developed areas.

The public health potential of these ecological interpretations rests on the assumption that community resources and public policies condition options and choices of behavior in diet, smoking, physical activity, and medical care utilization (especially in a country without a national medical care system).²⁸ A number of theorists have suggested specific forms of economic development that should be conducive to health, such as emphasis on local ownership to prevent the outflow of profits and to reduce incentives for industries to relocate to take advantage of cheaper labor or materials.²⁹ Although economic conditions related to basic living conditions may have the greatest potential for improving public health, such broad issues have political dimensions that limit their potential as direct public health interventions. Those changes will occur only as a function of a more broadly conceived social agenda.

Other ecological aspects of risk factor and disease distributions, however, may offer more realistic possibilities as public health interventions, because they involve medically recognized disease agents. For example, cigarette smoking and dietary fat are relevant to the epidemic pattern of CHD. It has been well documented that declines in smoking among higher-status individuals have been followed by more aggressive marketing of tobacco to the poor, racial minorities, and women, both in the United States and abroad.^{30,31} The geographic dimension of this marketing process may contribute to the patterns of onset of decline of CHD noted here as well as to growing geographic inequalities in CHD mortality. Empirical analyses documenting geographic variation in trends in tobacco advertising and consumption, if consistent with this interpretation, would support public health policies aimed at controlling tobacco production and marketing, because such interventions would protect against the shifting of consumption from populations in more developed to those in less developed areas.³² A similar situation could be described for production and marketing of high-fat foods, which are being forsaken, to some degree, by the better

educated and are being marketed in less developed areas. This situation extends even to the distribution of free dairy products, such as cheese, in government programs. These ecological mechanisms suggest that interventions in agricultural policy and marketing could be effective environmental controls on the population distribution of heart disease.³³ Because they involve more focused mechanisms than economic interventions targeted directly at community development, they constitute more realistic short-term health policy objectives.

The results presented here suggest that the onset of decline of CHD mortality in White women has been related to historical patterns of economic development in the United States. These analyses represent the first evidence, to our knowledge, of such patterns among women, inasmuch as previous analyses for the United States and the Netherlands excluded women.^{7,8,11} Internationally, CHD mortality levels and trends have shown less variability for women than for men,³⁴ but the onset of decline of CHD mortality in the United States appears to show at least as much variability in women as in men, and a clear pattern of differences between the South and other regions. We use the ecological study design to stress structural mechanisms operating in populations rather than to isolate risk factor-disease associations that could be better studied through an individual-level design. This epidemiological approach focuses on the determinants of the population distribution of disease rather than on the identification of specific risk factors, and draws attention to mechanisms involving the organization and relations of populations in addition to the behaviors, exposures, and pathologies of individuals; the logic is economic and environmental as well as clinical and behavioral. Future empirical work documenting the ecological determinants of the distributions of known risk factors could contribute to the development of preventive measures to create healthier environments. □

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References

- Havlik RJ, Feinleib M, eds. *Proceedings of the Conference on the Decline of Coronary Heart Disease Mortality*. Washington, DC: NIH; 1979.
- Cooper R, Stamler J, Dyer A, et al. The decline in mortality from coronary heart disease, USA, 1968-1975. *J Chron Dis*. 1978;31:709-720.
- Stallones RA. The rise and fall of ischemic heart disease. *Sci Am*. 1980;243:53-59.
- Borhani NO, Hechter HH. Recent changes in CVR disease mortality in California. *Pub Health Rep*. 1964;79:146-160.
- Winkelstein W. Some ecological studies of lung cancer and ischemic heart disease mortality: regional variation. *Int J Epidemiol*. 1985;14:39-47.
- Ragland KE, Selvin S, Merrill DW. The onset of decline in ischemic heart disease mortality in the United States. *Am J Epidemiol*. 1988;127:516-531.
- Wing S, Hayes C, Heiss G, et al. Geographic variation in the onset of decline of ischemic heart disease mortality in the United States. *Am J Public Health*. 1986;76:1404-1408.
- Wing S, Casper M, Riggan W, et al. Socioenvironmental characteristics associated with the onset of decline of ischemic heart disease mortality in the United States. *Am J Public Health*. 1988;78:923-926.
- Morgenstern H. Uses of ecologic analysis in epidemiologic research. *Am J Public Health*. 1982;72:1336-1344.
- Rosenberg HM, Klebba AJ. Trends in cardiovascular mortality with a focus on ischemic heart disease: United States, 1950-1976. In Havlik RJ, Feinleib M, eds. *Proceedings of the Conference on the Decline of Coronary Heart Disease Mortality*. Washington, DC: NIH; 1979.
- Wing S, Casper M, Davis W, et al. Trends in the geographic inequality of cardiovascular disease mortality in the United States, 1962-1982. *Soc Sci Med*. 1990;30(3):261-266.
- Mackenbach JP, Looman CWN, Kuna AE. Geographic variation in the onset of decline of male ischemic heart disease mortality in the Netherlands. *Am J Public Health*. 1989;79:1621-1627.
- Pell S, Fayerweather WE. Trends in the incidence of myocardial infarction and in associated mortality and morbidity in a large employed population, 1957-1983. *N Engl J Med*. 1985;312:1005-1011.
- Feldman JJ, Makuc DM, Kleinman JC, Cornoni-Huntley J. National trends in educational differentials in mortality. *Am J Epidemiol*. 1989;129:919-933.
- Schoenborn CA, Cohen BH. Trends in smoking, alcohol consumption, and other health practices among U.S. adults, 1977 to 1983. *Advance Data 118*, US National Center for Health Statistics; 1986.
- Loomis D, Wing S. Is molecular epidemiology a germ theory for the end of the twentieth century? *Int J Epidemiol*. 1980;19:1-3.
- Illsley R. Comparative review of sources, methodology and knowledge. *Soc Sci Med*. 1990;31:229-236.

18. Perksy J. Regional colonialism and the Southern economy. *Rev Rad Polit Econ*. 1972;5:70-79.
19. Fox K. Uneven regional development in the United States. *Rev Rad Polit Econ*. 1978;10:68-86.
20. Hirschman C, Blankenship K. The North-South earnings gap: changes during the 1960s and 1970s. *Am J Sociology*. 1981;87(2):388-403.
21. Falk WW, Lyson TA. *High Tech, Low Tech, No Tech. Recent Industrial and Occupational Change in the South*. Dworkin AG, ed. State University of New York Press; 1988.
22. Wing S, Casper M, Hayes CG, Dargent-Molina P, Riggan W, Tyroler HA. Changing association between community occupational structure and ischaemic heart disease mortality in the United States. *Lancet*. November 7, 1987;ii:1067-1070.
23. Brown ER. Public health in imperialism: early Rockefeller programs at home and abroad. *Am J Public Health*. 1976;66:897-903.
24. Kunitz SJ. Hookworm and pellagra: exemplary diseases in the New South. *J Health Soc Behav*. 1988;29:139-148.
25. Omran AR. Epidemiology transition in the U.S.: The Health Factor in Population Change. *Population Bull*. 1977;32(2):3-41.
26. Frenk J, Bobadilla JL, Sepulveda J, Cervantes ML. Health transition in the middle-income countries: new challenges for health care. *Health Policy Planning*. 1989;4:29-39.
27. Goves M. Statistical studies of heart disease: IV. Mortality from heart disease (all forms) related to geographic section and size of city. *Pub Health Rep*. 1949;64:439-456.
28. Milio N. Making healthy public policy; developing the science by learning the art: an ecological framework for policy studies. *Health Promotion* 1988;2:263-274.
29. Sclar ED. Community economic structure and individual well-being: a look behind the statistics. *Int J Health Serv*. 1980;10:563-579.
30. Davis RM. Current trends in cigarette advertising and marketing. *N Engl J Med*. 1987;316:725-732.
31. Barry M. The influence of the U.S. tobacco industry on the health, economy, and environment of developing countries. *N Engl J Med*. 1991;324:917-920.
32. Wing S. Social inequalities in the decline of coronary mortality. *Am J Public Health*. 1988;78:1415-1416.
33. Milio N. Promoting health through structural change: analysis of the origins and implementation of Norway's farm-food-nutrition policy. *Soc Sci Med*. 1981;15A:721-734.
34. Uemura K, Pisa Z. Trends in cardiovascular disease mortality in industrialized countries since 1950. *World Health Stat Q*. 1988;41:155-178.

APPENDIX—Distribution of State Economic Areas (SEAs) According to Estimated Period of Onset of Decline of Coronary Heart Disease Mortality, by Region and Metropolitan Status

Region	n	Percentage of SEAs			
		1 1962 or Earlier	2 1963-1965	3 1966-1968	4 1969 or Later
U.S.	507	53.5	20.1	18.5	7.9
Metro	206	58.7	21.4	13.6	6.3
Nonmetro	301	49.8	19.3	21.9	9.0
Northeast	70	78.6	14.3	7.1	0.0
Metro	39	82.1	15.4	2.6	0.0
Nonmetro	31	74.2	12.9	12.9	0.0
North Central	154	59.1	23.4	14.9	2.6
Metro	61	63.9	21.3	13.1	1.6
Nonmetro	93	55.9	24.7	16.1	3.2
South	211	39.3	19.0	25.1	16.6
Metro	79	41.8	21.5	21.5	15.2
Nonmetro	132	37.9	17.4	27.3	17.4
West	72	58.3	22.2	18.1	1.4
Metro	27	63.0	29.6	7.4	0.0
Nonmetro	45	55.6	17.8	24.4	2.2

Note. Thirty-five of the 40 SEAs in category 4 are in the South.