Is Income Inequality a Determinant of Population Health? Part 2. U.S. National and Regional Trends in Income Inequality and Age- and Cause-Specific Mortality

JOHN LYNCH, GEORGE DAVEY SMITH, SAM HARPER, and MARIANNE HILLEMEIER

University of Michigan; University of Bristol; Pennsylvania State University

This article describes U.S. income inequality and 100-year national and 30-year regional trends in age- and cause-specific mortality. There is little congruence between national trends in income inequality and age- or cause-specific mortality except perhaps for suicide and homicide. The variable trends in some causes of mortality may be associated regionally with income inequality. However, between 1978 and 2000 those regions experiencing the largest increases in income inequality had the largest declines in mortality ($r = 0.81, p < 0.001$). Understanding the social determinants of population health requires appreciating how broad indicators of social and economic conditions are related, at different times and places, to the levels and social distribution of major risk factors for particular health outcomes.

Our systematic review (Lynch et al. 2004) showed that links between income inequality and population health are strongest within the United States. Most of this evidence is based on cross-sectional studies of more general outcomes such as self-rated health and total mortality, especially using the income inequality estimates from the 1990 U.S. Census. In this article we compare 100-year national and 30-year regional age- and cause-specific mortality rates with income inequality trends.

Address correspondence to: John Lynch, Center for Social Epidemiology and Population Health, University of Michigan, 1214 South University Street, Ann Arbor, MI 48104-2548 (e-mail: jwlynch@umich.edu).

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Why Examine Long-Term Disease Trends?

The epidemiological identification of major risk factors for disease contributes to improving population health, because it helps explain why certain diseases wax and wane over time in populations and population subgroups. Some researchers have stressed the importance of considering the temporal patterns of relevant exposures and health outcomes (Davey Smith, Ben-Shlomo, and Lynch 2002; Davey Smith and Egger 1996; Leon 2001). Knowledge of risk factors at the individual level might help explain temporal trends in different aspects of population health and how social conditions affect the distribution of those risk factors in the population at a particular time (Davey Smith, Gunnell, and Ben-Shlomo 2001; Davey Smith and Kuh 2001; Kuh and Davey Smith 1993; Leon 2001; Leon and Davey Smith 2000).

These are not new ideas. Techniques like birth cohort analysis have been used for decades to suggest the importance of early life exposures for later disease trends (Davey Smith and Kuh 2001; Kermack, McKendrick, and McKinlay 1934; MacMahon and Terry 1958; Susser and Stein 1962). The need to incorporate our knowledge of individual-level risk factors for disease into the interpretation of overall population health patterns has been a feature of epidemiology from its earliest times (Davey Smith 2002; Frost 1939; Greenwood 1935; Snow 1855). This need was perhaps more relevant to infectious disease than to noninfectious disease, since epidemics clearly increase the risks for both individuals and populations simultaneously.

After World War II, epidemiological studies turned their focus to chronic diseases thought to be noninfectious in origin, thus diminishing the need to triangulate individual and population risks. Some authorities, however, continued to perceive the centrality of such risks (Morris 1957, 1975; Susser 1973). Morris (1975), for example, noted that the then fashionable notion (derived from individual-level studies) that peptic ulcers were caused by stress made no sense when compared with the alternately increasing and decreasing rates of peptic ulcers in Britain over the 20th century. In retrospect it appears that Morris was right, since *Helicobacter pylori* infection was probably the main determinant of population and individual risk in Britain during this period (Marshall 2002). Morris (1975) and others (Krieger 1994; Susser and Susser 1996) commented that it was unfortunate that in the last quarter of the 20th century, epidemiology had turned some of its attention from a

population perspective to a focus on technique and methodology. Reuel Stallones summed up this view in 1980, noting that the recent work in epidemiology had demonstrated a "continuing concern for methods, and especially the dissection of risk assessment, that would do credit to a Talmudic scholar and that threatens at times to bury all that is good and beautiful in epidemiology under an avalanche of mathematical trivia and neologisms" (Stallones 1980b, 69–70).

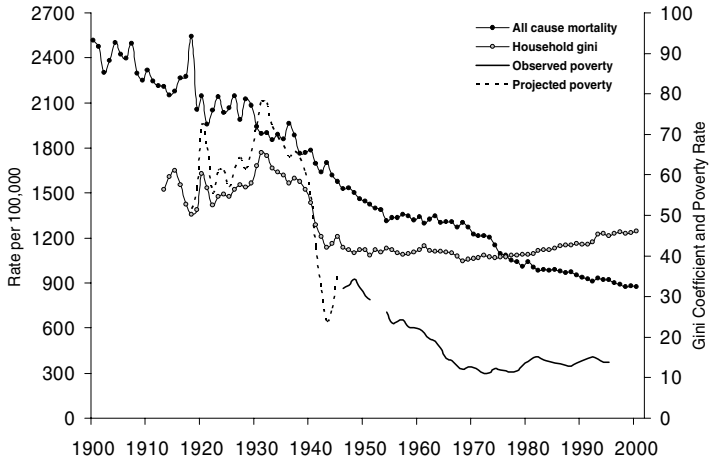
The debates over the major historical and contemporary patterns of population health continue, especially regarding the contributions of different factors to the transition from infectious to chronic diseases in wealthier countries in the 19th and 20th centuries (Colgrove 2002; Link and Phelan 2002; McKeown and Record 1962; Szreter 1988, 1994, 1997, 2002). These include the reasons for the rise and fall of ischemic heart disease (IHD) in many countries (Lawlor, Ebrahim, and Davey Smith 2001; Stallones 1980a; Vartiainen et al. 1994, 1995); the real contribution of traditional risk factors such as smoking, lipids, and hypertension to heart disease and stroke (Beaglehole and Magnus 2002; Greenland, Gidding, and Tracy 2002; Lawlor et al. 2002; Magnus and Beaglehole 2001; Nieto 2002); the rise and fall of the mid-20th-century epidemic of peptic ulcers and their association with *Helicobacter pylori* infection and perhaps interactions with other factors such as stress and diet (Davey Smith 2001; Langman 2002; Levenstein 2002; Marshall 2002; Sonnenberg, Cucino, and Bauerfeind 2002; Susser and Stein 2002); and, most recently, the role of binge alcohol consumption to Russia's mortality crisis (Bobak and Marmot 1999; Malyutina et al. 2002; Shkolnikov, McKee, and Leon 2001). Debates on these matters underscore the potential importance of, but difficulty with, linking our knowledge of causal processes at the individual level with what we observe in population-level trends in health outcomes (Morris 1957; Stallones 1980a; Susser and Stein 2002). Being able to move from the individual to the population level may also help explain how different social, economic, political, and institutional configurations are linked to population health through the distribution of exposure and susceptibility to specific diseases. All of this rests on our understanding the pathobiology of and the risk factors for particular health outcomes.

Both outcome-specific trends and overall population health indicators are important. For example, IHD and stroke are often combined as one outcome—cardiovascular disease—because of the apparent similarities

in risk factors identified from individual-level studies. But when we examine the long-term trends in these two conditions since the turn of the 20th century, they look dramatically different, with a steady decline in stroke—especially hemorrhagic stroke—but a sharp rise in IHD in the 1920s to a peak in the 1960s and, since then, a rapid decline of more than 50 percent. The apparent paradox—that individual-level epidemiologic studies have identified overlapping risk factors for stroke and IHD—has been reconciled by a recent study premised on the different long-term trends in hemorrhagic and ischemic stroke (Lawlor et al. 2002). When the hemorrhagic component of overall stroke was removed, the patterns of ischemic stroke were similar to those for IHD. This result underscores the value of investigating the epidemiologic profile of specific causes of death, even causes that appear closely related but in fact may have different etiological mechanisms and thus follow different trends over time.

There are two lessons here. First, we should take account of the temporal component of the links between risk factors and disease outcomes. This so-called lifecourse approach is now being given more attention (Ben-Shlomo and Kuh 2002; Davey Smith 2003; Hallqvist et al. 2004; Kuh et al. 2003; Kuh and Hardy 2002) and is used for both the individual and the population levels. Second, we should examine outcome-specific trends in addition to trends in overall indicators of population health. Although trends in all-cause mortality, life expectancy, disability-adjusted life years (DALYs), or self-rated health are informative for some purposes, they may also obscure these trends' considerable heterogeneity, which may be used to understand the dynamic and specific linkages among changing environments, particular risk exposures, and different health outcomes associated with specific pathophysiological processes (Davey Smith 2003).

In this article we first describe trends in income inequality in relation to 20th-century U.S. trends in age-specific, heart disease, cerebrovascular, infant, suicide, and homicide mortality rates. We chose these causes of death because heart and cerebrovascular disease are major components of population mortality burden in the 20th century, and because decline in infant mortality is a major component of the improvement in overall life expectancy. Suicide and homicide were chosen because they have plausible causal links with income inequality. Then, second, we examine these and other causes of death in a 30-year U.S. regional trend analysis.



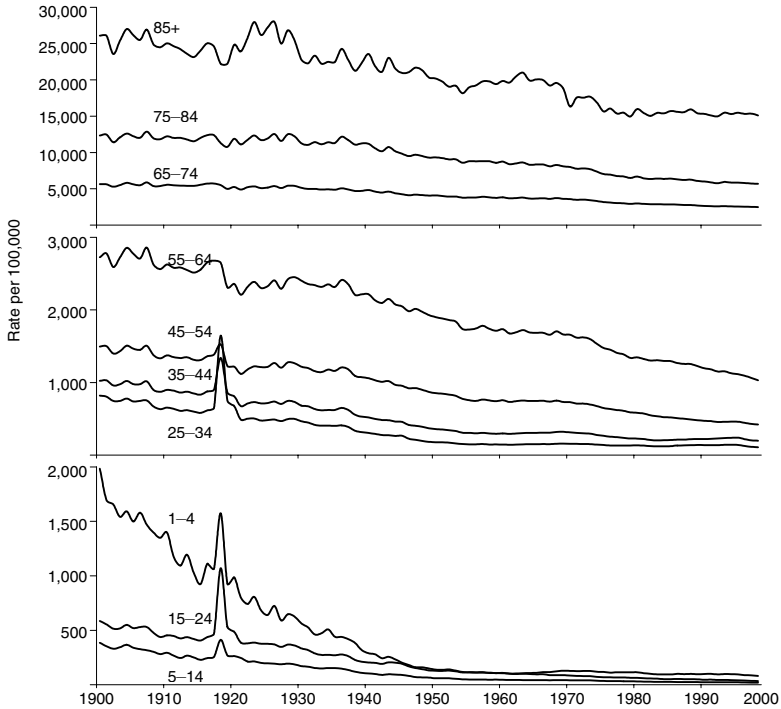
Source: National Center for Health Statistics 2001a; Plotnick et al. 2000.

FIGURE 1. U.S. Household Income Inequality, Poverty, and Age-Adjusted All-Cause Mortality, 1900–1998

U.S. National Cause-Specific Mortality Trends and Income Inequality during the 20th Century

Figure 1 shows U.S. trends in income inequality and poverty in the 20th century. Clearly there have been great changes, the most dramatic of which was the rise in income inequality and poverty before the Depression of the 1930s and then the steep declines through the early 1950s. Figure 2 is a more detailed examination of 20th-century U.S. trends in population health, showing age-specific death rates from all causes, the drop in infant and child mortality (lower panel of the Figure) in the first half of the century, and then the decline in middle age, due largely to less heart disease, in the second half of the century. The large spike in death rates for people under age 54 was due to the influenza epidemic in 1918.

The century-long mortality trends presented here are for the death registration states for 1900 to 1932 and the entire United States from 1933 onward. We recognize that the development of the United States' Vital Statistics System complicated the interpretation of mortality trends



Source: National Center for Health Statistics 2001b.

FIGURE 2. U.S. Age-Specific Mortality from All Causes, 1900–1998

during the early part of the century (Hetzel 1997). That is, the "death registration" areas in 1900 covered some 40 percent of the U.S. population and included several large cities in nonregistration states. In addition, the states in the registration area in 1900 (ten states plus the District of Columbia) covered approximately 26 percent of the U.S. population and overrepresented white, urban America (Woolsey and Moriyama 1948). By 1920 the coverage of registration states increased to 51.4 percent and, in 1930, was 80.4 percent (Hetzel 1997). Official publications note that the expanding registration states are "approximations to complete national rates" that permit general comparisons over time (National Office of Vital Statistics 1956). We know of no systematic analysis, however, of the extent to which the particular development of the registration area may misrepresent the actual mortality rates of the entire nation.

Income Inequality and Heart Disease

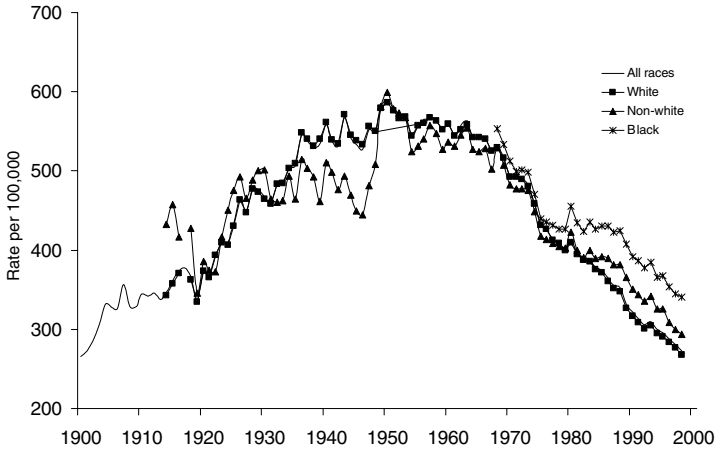
Heart disease is a good place to begin examining disease-specific trends in regard to income inequality. First, it is a major component of total mortality, and so any association between income inequality and total mortality in the second half of the 20th century must strongly reflect an association between income inequality and heart disease. Second, it is probably the most intensely studied disease in human history, and much is known about its causes (Yusuf et al. 2001a, b). Indeed, because diagnoses and definitions have changed over time, we have to use the broad category of "heart disease" here (see Table 1, the International Classification of Diseases [ICD] codes), so it is impossible to examine long-term trends in IHD alone (National Center for Health Statistics 1978). The category of heart disease is composed of a diverse set of pathological entities with disparate causal mechanisms, but it is the only way to provide comparability across time. Included in the category of heart disease are IHD, congestive heart failure, rheumatic heart disease, arrhythmia, hypertensive heart disease, and others. To make things even more complicated, the proportional contributions of these subcomponents have changed over time. Nevertheless, it is reasonable to propose that the largest contributor to the 20th-century epidemic of heart disease was IHD. Smoking, blood lipids, and hypertension have emerged as the three most recognized risk factors for IHD. While all these factors clearly have complex social and biological causes of their own that are worthy of explanation, there is little doubt that they are major contributors to IHD (Beaglehole and Magnus 2002; Magnus and Beaglehole 2001).

Figures 3 and 4 show race- and sex-specific rates of heart disease from 1900 to 1998. In some ways we have come full circle, with the current rates of heart disease now back to the levels observed at the turn of the century—265 per 100,000 persons in 1900 and 272 per 100,000 persons in 1998. However, the composition of the category of heart disease at the beginning of the century was different from that at mid-century or at the beginning of the 21st century. Over the 20th century, IHD became an increasingly important component of all heart disease, with rheumatic heart disease (related to an early-life infection with group A streptococcus) continuously declining. The epidemic of IHD was one of the most prominent features of population health in the 20th century, not just in the United States, but in many other developed nations as

TABLE 1
International Classification of Diseases (ICD) Codes and Revisions, Selected Causes of Death, 1900–present

ICD Revision	Years in Use	Cause of Death				
		Diseases of the Heart	Ischemic Heart Disease	Stroke	Suicide	Homicide
First	1900–09	77–80	. . .	64–66, 82	155–163	176(part)
Second	1910–20	77–80	. . .	64–66, 82	155–163	182–184
Third	1921–29	87–90	. . .	74, 75, 83	165–174	197–200 202(part)
Fourth	1930–38	90–95	94	82	163–171	172–175 198
Fifth	1939–48	90–95	94	83	163, 164	165–168 198
Sixth	1949–57	410–443	420	330–334	E963 E970–E979	E964 E980–E985
Seventh	1958–67	400–402 410–443	420	330–334	E963 E970–E979	E964 E980–E985
Eighth	1968–78	390–398, 402 404, 410–429	410–413	430–438	E950–E959	E960–E978
Ninth	1979–98	390–398 402,404–429	410–414	430–438	E950–E959	E960–E969
Tenth	1999–present	I00–I09, I11 I13,I20–I51	I20–I25	I60–I69	X60–X84 Y87.0	X85–Y09 Y87.1

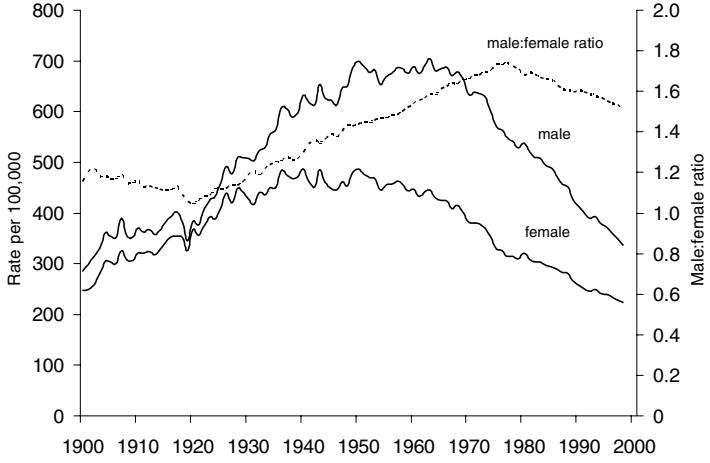
Sources: Anderson et al. 2001; Dunn and Shackley 1944; Faust and Dolman 1963, 1965; Klebba and Dolman 1975; Klebba and Scott 1980; Van Buren 1940.



Source: National Center for Health Statistics 2001a.

FIGURE 3. U.S. Race-Specific Heart Disease Mortality, 1900–1998

well (Beaglehole 1999). While it is clear that traditional risk factors like smoking, a high-fat diet, hypertension, and, more recently, advances in medical care help explain both the rise and fall of the incidence and death rate of heart disease, we still do not know precisely how these



Source: National Center for Health Statistics 2001a.

FIGURE 4. U.S. Age-Adjusted Mortality Rates from Heart Disease by Sex, 1900–1998

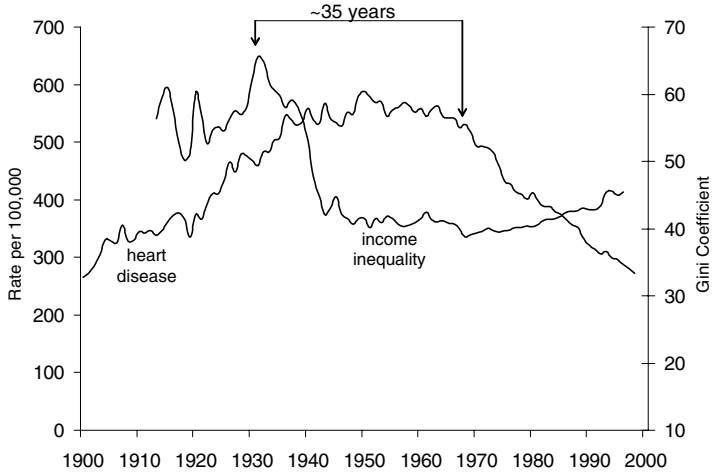
risk factors interacted to generate the 20th-century epidemic of IHD (Kelleher, Harper, and Lynch 2003; Stallones 1980a).

One important feature of Figure 3 is how the drop in the rate of heart disease differed for blacks and whites in the mid-1970s. After almost identical declines from the peak of the epidemic in the 1950s, from 1975 to 1990 the drop in heart disease was significantly slower for blacks than for whites and was the most important component of the difference in blacks' and whites' life expectancy in the 1980s (Kochanek, Maurer, and Rosenberg 1994). Could this be an effect of rising income inequality? Perhaps, but even this is hard to match with income inequality trends, which continued to rise after the 1990s, when the rates of decline for blacks and whites were virtually identical, although blacks continued to be at an overall higher risk of death from this generic category of heart disease. It may be that the failure to adequately control hypertension in African Americans contributed to both their slower decline in IHD and their higher rates of hypertensive heart disease (Cooper et al. 2000).

Figure 4 illustrates the alternately increasing and decreasing sex ratio in heart disease in the United States since the beginning of the epidemic in the 1920s, which has also been documented for other countries (Lawlor, Ebrahim, and Davey Smith 2001). Whatever the combination of factors was, it caused a much more rapid increase in heart disease in men than in women. This result likely reflects different gender distributions and time trends in some of the main risk factors (Lawlor, Ebrahim, and Davey Smith 2001), such as smoking and dietary fat, possibly combined with women's biological predisposition to metabolize saturated fat differently than men do (Lawlor, Ebrahim, and Davey Smith 2002). This means that if income inequality is implicated in these changing sex ratios, there also must be gender differences in the distribution of exposure and/or susceptibility to the main risk factors. Therefore, the rising inequality of income would make the effects of risk factors like smoking, hypertension, and fat consumption more severe in men than in women.

Time Lags

If income inequality is important to determining population health, then the much larger changes from the 1930s to the mid-1940s should have left a population health "footprint." In addition, given the evidence of the

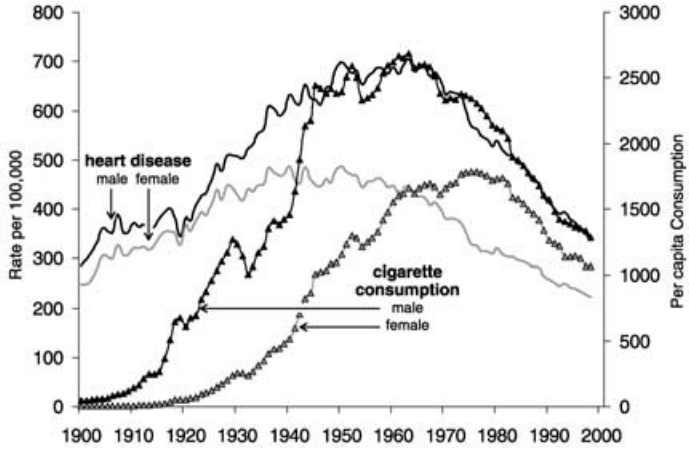


Source: National Center for Health Statistics 2001a; Plotnick et al. 2000.

FIGURE 5. U.S. Age-Adjusted Heart Disease Mortality, 1900–1998, and U.S. Household Income Inequality, 1913–1996

rising inequality in wealth in the late 19th century, it seems reasonable to assume that income inequality also was increasing during the latter part of the 19th century (Lindert 2000; Pope 2000). In regard to heart disease, if we placed a 30-to-35-year time lag on the link between exposure to income inequality and heart disease mortality, we might propose how the rise in income inequality from the late 19th century to its peak in the early 1930s, followed by steep declines until the late 1940s, affected the subsequent rise and fall of heart disease.

Placing a 35-year time lag on the trends in income inequality and heart disease (Figure 5) shows that at least the decline in income inequality fits reasonably well with the decline in heart disease. We do not know precisely what happened to income inequality before 1913, but it probably rose during the growing industrialization starting in the 1850s (Pope 2000; Williamson 1985) and was relatively stable from 1890 to the 1920s, when it peaked during the Depression. This is broadly consistent with the available data on wealth inequality. If that theory is credible, then we might argue that a 35-year time lag fits these heart disease trend data reasonably well. Such a hypothesis, however, would also have to include the mechanisms. Here we should ask whether the long-term trends in income inequality are consistent with our knowledge of trends



Source: Burns et al. 1997; Centers for Disease Control and Prevention 2002; National Center for Health Statistics 2001a; U.S. Department of Agriculture 2001.

FIGURE 6. U.S. Age-Adjusted Heart Disease Mortality and Estimated Annual Adult per Capita Cigarette Consumption, Males and Females, 1900–1998

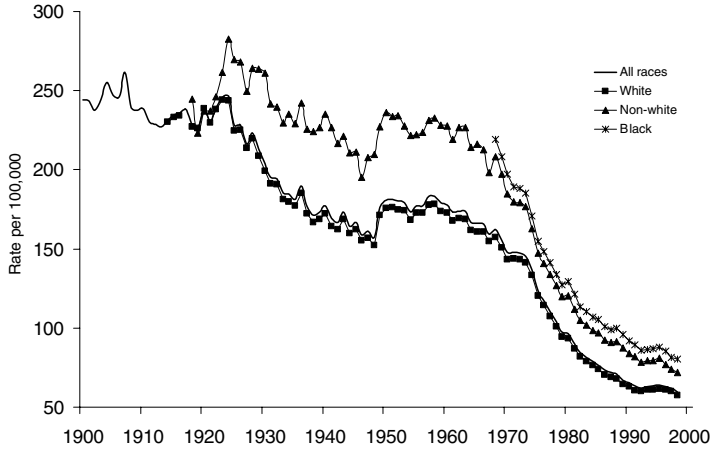
in the main individual-level risk factors for IHD: smoking, lipids, and hypertension.

Based on the individual-level data on sex-specific smoking prevalences by birth cohort (Burns et al. 1997; Escobedo and Peddicord 1996) and the historical data on cigarette smoking (Centers for Disease Control and Prevention 2002; U.S. Department of Agriculture 2001), Figure 6 traces the trends in sex-specific cigarette smoking over the 20th century and compares them with the trends in sex-specific heart disease. For males, these patterns suggest that at the population level, there is little time lag between smoking and heart disease. But the apparent simultaneity of the rise and fall of smoking with heart disease in American men is not evident in all countries. For example, in the United Kingdom, smoking was at its height about ten years before the peak of the heart disease epidemic (Charlton et al. 1997). In the United States there seems to have been little or no time lag between the rapid rise of smoking in the population and the equally steep increase and decrease in smoking and heart disease. This was not the case for women, however, whose peak in heart disease came 25 to 30 years before their peak in smoking. Clearly, we need to examine more closely these sex-specific links between smoking and IHD, especially in

regard to the age in various birth cohorts when smoking began (Glied 2003), the total exposure to smoking, and the age when quitting. The apparent sex difference in the short time lag between smoking and heart disease is less evident for lung cancer. This disease shows a smaller sex difference in the time lag of 40 or more years between the initial exposure of different birth cohorts to high rates of smoking and the subsequent population mortality from lung cancer (Lopez 1995; Wingo et al. 1999).

At the risk of great oversimplification, there are three main IHD processes: through the development of atheroma, thromboembolic processes, or arrhythmia. Smoking not only may affect the development of atheroma but also may operate through the thromboembolic and/or arrhythmic pathways. Thus smoking may affect heart disease almost immediately, given sufficient exposure over a lifetime and perhaps some underlying susceptibility reflected in the presence of vulnerable atherosclerotic plaque—itself associated with blood lipids and hemostatic function. Income inequality may well work through behavioral risk factors like smoking, and smoking certainly maps almost directly onto trends in heart disease in the United States (at least for men). Therefore, the data here suggest that a 30-to-35-year time lag would be needed between population changes in exposure to income inequality and changes in smoking. But this is implausible. In fact, Figures 1 and 6 show that from the end of the Depression through the 1950s, smoking increased most steeply in the population at precisely the same time that income inequality fell most dramatically. While it may be possible to build the case that long-term trends in income inequality contribute to the rise and fall of heart disease, it seems unlikely that such a case could hold up in regard to the major established risk factors for IHD, such as smoking, or the documented cohort-specific declines in population levels of blood pressure (Goff et al. 2001).

The psychosocial explanation of the income inequality hypothesis relies on two pathways in order for income inequality to affect health: health behaviors and stress. We already have shown that trends in income inequality do not easily map onto trends in smoking. Accordingly, we should next examine dietary factors affecting lipids, hypertension, or the stress pathway. However, as Morris (1975) pointed out in regard to the hypothesis that stress causes peptic ulcers, it is difficult to see how a stress pathway could be examined using historical data.



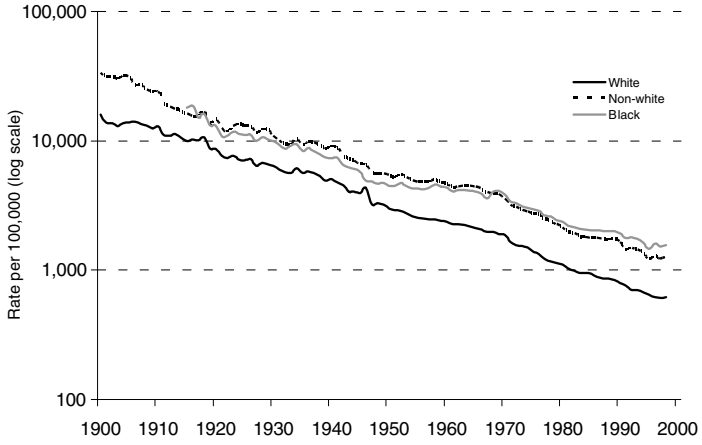
Source: National Center for Health Statistics 2001a.

FIGURE 7. U.S. Age-Adjusted Death Rates by Race per 100,000 for Vascular Lesions Affecting the Central Nervous System, 1900–1967, and Cerebrovascular Disease, 1968–1998

Income Inequality and Stroke, Infant Mortality, Suicide, and Homicide

Figure 7 shows the race-specific rates of cerebrovascular disease over the 20th century, and as with heart disease it is not clear how trends in income inequality could easily map onto the almost continuous declines in stroke over the century. The apparent upward turn around 1950 is probably a result of changes in disease coding, with the shift from ICD-5 to ICD-6 (see Table 1). It also is important to reiterate the limitations of such data, as the class of deaths labeled as strokes is, like heart disease, heterogeneous. As we pointed out earlier, we must be aware of the differences between the hemorrhagic and ischemic components of stroke and their long-term trends (Lawlor et al. 2002). Nevertheless, it is difficult to see how trends in income inequality could directly affect the consistent declines in hemorrhagic stroke during the 20th century. Likewise, the situation for ischemic stroke is much the same as for ischemic heart disease (Shi et al. 2003).

Several cross-sectional studies have shown that income inequality strongly affects infant and child health (Lynch et al. 2004). The reason may be that the time lag between exposure and outcome may be

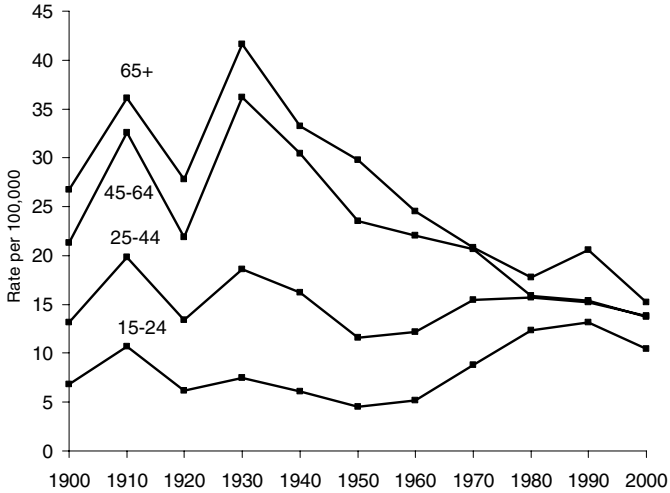


Source: National Center for Health Statistics 2001b.

FIGURE 8. U.S. Infant Mortality by Race, 1900–1998

relatively shorter, and there is good evidence at the individual level that maternal stress, infection, and health behaviors such as nutrition and smoking—before and during pregnancy—are associated with poorer birth outcomes (Kramer et al. 2001). Figure 8 shows 20th-century trends in infant mortality by race. The y-axis is on a log scale to overcome the distorting effect of high infant mortality rates up until the 1920s on contemporary rates. We found no clear link between 100-year trends in income inequality and the inexorable decline in infant mortality in all racial groups over the century. So even for a cause of death that has been strongly linked, in both national and international cross-sectional studies, to income inequality and that may be linked to proposed income inequality mechanisms that do not involve long time lags, there appears to be no simple population-level association between long-term trends in infant mortality and income inequality or in the size of proportional racial disparity in infant mortality over the century.

Suicide and homicide have been implicated as being especially sensitive to income inequality. Here, the 100-year trends in causes of mortality do bear some resemblance to trends in income inequality. Figure 9 shows age-specific suicide trends for the 20th century, with the determinants of suicide likely to differ between the young and the old. The suicide rate for persons older than 45 climbed noticeably during the Depression and since then has fallen almost continuously until today. This rise was

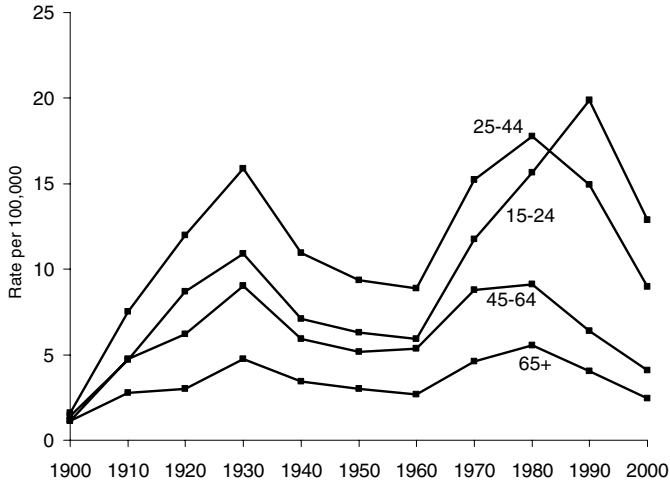


Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003); National Office of Vital Statistics 1956.

FIGURE 9. U.S. Age-Specific Suicide Rates, 1900–2000

less prominent among younger ages and hardly evident at all among adolescents and young adults. In contrast to the pattern among older adults, suicide in the young increased sharply in the early 1960s and peaked during the 1980s and 1990s.

One reading of these data is that the rise and fall in income inequality from the Depression until the end of World War II was reflected in the rise and fall of the suicide rate. Suicide mortality at older ages has dropped continuously since the mid-1930s, perhaps as a result of the simultaneous declines in income inequality and old-age poverty and the beneficial effects of programs like Social Security. The pattern for suicide at younger ages shows an initial decline but later increases and could be consistent with the link between the rise in income inequality in the 1970s and the changes in the age-distribution of poverty (Corcoran and Chaudry 1997; Lobmayer and Wilkinson 2000). The increases in income inequality since the early 1970s have fallen disproportionately on the young and families with children (Rainwater, Smeeding, and Coder 2001). But the suicide rate for younger ages already was climbing in the early 1960s, somewhat before the documented rises in income inequality. Thus, while a concordance between trends in income inequality and



Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003); National Office of Vital Statistics 1956.

FIGURE 10. U.S. Age-Specific Homicide Rates, 1900–2000

suicide may be suggested, it is not clear that they can be separated from the impact of other social changes and policies, like poverty reduction and Social Security, on suicide at older ages or that the timing of the increase in income inequality is consistent with the higher suicide rate for younger persons. Instead, this superficial evidence more likely reveals the effect of income inequality, perhaps in combination with other factors, on the suicide rate.

Figure 10 shows age-specific homicide trends for the 20th century and also provides some possible evidence for the effect of income inequality, at least on the coincidence of its trends with increases and decreases in the homicide rate from the Depression until the end of World War II. Waldron and Eyer (1975) argued that the relative income of young men—the ratio of their median income to their parents' median income—was important to understanding the rising death rates due to suicide and homicide among young males during the 1960s. However, the increases in homicide at all ages during the 1960s appear to predate the general rise in income inequality in the 1970s, and the dramatic falls in the mid-1980s are inconsistent with the sustained increases in income inequality starting in the 1970s. These trends in the suicide and homicide rates should be investigated further and offer an interesting possibility

for the more direct influence of income inequality on important aspects of population health.

U.S. Regional Trends in Age- and Cause-Specific Mortality and Income Inequality, 1968 to 1998

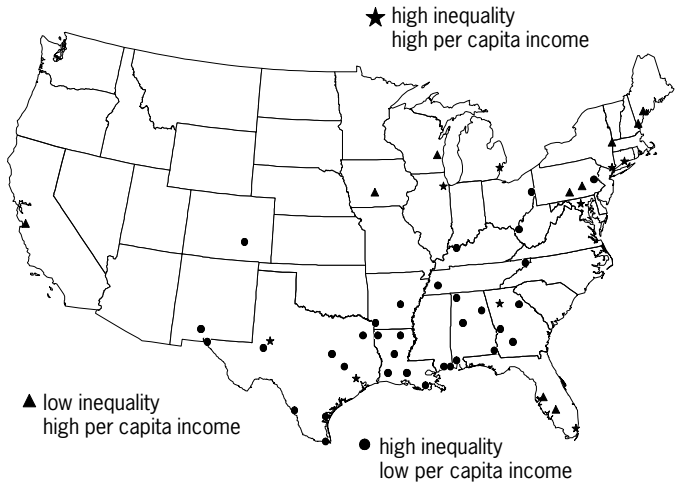
Next we examine whether the national trends mask regional differences in changes in income inequality and its effects on mortality trends. In regard to the changes in mortality between the 18th and 19th century, Johansson and Kasakoff (2000, 58) explained, "In time-series research, national life-expectancy trends over time are most misleading when local trends follow a jumble of divergent paths, which, when aggregated, create a national trend that is not followed by most local populations." In addition, looking at 30-year regional trends minimizes the comparability problems encountered in examining 100-year trends in outcomes such as heart disease, whose definitions have changed over time. One limitation of our analyses may be that the use of regional trends requires a high level of aggregation that may mask more local deviations from these trends. In the future, research on the effects on trends in smaller areas may be helpful.

Aggregate studies of U.S. states show an apparently strong regional pattern in the link between income inequality and mortality (Kaplan et al. 1996; Kennedy, Kawachi, and Prothrow-Stith 1996), in which the most unequal and the highest mortality states were predominantly in the southern United States (Figure 11). Similar patterns were found in metropolitan areas. Figure 12 illustrates those metropolitan areas receiving both lower average incomes and higher income inequality. According to the 1990 U.S. Census, these areas were mainly in the southern United States. The importance of such regional variation is supported by analyses indicating that controlling for census region removes the effects of state-level income inequality on health (Mellor and Milyo 2002).

There is evidence of a regional component in the overall link between mortality and income inequality, in that the southern states and metropolitan areas have lower average incomes, higher income inequality, and generally higher mortality rates. Regional differences also appear in the strength of the association between income inequality and

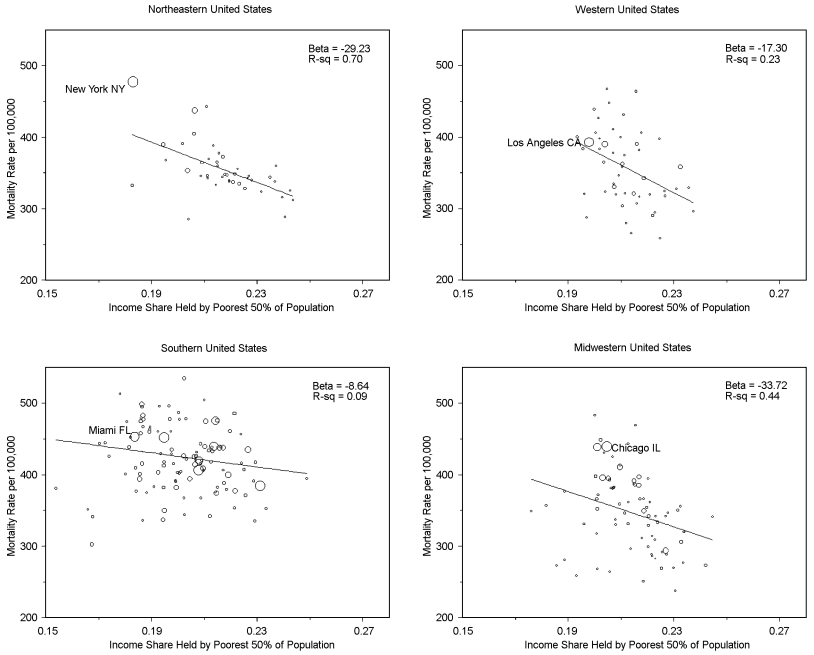
Source: Kaplan et al. 1996.

FIGURE 11. U.S. Median Income Share and Mortality, 1990



Source: Lynch et al. 1998.

FIGURE 12. U.S. Metropolitan Areas' Income Inequality and per Capita Income, 1990



Source: Unpublished analyses by Nancy Ross, Statistics Canada.

FIGURE 13. Associations between Income Inequality and Mortality among Metropolitan Areas, within Regions of the United States, 1990

mortality. Figure 13 shows the associations between income inequality and mortality among metropolitan areas (net median income differences) within each census region of the United States. Although there is a statistically significant association between income inequality and mortality in all regions of the United States, it is stronger in the Midwest and Northeast than in the West. Interestingly, the weakest association was among metropolitan areas in the South. So, while an important component of the overall national picture is the position of southern states and metropolitan areas relative to the others, within the South itself there is a weaker link between income inequality and mortality. This is not the result of a truncated exposure to income inequality. Rather, the range of income inequality within the South is at least as large or larger than within other regions.

Source: U.S. Department of Energy (http://www.eia.doe.gov/cneaf/electricity/epav1/figa1.html).

FIGURE 14. Standard U.S. Census Regions

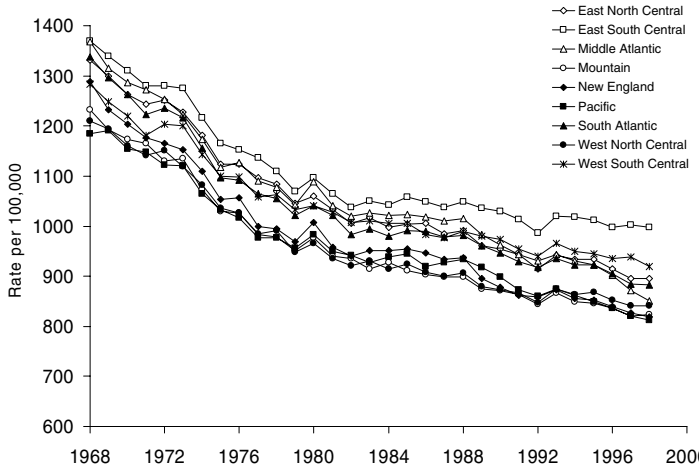
Regional Differences in Income Inequality

In regard to regional changes in income inequality, a report based on the Current Population Survey (CPS) by Bernstein and colleagues (2002) revealed variability in changes in the "top-to-bottom ratio" (incomes of the top 20 percent versus incomes of the bottom 20 percent of the population) among regions of the United States between 1978 and 2000, the period of the large national rise in income inequality. Figure 14 shows the standard census regions, and Table 2 shows that the largest increases in income inequality were along the eastern seaboard—in the middle and southern Atlantic states and New England. There is no association between the starting levels of income inequality in 1978 and the changes over the next 20 years. For instance, in 1978 New England had the lowest ratio at 6.3 and then experienced a large absolute and percentage increase to a ratio of 9.0, an increase of 43 percent. In contrast, the highest starting levels of inequality in 1978 were in the west south central and east south central regions, but they had some of the smallest increases between 1978 and 2000. Thus the changes in income inequality among regions during this period varied. The regions with the highest levels of income inequality in 1978 were the east south

TABLE 2
Changes in Regional Population[a] and Income Inequality[b] in the United States, 1978–2000

Division	% Change in Share of Total U.S. Population 1978–80 to 1996–98	Proportion Black		Increase in Proportion Black Absolute (%)	Top-to-Bottom 20% Income Ratio[c]		Increase in Top-to-Bottom 20% Income Ratio (%)	Reduction in Total Mortality Absolute (%)
		1978–80	1996–98		1978–80	1998–2000		
Middle Atlantic	−13.7	12.0	14.6	2.6 (21.6)	7.1	10.4	3.3 (46.5)	222 (20.6)
South Atlantic	11.5	20.7	21.6	1.0 (4.7)	7.8	10.4	2.6 (33.3)	159 (15.1)
New England	−9.5	4.0	5.7	1.7 (43.8)	6.3	9.0	2.6 (42.9)	167 (16.7)
East North Central	−11.7	10.8	12.0	1.3 (11.8)	6.5	8.7	2.2 (33.8)	188 (17.0)
Pacific	15.5	6.3	6.4	0.1 (2.2)	7.5	9.5	2.0 (26.7)	141 (14.8)
East South Central	−5.9	19.5	20.2	0.6 (3.3)	8.3	10.1	1.9 (21.7)	95 (8.6)
Mountain	25.8	2.3	3.3	0.9 (39.9)	6.8	8.6	1.8 (26.5)	137 (13.8)
West South Central	7.0	14.8	15.0	0.2 (1.3)	8.5	10.2	1.8 (20.0)	148 (13.9)
West North Central	−9.3	4.5	5.6	1.0 (23.1)	6.6	8.1	1.5 (22.7)	143 (14.4)
Average		11.7	12.7	1.1 (9.2)	7.2	9.3	2.1 (29.2)	156 (15.0)

[a]Calculated from Compressed Mortality Files (National Center for Health Statistics 2000, 2001b). Population data based on the U.S. Bureau of the Census intercensal estimates of the July 1 resident population, with the exception of 1980, which is based on the modified age-race-sex (MARS) census counts.
[b]Adapted from Bernstein et al. 2002.
[c]The top-to-bottom 20% income ratio is the ratio of the average incomes of the top 20% versus the bottom 20% of the income distribution.
Note: Regions are sorted in order of largest-to-smallest absolute change in top-to-bottom ratio.



Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 15. U.S. Regions: 30-Year Trends in Age-Adjusted All-Cause Mortality, 1968–1998

central and west south central, and the largest increases over the period were found in the middle Atlantic, south Atlantic, and New England. The regional patterns observed by Bernstein and colleagues (2002) are similar to those reported in other regional analyses of changes in income inequality during the latter part of the 20th century (Morrill 2000; Partridge, Rickman, and Levernier 1996).

Regional Mortality Trends

Figures 15 to 22 show the trends in mortality in nine regions from all causes; IHD; stroke; lung, breast, and prostate cancer; suicide; and homicide. For simplicity, we combined male and female mortality, which conceals some of the differences in some causes of death, especially for IHD and lung cancer, in which the sex-specific trends have significant generational differences. Before examining age-adjusted trends, we first disaggregated and examined age-specific regional trends in infants (ages 0 to 1), children and young adults (ages 1 to 14, 15 to 24), working ages (ages 25 to 64), and older ages (ages 65 and over). Our goal was to detect any regional differences in age-specific mortality trends that could be

linked to income inequality. The overriding impression was the same as that of the age-adjusted national trends in Figure 2: declining rates in all regions, except perhaps for older-age mortality, and of regionally stable trends, so that those regions with the best and worst age-specific mortality profiles maintained their rankings between 1968 and 1998. (These age-specific regional trend plots are available from us.)

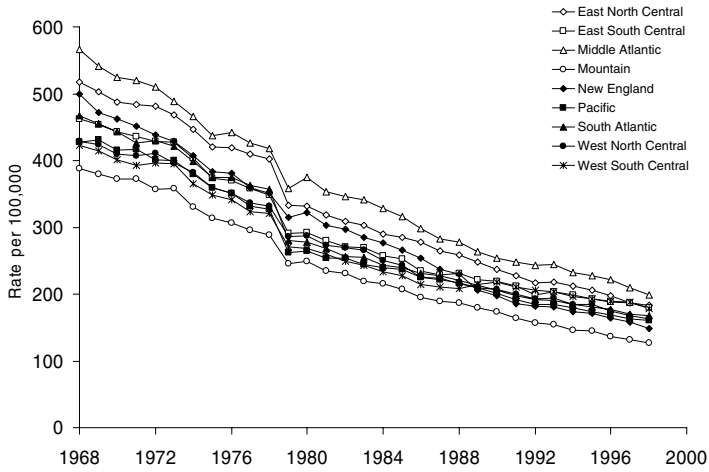
We next describe the main features of the regional trends for different causes of death. The purpose is to see whether there are regional differences not captured by the national trends already shown, and whether there are any differences in the regions' rankings for different causes of mortality. One hypothesis is that those regions with the greatest income inequality (the west south central and east south central) have higher initial mortality rates and worse mortality trajectories and/or that the relatively larger increases in income inequality in the middle Atlantic and New England regions may be reflected in less desirable mortality trends.

Mortality from All Causes

The mortality rates from all causes, seen in Figure 15, show that every region of the United States has improved substantially, but with some widening of regional inequality. The age-specific trends (not shown) suggest that this is due mainly to divergent regional mortality rates of persons aged 65 and over. As we described earlier in our systematic review (Lynch et al. 2004), an unpublished multilevel analysis of the U.S. states found that income inequality is weakly negatively associated with mortality in this age group (Backlund et al. 2003). The Pacific region has the healthiest all-cause mortality profile for the 30 years. In contrast, the east south central region—one of the areas with the highest income inequality in 1978—has the opposite. There also is evidence of slower declines in mortality from all causes in the west south central and east south central regions, beginning in the early 1980s, that may coincide with the period of widening income inequality. But these also are the regions where the links between income inequality and mortality are the weakest (Figure 13).

Ischemic Heart Disease

Figure 16 shows that the middle Atlantic region has historically had the highest levels of IHD and that the mountain states of the west have



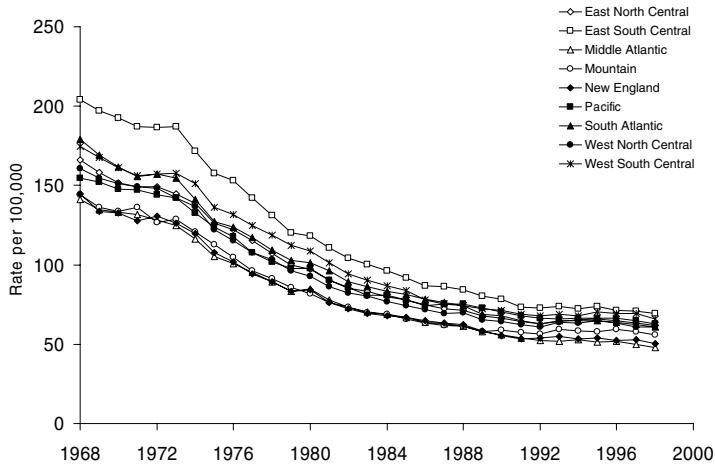
Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 16. U.S. Regions: 30-Year Trends in IHD Mortality, 1968–1998

had the lowest. On balance, this regional inequality in IHD has diminished over the last 30 years at the same time that income inequality has increased within all regions. However, within these relatively stable patterns, New England (with the lowest levels of income inequality in 1978) has shown strong improvements over time—from third worst to second best in 30 years—despite having one of the largest increases in income inequality. In contrast, the west south central and east south central (with the highest levels of income inequality in 1978) reversed their declines in IHD in the mid-1980s and, for a time, were the only regions in the United States to show rising death rates from IHD (Barnett and Halverson 2001). This means that the initial levels of income inequality may have played a role in the differing trends in these regions, in that they swapped their IHD rankings over 30 years.

Stroke

The death rate from stroke fell sharply from 1968 to the early 1980s, with the east south central region experiencing reductions of 50 percent in the 15 years between 1968 and 1983 (Figure 17). This finding contrasts with the slower decrease in overall mortality in the east south central



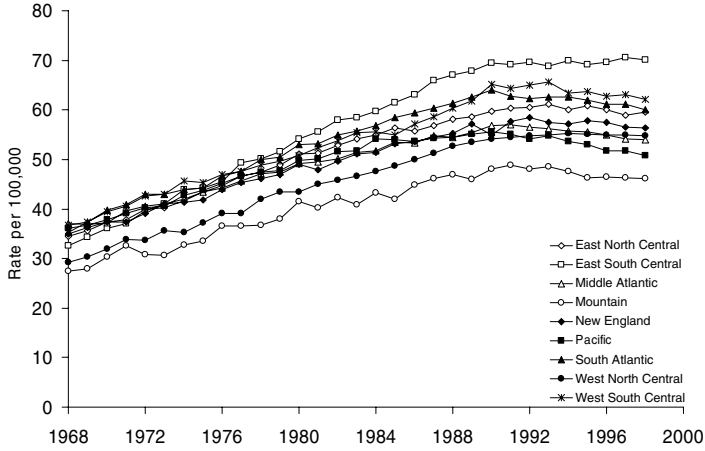
Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 17. U.S. Regions: 30-Year Trends in Stroke Mortality, 1968–1998

region. For stroke, it is the middle Atlantic that consistently had the lowest rates—almost the opposite pattern to that of IHD, for which the middle Atlantic had the highest rates. Regional trends for stroke show a narrowing of absolute regional inequality over time, and a shifting of the stroke burden to the Pacific (which dropped from fourth best to seventh best) and west south central regions (Howard et al. 2001) that does not seem to correspond to regional patterns or trends in income inequality.

Lung Cancer

The mortality rate from lung cancer in Figure 18 indicates the widening absolute regional inequality between 1968 and 1998. Increases in all regions follow the time-lagged increase in smoking prevalence from World War I to its peak in the mid-1960s. Figure 18 also shows that in 1968 (40 to 50 years after the first cohorts began smoking in large numbers), there was a relatively tight clustering of lung cancer rates across regions, with the exceptions being substantially lower rates historically evident in the western, mountain, and north central regions. These rates, of course, differed for men and women (Escobedo and Peddicord 1996). Over 30 years the regional disparity widened markedly, with the east south central and

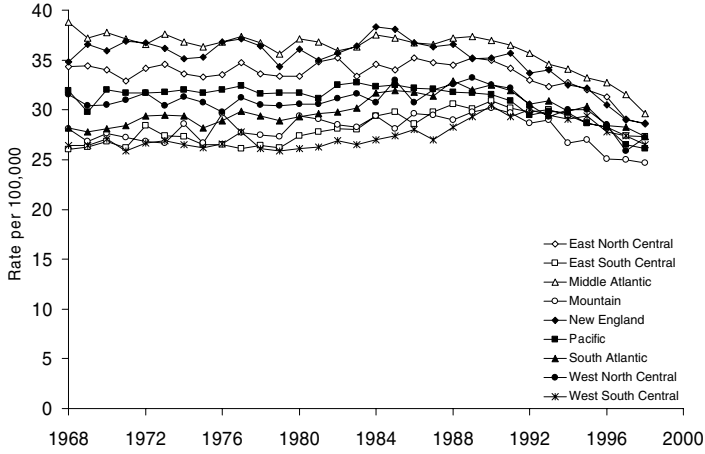


Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 18. U.S. Regions: 30-Year Trends in Lung Cancer Mortality, 1968–1998

the south Atlantic regions showing the largest increases in lung cancer mortality. These are also the main tobacco-producing regions of the United States.

Whatever was initially protective for the uptake of smoking and of later lung cancer mortality in the mountain and west north central regions, however, continued to generate the lowest rates of lung cancer over the next 30 years. It is perhaps not coincidental that the east south central and mountain regions also have, respectively, the highest and lowest IHD rates. These regional trends in lung cancer mortality may be sensitive to ethnic-group and sex-specific birth cohort effects associated with the different beginning ages and cumulative exposure to smoking (Strachan and Perry 1997). For instance, white males born in the decades immediately before and after World War I were the first to begin smoking in large numbers, with smoking among women and minorities becoming most prevalent in cohorts born around World War II (Burns et al. 1997; Escobedo and Peddicord 1996). Thus, the differing regional trends in lung cancer mortality shown in Figure 18 may also reflect the different regional start and cessation of smoking by different population subgroups. These trends are also consistent with Pampel's (2002) findings regarding the importance of the stage at which



Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 19. U.S. Regions: 30-Year Trends in Female Breast Cancer Mortality, 1968–1998

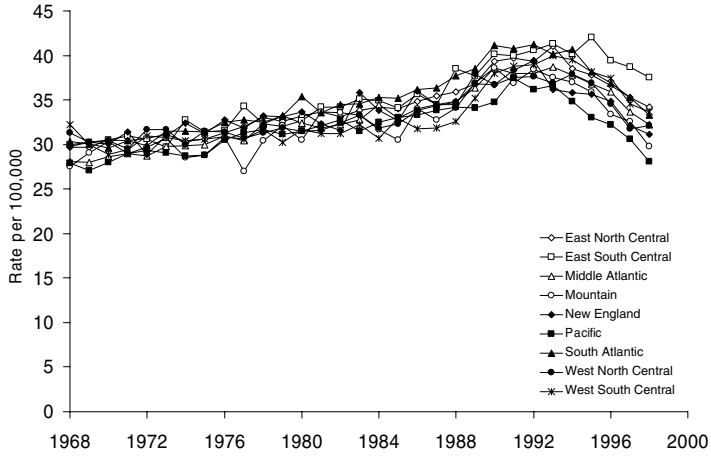
smoking becomes common in the population to understanding current smoking patterns. Interpreting these regional differences may be related to the historical roles of tobacco use in the economies and cultures of the tobacco-producing areas of the southern United States and the more religiously and socially conservative Midwest and mountain areas (Studlar 1999).

Breast and Prostate Cancer

Figures 19 and 20 show that the New England and middle Atlantic regions have the highest breast cancer rates and that the east south central region the highest prostate cancer mortality rates. Regional inequality declines for breast cancer mortality but widens for prostate cancer mortality, with overall declines in both cancers during the 1990s.

Homicide and Suicide

Figure 21 shows relatively stable regional patterns of suicide, with the highest rates in the mountain region. These trends do not seem to have been affected by shifts in income inequality over the same time period.

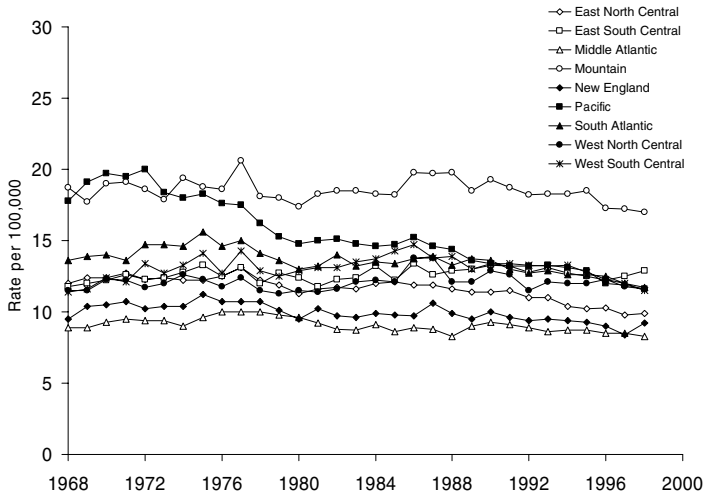


Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 20. U.S. Regions: 30-Year Trends in Prostate Cancer Mortality, 1968–1998

In particular, the mountain region has had high suicide rates but lower levels of income inequality, which grew only modestly over the past 30 years (Morrill 2000). In contrast, the middle Atlantic region, with the lowest (and most stable) suicide rate, experienced one of the largest increases in income inequality during this time.

Some of the strongest arguments supporting the theory that greater income inequality produces worse population health have come from analyses of homicide (Lynch et al. 2004). Inequality may produce distrust and hostility in individuals, causing a breakdown of social cohesion (Kawachi, Kennedy, and Wilkinson 1999), which in turn leads to homicide and violence, presumably within a relatively short time, given that the highest rates are among young adults. Accordingly, there should be a relatively short lag between change in the exposure and trends in the outcome. The regional data in Figure 22 suggest that any changes in income inequality between 1968 and 1998 did not produce any obvious trend changes in homicide mortality in any region of the United States. In some regions, the homicide rate was rather stable, whereas in others it fluctuated throughout the period in which income inequality was consistently rising to a greater or lesser extent in all regions. For example, note the substantial decline in homicide in the middle Atlantic region



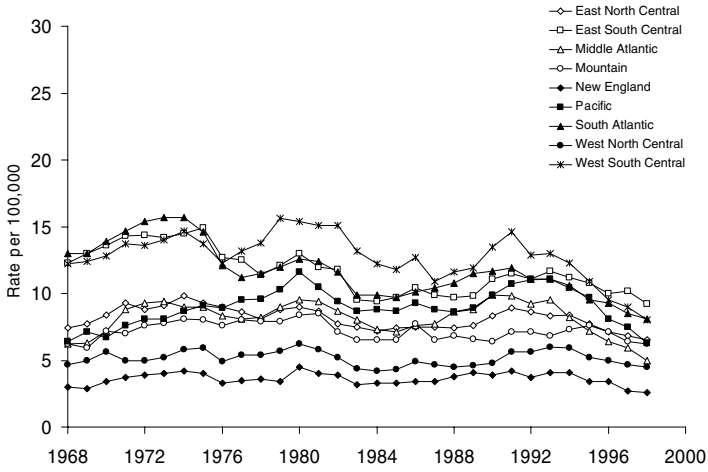
Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 21. U.S. Regions: 30-Year Trends in Suicide Mortality, 1968–1998

since 1994 (most likely due to the falling homicide rate in New York City) that occurred during the strong growth in income inequality.

Income Inequality and Regional Mortality Trends

How might income inequality have influenced these disease-specific trends? First, we examined the regional trends between 1968 and 1998 because this was the period when income inequality rose sharply in the United States—at least in relation to postwar levels. We assumed that there was no time lag between the exposure to income inequality and its effects on mortality trends, which may be more plausible for some health outcomes than others. If historical levels and changes in income inequality are important determinants of these mortality trends, it would be difficult to find the particular mechanisms accounting for the different trends for different diseases and the variable ranking of each region according to the cause of death. Several different mechanisms and time lags seem to produce these patterns, which do not easily fit the theory that the levels of income inequality themselves are responsible for these regionally different cause-specific levels and trends in mortality.



Source: Authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

FIGURE 22. U.S. Regions: 30-Year Trends in Homicide Mortality, 1968–1998

Nor is there any clear association between regional changes in income inequality and reductions in total mortality. All the regions with above-average absolute and relative increases in income inequality also had above-average absolute and relative decreases in mortality (see Table 2). In fact, the region with the largest absolute and relative increases in income inequality from 1978 to 2000—the middle Atlantic—also had the largest absolute and relative reductions in mortality over the same time period, mainly due to declines in heart disease. We summarized the data on these changes in income inequality and changes in mortality by correlating the change in the top-to-bottom income ratio and the reduction in mortality from 1978 to 2000. The Pearson correlation is 0.76 for the absolute change in income inequality with all-cause mortality decline and is 0.81 for the change in relative inequality and mortality decline. Thus from these numbers we concluded that between 1968 and 1998, the greater increases in income inequality within U.S. regions were strongly associated with the greater decreases in mortality.

The associations between income inequality and mortality are confounded, it has been argued, by the particular composition of race and/or ethnicity, in which the higher poverty and increased mortality rates of blacks lead to spurious ecological associations between income inequality

and mortality (Deaton and Lubotsky 2003). If this is true, then we might expect that those regions with higher increases in the proportion of blacks would have smaller reductions in mortality over time. Table 2 also presents data on the proportion of African Americans in U.S. regions from 1978 to 1998. Similar to the trend for income inequality, the region with the largest absolute increase in the proportion of blacks (the middle Atlantic) also had the largest total reduction in mortality. No relationship between the changes in the proportion of African Americans and the changes in income inequality was discerned, a finding that has been replicated in other studies of the determinants of changes in income inequality among U.S. metropolitan areas (Madden 2000). In addition, there is no systematic relationship between the initial proportion of blacks and the reduction of mortality. The region with the highest initial proportion of blacks—the south Atlantic at 20.7 percent—had a better-than-average absolute mortality reduction of 159 per 100,000 persons, whereas the mortality rate of the region with the second highest proportion of blacks—the east south central at 19.5 percent—fell by only 95 per 100,000—the smallest reduction of all the regions.

Conclusion

In this article we examined 100-year national and 30-year regional trends in different causes of death in order to see whether they matched trends in income inequality.

- At the national level, the large declines in income inequality from the end of the Depression to the end of World War II may be reflected in the lower mortality rate of 65-to-74- and 75-to-84-year-olds, but they also may be related to coincidental changes in poverty and the establishment of state-sponsored welfare programs, including Social Security.
- At the national level, 20th-century trends in heart disease appear more compatible with what is known about trends in established risk factors for IHD than with trends in income inequality or, more important, with how income inequality trends could be linked with trends in the major risk factors for IHD. In fact, smoking was rising most steeply at precisely the same time that income inequality fell most dramatically in the 20th century.

- At the national level, the continuous fall of infant mortality and stroke during the 20th century is inconsistent with the fluctuations in income inequality.
- There is some evidence at the national level that during the 20th century the trends in suicide and homicide, particularly between the Depression and the 1950s, are at least partly consistent with the trends in income inequality.
- In the cross section, income inequality is associated with the mortality rates from all causes in each region of the United States, but that association is weakest in the South, where the overall levels of population health are worse than those in other U.S. regions.
- Despite some regional variations in the initial levels and 30-year trends in different causes of mortality, there is little evidence that the national data mask significant regional variations in the association between income inequality and different causes of death.
- The trends in regional income inequality do not seem to be able to explain directly both the differences in the initial levels and the cause-specific regional rankings of mortality, or their trends between 1968 and 1998. We need a more detailed examination of some specific instances, such as the role of income inequality in the differing trends in IHD in the west south central, east south central, and New England regions in the 1980s.

The national and regional trend data shown here illustrate how theories concerning the determinants of population health, which are based on seemingly credible cross-sectional or short-term prospective evidence at the aggregate or individual levels, fail to generate as much support when examined over longer periods of time. The cross-sectional U.S. geographic variation in income inequality has been linked to geographic variation in infant and child outcomes, homicide, heart disease, and other causes of death; and it also has been linked to state and regional differences in self-rated health and mortality in multilevel analyses. But when examined over time, such links are less clear.

The most supportive evidence has been derived from income inequality measured around 1990. Table 3 shows the cross-sectional correlations between income inequality and mortality among U.S. states for each census year from 1950 to 2000. The cross-sectional association between income inequality and total mortality grows stronger after 1950, with the weakest correlation, $r = -0.06$, in 1960, and the highest, $r = 0.58$,

TABLE 3
Contemporaneous Pearson Correlations between Income Inequality[a] and Mortality[b] in U.S. States (N = 50) for Census Years 1950, 1960, 1970, 1980, 1990, and 2000

Census Years	*r*	*p*-value
1950	0.11	0.477
1960	−0.06	0.689
1970	0.22	0.132
1980	0.39	0.006
1990	0.58	<0.001
2000	0.44	0.002

[a]Income inequality measured by Gini coefficient for household income, taken from Langer (1999) for 1950–1980 and calculated by the authors from decennial census files for 1990 and 2000.
[b]Mortality is for all causes and age-adjusted to the year 2000 distribution, taken from the authors' tabulations of the Compressed Mortality Files (National Center for Health Statistics 2000, 2003).

in 1990. It seems possible that the particular configuration of state-level income inequality and all-cause mortality in 1990—precisely the period on which most studies of income inequality and health in the United States focus (Lynch et al. 2004)—fails to capture the dynamics of changes in mortality and the changes in income inequality.

In 1950 Michigan and Arizona were the most unequal states, but between 1950 and 1970 the level of income inequality rose for the southern states, despite the overall decline in income inequality (Langer 1999). The drop in mortality rates during this period was modest in all states, probably because the epidemic of heart disease peaked in the 1960s. There was thus little correlation between income inequality and mortality in 1950, 1960, or 1970.

During the 1970s income inequality began to rise in the United States, and this increase was concentrated among the New England and middle Atlantic states. In 1980 the correlation between income inequality and mortality strengthened because the east south central and west south central states maintained their high levels of inequality but experienced smaller declines in mortality, possibly due to the slower decrease of IHD. By 1990 there was a strong correlation largely driven by almost stagnant mortality declines among the east south central and west south central states and the greater drop in mortality rates and lower income inequality in states like Utah, Vermont, New Hampshire, and South Dakota.

By 1990 mortality rates in New England and the middle Atlantic states had fallen as well, but they had shifted to become middle-range income inequality states. In 2000 the correlation between income inequality and mortality weakened because the New England and middle Atlantic states had become among the least equal. New York was actually the least equal state, with California, Connecticut, Rhode Island, and Massachusetts now joining Louisiana, Mississippi, and Alabama among the other high inequality states. But because these northern states also had stronger decreases in mortality than the southern states had, the correlation between income inequality and mortality weakened.

Considering the entire period from 1950 to 2000, the correlation between the percentage increase in income inequality and the percentage decline in mortality among U.S. states is $r = 0.61$. This description of changes in income inequality and mortality rates among the states helps explain why time-series analyses using similar data failed to find consistent associations (Mellor and Milyo 2001).

Seeking evidence for a concordance between the long-term trends in income inequality and the population health outcomes used here may be a somewhat unreliable way of judging its importance to a population's health. It is possible that the same test applied to population trends in education, poverty, or per capita income would also fail to explain trends in different diseases. Nevertheless, these trends are important to health at the individual and population levels (Sen 2001). Should we even expect potential population-level social determinants to map onto population health trends? Kunitz and Engerman showed a low concordance between trends in real wages and mortality from the 18th to the 20th century. They discussed various reasons for this, related to how cultural practices, geography, technological changes, and government interventions altered the disease environment in ways that afforded some protection, regardless of changes in economic conditions. They concluded that "what is surprising is not that there is no very impressive temporal association between real wages and mortality, but that anyone ever expected there would be" (Kunitz and Engerman 1992, 42).

In contrast, some examples show trends in more proximal risk factors that do map more easily onto population trends in ways that are relatively consistent with causal processes at both the individual and population levels. The most obvious case is for smoking and lung cancer. Although not by any means a perfect concordance, smoking

explains most of the differences in lung cancer risk among individuals and between populations, as well as the trends in a population's lung cancer rates (Lopez 1995). Similar cases might be made, to a greater or lesser extent, for *Helicobacter pylori* infection and peptic ulcer and stomach cancer; rheumatic fever and rheumatic heart disease; folate and neural tube defects; bicycle helmet use and injury; alcohol and liver cirrhosis; drunk driving and motor vehicle accidents; or height and insulin growth factor-1 (IGF-1) related cancer. Such examples are likely to be limited to those situations in which most of the cases arise from a small number of sufficient causes with relatively few component causes (Rothman 1976).

Outcomes that have more diverse pathways and more complicated interactions of risk factors—such as heart disease—make it more difficult to map trends in a single exposure onto trends in disease, because disease trends are the result of complicated interactions with other factors that may not move consistently over time with the single exposure of interest. But if we knew more about how the major risk factors for IHD interacted, then we probably could explain a large part of the population-level trend as a combination of major risk factors—lipids, hypertension, and smoking. This is perhaps less clear for other etiologically complex diseases, such as breast cancer. In such diseases, the population-level trends in known risk factors or their combinations do not map quite so easily onto disease trends within and among populations. But even for breast cancer, some of the population trend is likely to be explained by complicated interactions and trends in known risk factors. Nevertheless, complex diseases (like breast cancer) do present something of a paradox in this regard, and they await greater biological insights into their etiology (Leon 2002).

The concordance between population-level social determinants and population health trends depends on the temporal linkage between a particular social determinant and the specific risk factors for the outcome of interest. A better understanding of the social determinants of population health will require the integration of knowledge across levels. This means that we must understand how broad social and economic conditions in the population are related to the levels and social distribution of the major behavioral and biological risk factors linked to specific pathobiological processes and thus to particular health outcomes. Such linkages change over time and place. The need is for historical and contextual "particularism" (Kunitz 1990) in how broad social

determinants align with specific risk factors for different outcomes (Davey Smith, Gunnell, and Ben-Shlomo 2001; Kunitz 1994; Lynch 2000). A consideration of this evidence on long-term trends in income inequality and mortality underscores the earlier conclusion of Davey Smith and Egger (1996, 1585) that "for different causes of death, and in different temporal and geographical situations, the determinants of mortality patterns will be distinct. Extrapolating from the past to the present and from one place to another is necessary for broad theorizing on the underlying determinants of mortality trends but, in the end, this can only be the start of the more difficult empirical task of understanding the particular factors which act together to produce the patterns seen in any one specific instance."

References

Anderson, R.N., A.M. Miniño, D.L. Hoyert, and H.M. Rosenberg. 2001. Comparability of Cause of Death between ICD–9 and ICD–10: Preliminary Estimates. *National Vital Statistics Reports* 49:2. Washington, D.C.: U.S. Department of Health and Human Services, National Center for Health Statistics.

Backlund, E., G. Rowe, J.W. Lynch, M. Wolfson, G.A. Kaplan, and P. Sorlie. 2003. Income Inequality and Mortality: A Multilevel Study of 521,247 Individuals in 50 U.S. States. Unpublished manuscript (U.S. Census Bureau).

Barnett, E., and J. Halverson. 2001. Local Increases in Coronary Heart Disease Mortality among Blacks and Whites in the United States, 1985–1995. *American Journal of Public Health* 91(9):1499–506.

Beaglehole, R. 1999. International Trends in Coronary Heart Disease Mortality and Incidence Rates. *Journal of Cardiovascular Risk* 6:63–8.

Beaglehole, R., and P. Magnus. 2002. The Search for New Risk Factors for Coronary Heart Disease: Occupational Therapy for Epidemiologists? *International Journal of Epidemiology* 31(6):1117–22.

Ben-Shlomo, Y., and D. Kuh. 2002. A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives. *International Journal of Epidemiology* 31(2):285–93.

Bernstein, J., H. Boushey, E.C. McNichol, and R. Zahradnik. 2002. Pulling Apart: A State-by-State Analysis of Income Trends. Washington, D.C.: Center on Budget and Policy Priorities.

Bobak, M., and M. Marmot. 1999. Alcohol and Mortality in Russia: Is It Different than Elsewhere? *Annals of Epidemiology* 9(6):335–8.

Burns, D.M., L. Lee, L.Z. Shen, E. Gilpin, H.D. Tolley, J. Vaughn, and T.G. Shanks. 1997. Cigarette Smoking Behavior in the United States. In *Smoking and Tobacco Control Monograph 8: Changes in Cigarette-Related Disease Risks and Their Implications for Prevention and Control*, edited by D.M. Burns, L. Garfinkel, and J.M. Samet, 13–42. Bethesda, Md.: U.S. Department of Health and Human Services, National Cancer Institute.

Centers for Disease Control and Prevention. 2002. Tobacco Information and Prevention Source, Consumption Data. Available at http://www.cdc.gov/tobacco/research_data/economics/consump1.htm (accessed Sept. 11, 2002).

Charlton, J., M. Murphy, K.T. Khaw, S. Ebrahim, and G. Davey Smith. 1997. Cardiovascular Diseases. In *The Health of Adult Britain 1841–1994*, Vol. 2, edited by J. Charlton, M. Murphy, and U.K. Office for National Statistics, 60–80. London: Stationery Office.

Colgrove, J. 2002. The McKeown Thesis: A Historical Controversy and Its Enduring Influence. *American Journal of Public Health* 92(5):725–9.

Cooper, R., J. Cutler, P. Desvigne-Nickens, S.P. Fortmann, L. Friedman, R. Havlik, G. Hogelin, J. Marler, P. McGovern, G. Morosco, L. Mosca, T. Pearson, J. Stamler, D. Stryer, and T. Thom. 2000. Trends and Disparities in Coronary Heart Disease, Stroke, and Other Cardiovascular Diseases in the United States: Findings of the National Conference on Cardiovascular Disease Prevention. *Circulation* 102(25):3137–47.

Corcoran, M.E., and A. Chaudry. 1997. The Dynamics of Childhood Poverty. *Future of Children* 7(2):40–54.

Davey Smith, G. 2001. The Uses of "Uses of Epidemiology." *International Journal of Epidemiology* 30(5):1146–55.

Davey Smith, G. 2002. Commentary: Behind the Broad Street Pump: Aetiology, Epidemiology and Prevention of Cholera in Mid-19th Century Britain. *International Journal of Epidemiology* 31(5):920–32.

Davey Smith, G., ed. 2003. *Health Inequalities Lifecourse Approaches*. Bristol, U.K.: Policy Press.

Davey Smith, G., Y. Ben-Shlomo, and J.W. Lynch. 2002. Lifecourse Approaches to Inequalities in Coronary Heart Disease Risk. In *Stress and the Heart: Psychosocial Pathways to Coronary Heart Disease*, edited by S. Stansfield and M. Marmot, 20–49. London: BMJ Books.

Davey Smith, G., and M. Egger. 1996. Commentary: Understanding It All–Health, Meta-Theories, and Mortality Trends. *BMJ* 313(7072):1584–5.

Davey Smith, G., D. Gunnell, and Y. Ben-Shlomo. 2001. Life-Course Approaches to Socio-Economic Differentials in Cause-Specific Adult Mortality. In *Poverty, Inequality, and Health*, edited by D.A. Leon and G. Walt, 88–124. New York: Oxford University Press.

Davey Smith, G., and D. Kuh. 2001. Commentary: William Ogilvy Kermack and the Childhood Origins of Adult Health and Disease. *International Journal of Epidemiology* 30(4):696–703.

Deaton, A., and D. Lubotsky. 2003. Mortality, Inequality and Race in American Cities and States. *Social Science & Medicine* 56(6):1139–53.

Dunn, H.L., and W. Shackley. 1944. Comparison of Cause-of-Death Assignments by the 1929 and 1938 Revisions of the International List Deaths in the United States, 1940. *Vital Statistics—Special Reports* 19:14. Washington, D.C.: U.S. Department of Commerce, Bureau of the Census.

Escobedo, L., and J. Peddicord. 1996. Smoking Prevalence in U.S. Birth Cohorts: The Influence of Gender and Education. *American Journal of Public Health* 86(2):231–6.

Faust, M.M., and A.B. Dolman. 1963. Comparability of Mortality Statistics for the Fifth and Sixth Revisions: United States, 1950. *Vital Statistics—Special Reports* 51:2. Washington, D.C.: U.S. Department of Health, Education and Welfare, National Center for Health Statistics.

Faust, M.M., and A.B. Dolman. 1965. Comparability of Mortality Statistics for the Sixth and Seventh Revisions: United States, 1958. *Vital Statistics—Special Reports* 51:4. Washington, D.C.: U.S. Department of Health, Education and Welfare, National Center for Health Statistics.

Frost, W.H. 1939. Age Election of Tuberculosis Mortality in Successive Decades. *American Journal of Hygiene* 30:91–6.

Glied, S. 2003. Is Smoking Delayed Smoking Averted? *American Journal of Public Health* 93:412–6.

Goff, D.C., G. Howard, G.B. Russell, and D.R. Labarthe. 2001. Birth Cohort Evidence of Population Influences on Blood Pressure in the United States, 1887–1994. *Annals of Epidemiology* 11(4):271–9.

Greenland, P., S.S. Gidding, and R.P. Tracy. 2002. Commentary: Lifelong Prevention of Atherosclerosis: The Critical Importance of Major Risk Factor Exposures. *International Journal of Epidemiology* 31(6):1129–34.

Greenwood, M. 1935. *Epidemics and Crowd-Diseases: An Introduction to the Study of Epidemiology*. New York: Macmillan.

Hallqvist, J., J.W. Lynch, D. Blane, M. Bartley, and T. Lange. 2004. Critical Period, Accumulation and Social Trajectory: Can We

Empirically Distinguish Lifecourse Processes? *Social Science and Medicine* 58:1555–62.

Hetzel, A.M. 1997. U.S. Vital Statistics System: Major Activities and Developments, 1950–95. Department of Health and Human Services Publication No. (PHS) 97–1003. Hyattsville, Md.: U.S. Department of Health and Human Services.

Howard, G., V.J. Howard, C. Katholi, M.K. Oli, and S. Huston. 2001. Decline in U.S. Stroke Mortality: An Analysis of Temporal Patterns by Sex, Race, and Geographic Region. *Stroke* 32(10):2213–20.

Johansson, S.R., and A.B. Kasakoff. 2000. Mortality History and the Misleading Mean. *Historical Methods* 33(2):56–8.

Kaplan, G.A., E.R. Pamuk, J.W. Lynch, R.D. Cohen, and J.L. Balfour. 1996. Inequality in Income and Mortality in the United States: Analysis of Mortality and Potential Pathways. *BMJ* 312(7037):999–1003 (published erratum appears in *BMJ* [1996] 312[7041]: 1253).

Kawachi, I., B.P. Kennedy, and R.G. Wilkinson. 1999. Crime: Social Disorganization and Relative Deprivation. *Social Science and Medicine* 48(6):719–31.

Kelleher, C.C., S. Harper, and J.W. Lynch. 2003. What Goes Up Must Come Down? Evidence for the Contribution of Migration, Social Deprivation and Life-Course Processes to the Rise and Fall of the Coronary Heart Disease Epidemic in the United States of America. Unpublished manuscript (University of Dublin).

Kennedy, B.P., I. Kawachi, and D. Prothrow-Stith. 1996. Income Distribution and Mortality: Cross Sectional Ecological Study of the Robin Hood Index in the United States. *BMJ* 312(7037):1004–7 (published erratum appears in *BMJ* [1996] 312[7040]:1194).

Kermack, W.O., A.G. McKendrick, and P.L. McKinlay. 1934. Death-Rates in Great Britain and Sweden. Some General Regularities and Their Significance. *Lancet* 31:698–703 (reprinted in *International Journal of Epidemiology* [2001] 30:678–83).

Klebba, A.J., and A.B. Dolman. 1975. Comparability of Mortality Statistics for the Seventh and Eighth Revisions of the International Classification of Diseases: United States. *Vital and Health Statistics* 2:66. Washington, D.C.: U.S. Department of Health, Education, and Welfare, National Center for Health Statistics.

Klebba, A.J., and J.H. Scott. 1980. Estimates of Selected Comparability Ratios Based on Dual Coding of 1976 Death Certificates by the Eighth and Ninth Revisions of the International Classification of Diseases. *Monthly Vital Statistics Reports* 28(suppl.):11. Washington, D.C.: U.S. Department of Health, Education, and Welfare, National Center for Health Statistics.

Kochanek, K.D., J.D. Maurer, and H.M. Rosenberg. 1994. Why Did Black Life Expectancy Decline from 1984 through 1989 in the United States? *American Journal of Public Health* 84(6):938–44.

Kramer, M.S., L. Goulet, J. Lydon, L. Séguin, H. McNamara, C. Dassa, R.W. Platt, M. Fong Chen, H. Gauthier, J. Genest, Jr., S. Kahn, M. Libman, R. Rozen, A. Masse, L. Miner, G. Asselin, A. Benjamin, J. Klein, and G. Koren. 2001. Socio-Economic Disparities in Preterm Birth: Causal Pathways and Mechanisms. *Paediatric and Perinatal Epidemiology* 15(suppl. 2):104–23.

Krieger, N. 1994. Epidemiology and the Web of Causation: Has Anyone Seen the Spider? *Social Science & Medicine* 39(7):887–903.

Kuh, D., Y. Ben-Shlomo, J. Lynch, J. Hallqvist, and C. Power. 2003. Life Course Epidemiology. *Journal of Epidemiology and Community Health* 57(10):778–83.

Kuh, D., and G. Davey Smith. 1993. When Is Mortality Risk Determined? Historical Insights into a Current Debate. *Social History of Medicine* 6(1):101–23.

Kuh, D., and R. Hardy. 2002. *Life Course Approach to Women's Health*. Oxford, U.K.: Oxford University Press.

Kunitz, S. 1990. The Value of Particularism in the Study of the Cultural, Social and Behavioural Determinants of Mortality. In *Health Transition: The Cultural, Social, Behavioural Determinants of Health*, edited by J. Caldwell, S. Findley, P. Caldwell, G. Santow, W. Cosford, J. Braid, and D. Broers-Freeman, 92–109. Canberra: Australia National University Press.

Kunitz, S. 1994. *Disease and Social Diversity*. New York: Oxford University Press.

Kunitz, S., and S. Engerman. 1992. The Ranks of Death: Secular Trends in Income and Mortality. *Health Transition Review* 2(suppl.):29–46.

Langer, L. 1999. Measuring Income Distribution across Space and Time in the American States. *Social Science Quarterly* 80(1):55–67.

Langman, M. 2002. Commentary: Peptic Ulcer, Susser and Stein and the Cohort Phenomenon. *International Journal of Epidemiology* 31(1):27–8.

Lawlor, D.A., G. Davey Smith, D. Leon, J. Sterne, and S. Ebrahim. 2002. Secular Trends in Mortality by Stroke Subtype over the Twentieth Century: Resolution of the Stroke-Coronary Heart Disease Paradox? *Lancet* 360(9348):1818–23.

Lawlor, D.A., S. Ebrahim, and G. Davey Smith. 2001. Sex Matters: Secular and Geographical Trends in Sex Differences in Coronary Heart Disease Mortality. *BMJ* 323(7312):541–5 (published erratum appears in *BMJ* [2002] 325[7364]:580).

Lawlor, D.A., S. Ebrahim, and G. Davey Smith. 2002. A Lifecourse Approach to Coronary Heart Disease and Stroke. In *A Lifecourse Approach to Women's Health*, edited by D. Kuh and R. Hardy, 86–120. Oxford, U.K.: Oxford University Press.

Leon, D. 2001. Common Threads: Underlying Components of Inequalities in Mortality between and within Countries. In *Poverty, Inequality and Health*, edited by D.A. Leon and G. Walt, 58–87. Oxford, U.K.: Oxford University Press.

Leon, D. 2002. Commentary on "Disease Trends in Women Living in Established Market Economies: Evidence of Cohort Effects during the Epidemiological Transition." In *A Life Course Approach to Women's Health*, edited by D. Kuh and R. Hardy, 366–73. Oxford, U.K.: Oxford University Press.

Leon, D., and G. Davey Smith. 2000. Infant Mortality, Stomach Cancer, Stroke, and Coronary Heart Disease: Ecological Analysis. *BMJ* 320(7251):1705–6.

Levenstein, S. 2002. Commentary: Peptic Ulcer and Its Discontents. *International Journal of Epidemiology* 31(1):29–33.

Lindert, P.H. 2000. Three Centuries of Inequality in Britain and America. In *Handbook of Income Distribution*, Vol. 1, edited by A.B. Atkinson and F. Bourguignon, 167–216. Amsterdam: Elsevier.

Link, B.G., and J.C. Phelan. 2002. McKeown and the Idea that Social Conditions Are Fundamental Causes of Disease. *American Journal of Public Health* 92(5):730–2.

Lobmayer, P., and R. Wilkinson. 2000. Income, Inequality and Mortality in 14 Developed Countries. *Sociology of Health & Illness* 22(4):401–14.

Lopez, A.D. 1995. The Lung Cancer Epidemic in Developed Countries. In *Adult Mortality in Developed Countries: From Description to Explanation*, edited by A.D. Lopez, G. Caselli, and T. Valkonen, 111–34. New York: Oxford University Press.

Lynch, J. 2000. Income Inequality and Health: Expanding the Debate. *Social Science and Medicine* 51(7):1001–5.

Lynch, J., G. Davey Smith, S. Harper, M. Hillemeier, N. Ross, G.A. Kaplan, and M. Wolfson. 2004. Is Income Inequality a Determinant of Population Health? Part 1. A Systematic Review. *Milbank Quarterly* 82(1):5–99.

Lynch, J.W., G.A. Kaplan, E.R. Pamuk, R.D. Cohen, K.E. Heck, J.L. Balfour, and I.H. Yen. 1998. Income Inequality and Mortality in Metropolitan Areas of the United States. *American Journal of Public Health* 88(7):1074–80.

MacMahon, B., and W.D. Terry. 1958. Application of Cohort Analysis to the Study of Time Trends in Neoplastic Disease. *Journal of Chronic Diseases* 7:24–35.

Madden, J.F. 2000. *Changes in Income Inequality within U.S. Metropolitan Areas*. Kalamazoo, Mich.: W.E. Upjohn Institute for Employment Research.

Magnus, P., and R. Beaglehole. 2001. The Real Contribution of the Major Risk Factors to the Coronary Epidemics—Time to End the "Only-50%" Myth. *Archives of Internal Medicine* 161(22):2657–60.

Malyutina, S., M. Bobak, S. Kurilovitch, V. Gafarov, G. Simonova, Y. Nikitin, and M. Marmot. 2002. Relation between Heavy and Binge Drinking and All-Cause and Cardiovascular Mortality in Novosibirsk, Russia: A Prospective Cohort Study. *Lancet* 360(9344):1448–54.

Marshall, B. 2002. Commentary: *Helicobacter* as the "Environmental Factor" in Susser and Stein's Cohort Theory of Peptic Ulcer Disease. *International Journal of Epidemiology* 31(1):21–2.

McKeown, T., and R. Record. 1962. Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century. *Population Studies* 16(2):94–122.

Mellor, J.M., and J. Milyo. 2001. Reexamining the Evidence of an Ecological Association between Income Inequality and Health. *Journal of Health Politics, Policy and Law* 26(3):487–522.

Mellor, J.M., and J. Milyo. 2002. Income Inequality and Individual Health: Evidence from the Current Population Survey. *Journal of Human Resources* 37(3):510–39.

Morrill, R. 2000. Geographic Variation in Change in Income Inequality among U.S. States, 1970–1990. *Annals of Regional Science* 34(1):109–30.

Morris, J.N. 1957. *Uses of Epidemiology*, 1st ed. London: E & S Livingstone.

Morris, J.N. 1975. *Uses of Epidemiology*, 3rd ed. London: Churchill Livingston.

National Center for Health Statistics. 1978. *Chartbook for the Conference on the Decline in Coronary Heart Disease Mortality: Comparability of Cause-of-Death Statistics, Figures and Tables, and Technical Notes Describing Trends in Ischemic Heart Disease Mortality.* Hyattsville, Md.: U.S. Department of Health, Education and Welfare.

National Center for Health Statistics. 2000. Data file documentation, Compressed Mortality File 1968–88 (machine readable data file and documentation, CD-ROM Series 20, No. 2A). Hyattsville, Md.: U.S. Department of Health and Human Services, National Center for Health Statistics.

National Center for Health Statistics. 2001a. Age-Adjusted Death Rates for Selected Causes, Death Registration States, 1900–32, and United States, 1933–98. Available at http://www.cdc.gov/nchs/

datawh/statab/unpubd/mortabs/hist293.htm (accessed July 30, 2001).

National Center for Health Statistics. 2001b. Death Rates for Selected Causes by 10-Year Age Groups, Race, and Sex: Death Registration States, 1900–32, and United States, 1933–98. Available at http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/hist290.htm (accessed July 30, 2001).

National Center for Health Statistics. 2003. Data file documentation, Compressed Mortality File 1989–98 (machine readable data file and documentation, CD-ROM Series 20, No. 2E). Hyattsville, Md.: U.S. Department of Health and Human Services, National Center for Health Statistics.

National Office of Vital Statistics. 1956. Death Rates by Age, Race, and Sex, United States, 1900–1953: Selected Causes. *Vital Statistics—Special Reports* 43:1–31. Washington, D.C.: U.S. Department of Health, Education, and Welfare.

Nieto, F. 2002. Commentary: The Epidemiology of Self-Deprecation. *International Journal of Epidemiology* 31(6):1124–7.

Pampel, F.C. 2002. Inequality, Diffusion, and the Status Gradient in Smoking. *Social Problems* 49(1):35–57.

Partridge, M.D., D.S. Rickman, and W. Levernier. 1996. Trends in U.S. Income Inequality: Evidence from a Panel of States. *The Quarterly Review of Economics and Finance* 36(1):17–37.

Plotnick, R.D., E. Smolensky, E. Evenhouse, and S. Reilly. 2000. The Twentieth Century Record of Inequality and Poverty in the United States. In *The Cambridge Economic History of the United States*, Vol. 3: *The Twentieth Century*, edited by S.L. Engerman and R.E. Gallman, 249–300. Cambridge, U.K.: Cambridge University Press.

Pope, C. 2000. Inequality in the Nineteenth Century. In *The Cambridge Economic History of the United States*, Vol. 2: *The Long Nineteenth Century*, edited by S.L. Engerman and R.E. Gallman, 109–42. Cambridge, U.K.: Cambridge University Press.

Rainwater, L., T.M. Smeeding, and J. Coder. 2001. Poverty across States, Nations, and Continents. In *Child Well-Being, Child Poverty and Child Policy in Modern Nations. What Do We Know?* edited by K. Vleminckx and T.M. Smeeding, 33–74. Bristol, U.K.: Policy Press.

Rothman, K.J. 1976. Causes. *American Journal of Epidemiology* 104(6):587–92.

Sen, A. 2001. Economic Progress and Health. In *Poverty, Inequality and Health*, edited by D. Leon and G. Walt, 333–45. Oxford, U.K.: Oxford University Press.

Shi, L.Y., J. Macinko, B. Starfield, J.H. Xu, and R. Politzer. 2003. Primary Care, Income Inequality, and Stroke Mortality in the United

States—A Longitudinal Analysis, 1985–1995. *Stroke* 34(8):1958–64.

Shkolnikov, V., M. McKee, and D.A. Leon. 2001. Changes in Life Expectancy in Russia in the Mid-1990s. *Lancet* 357(9260):917–21.

Snow, J. 1855. *On the Mode of Communication of Cholera*. London: Churchill.

Sonnenberg, A., C. Cucino, and P. Bauerfeind. 2002. Commentary: The Unresolved Mystery of Birth-Cohort Phenomena in Gastroenterology. *International Journal of Epidemiology* 31(1):23–6.

Stallones, R.A. 1980a. The Rise and Fall of Ischemic Heart Disease. *Scientific American* 243(5):53–9.

Stallones, R.A. 1980b. To Advance Epidemiology. *Annual Review of Public Health* 1:69–82.

Strachan, D.P., and I.J. Perry. 1997. Time Trends. In *A Life Course Approach to Chronic Disease Epidemiology*, edited by D. Kuh and Y. Ben-Shlomo, 201–19. Oxford, U.K.: Oxford University Press.

Studlar, D.T. 1999. Diffusion of Tobacco Control in North America. *Annals of the American Academy of Political and Social Science* 566:68–79.

Susser, M. 1973. *Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology*. New York: Oxford University Press.

Susser, M., and Z. Stein. 1962. Civilisation and Peptic Ulcer. *Lancet* 1(7221):115–9.

Susser, M., and Z. Stein. 2002. Commentary: Civilization and Peptic Ulcer 40 Years On. *International Journal of Epidemiology* 31(1):18–21.

Susser, M., and E. Susser. 1996. Choosing a Future for Epidemiology: I. Eras and Paradigms. *American Journal of Public Health* 86(5):668–73.

Szreter, S. 1988. The Importance of Social Intervention in Britain's Mortality Decline c. 1850–1914: A Re-Interpretation of the Role of Public Health. *Social History of Medicine* 1(1):1–39.

Szreter, S. 1994. Mortality in England in the Eighteenth and the Nineteenth Centuries: A Reply to Sumit Guha. *Social History of Medicine* 7(2):269–82.

Szreter, S. 1997. Economic Growth, Disruption, Deprivation, Disease, and Death: On the Importance of the Politics of Public Health for Development. *Population and Development Review* 23(4):693–728.

Szreter, S. 2002. Rethinking McKeown: The Relationship between Public Health and Social Change. *American Journal of Public Health* 92(5):722–5.

U.S. Department of Agriculture, Economic Research Service. 2001. *Tobacco Situation and Outlook Report*, TBS-249. Washington, D.C.: U.S. Department of Agriculture.

Van Buren, G.H. 1940. Some Things You Can't Prove by Mortality Statistics. *Vital Statistics—Special Reports* 12:13. Washington, D.C.: U.S. Bureau of the Census.

Vartiainen, E., P. Puska, J. Pekkanen, J. Toumilehto, and P. Jousilahti. 1994. Changes in Risk Factors Explain Changes in Mortality from Ischaemic Heart Disease in Finland. *BMJ* 309(6946): 23–7.

Vartiainen, E., C. Sarti, J. Tuomilehto, and K. Kuulasmaa. 1995. Do Changes in Cardiovascular Risk Factors Explain Changes in Mortality from Stroke in Finland? *BMJ* 310(6984):901–4.

Waldron, I., and J. Eyer. 1975. Socioeconomic Causes of Recent Rise in Death Rates for 15–24-Yr-Olds. *Social Science & Medicine* 9(7):383–96.

Williamson, J.G. 1985. *Did British Capitalism Breed Inequality?* Boston: Allen and Unwin.

Wingo, P.A., L.A.G. Ries, G.A. Giovino, D.S. Miller, H.M. Rosenberg, D.R. Shopland, M.J. Thun, and B.K. Edwards. 1999. Annual Report to the Nation on the Status of Cancer, 1973–1996, with a Special Section on Lung Cancer and Tobacco Smoking. *Journal of the National Cancer Institute* 91(8):675–90.

Woolsey, T.D., and I.M. Moriyama. 1948. Statistical Studies of Heart Diseases: 2. Important Factors in Heart Disease Mortality Trends. *Public Health Reports* 63(39):1247–69.

Yusuf, S., S. Reddy, S. Ounpuu, and S. Anand. 2001a. Global Burden of Cardiovascular Diseases: Part 1: General Considerations, the Epidemiologic Transition, Risk Factors, and Impact of Urbanization. *Circulation* 104(22):2746–53.

Yusuf, S., S. Reddy, S. Ounpuu, and S. Anand. 2001b. Global Burden of Cardiovascular Diseases: Part 2: Variations in Cardiovascular Disease by Specific Ethnic Groups and Geographic Regions and Prevention Strategies. *Circulation* 104(23):2855–64.

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