DEMOGRAPHIC DESTINIES

Interviews with Presidents of the Population Association of America

Interviews Referencing Evelyn Kitagawa
PAA President in 1977

This series of interviews with Past PAA Presidents was initiated by Anders Lunde (PAA Historian, 1973 to 1982)
And continued by Jean van der Tak (PAA Historian, 1982 to 1994)
And then by John R. Weeks (PAA Historian, 1994 to present)
With the collaboration of the following members of the PAA History Committee: David Heer (2004 to 2007), Paul Demeny (2004 to 2012), Dennis Hodgson (2004 to present), Deborah McFarlane (2004 to 2018), Karen Hardee (2010 to present), Emily Merchant (2016 to present), and Win Brown (2018 to present)

EVELYN KITAGAWA

We do not have an interview with Evelyn Kitagawa, who was the 40th PAA President (1977). However, as Andy Lunde and Jean van der Tak (VDT) were interviewing other past presidents, as well as secretary-treasurers, they regularly asked questions about those early presidents whom they had been unable to interview. Below are the excerpted comments about Evelyn Kitagawa.

CAREER HIGHLIGHTS

Evelyn Mae (Rose) Kitagawa was born in 1920 in Hanford, California. In 1941 she received a B.A. with highest honors in Mathematics from the University of California, Berkeley. She then went to work from 1942 to 1946 as head of the statistical analysis unit of the War Relocation Authority in Washington, D.C. Her job included living and working in Japanese relocation camps, where she met her husband, Joseph Mitsuo Kitagawa. After the war, they moved to Chicago where she studied sociology at the University of Chicago, receiving her Ph.D. in 1951, under the mentorship of Philip Hauser (PAA President in 1950-51). She immediately began work as a Research Fellow at the Chicago Community Inventory, a University of Chicago urban research center directed by Hauser. In 1954, she became an Assistant Professor of Sociology. She became a Professor in 1970 and served as the Sociology Department Chair from 1972 to 1978. She was also Director of the Population Research Center from 1977 to 1987. She retired in 1989.

She is probably most famous for her work on differential mortality, especially Evelyn M. Kitagawa and Philip M. Hauser, (Cambridge, Mass: Harvard University Press, 1973). Differential Mortality in the United States: A Study in Socioeconomic Epidemiology (Cambridge: Harvard University Press, 1973). She also published extensively on differential fertility, as we as on the demography of cities and neighborhoods. She died in Chicago in 2007.

From Jean van der Tak's interview with Philip Hauser in 1988:

VDT: Tell me about Evelyn Kitagawa. I understand that you are the godfather of her daughter.

HAUSER: Yes. My goddaughter, by the way, having received offers of scholarships for tuition plus $7500 living expenses from Harvard, Yale, Chicago, and Princeton, is now at Princeton studying art history, with special reference to Japanese. She refused all those scholarships because she received a Mellon fellowship which she could use for three years of graduate work at any institution she selected, with tuition plus a $10,000 per year stipend. Imagine that!

VDT: Wow! Did she do her undergraduate work at Chicago?

HAUSER: No. She was a student here at high school, the Laboratory School, and grade school, but she did her bachelor's degree at Oberlin.

VDT: In art history?

HAUSER: Yes, she had a double major. She's brilliant and beautiful; amazing woman.

VDT: Is she an only child?

HAUSER: Yes. She was born to Evelyn when Evelyn was in her forties. Evelyn was one of my

students; she finished in 1951. She came with her husband, Joseph, who is also an amazing person. He's an Episcopalian priest. Born in Japan, but he came over here to do some studying and was entrapped after Pearl Harbor and put in a concentration camp. That's where Evelyn met him; she was working for the agency that was running the concentration camps. Romantic.

She came to Chicago. She had been a math major at Berkeley, California; bright as a whip. Became interested in demography and I was able to give her a fellowship. She went through everything that we had to teach her, absorbed it all, and was among the top half dozen, I think, that I had occasion to train. Top half dozen, among other things, in methodology, utilizing her math background. She is an excellent methodologist. She became Associate Director of the Center and in due course, I was even able to persuade the Department of Sociology that it was not undesirable or immoral for a female to become a member of the faculty.

VDT: Was she first female on your faculty?

HAUSER: No, I don't think she was the first, but it was very rare. I developed a technique that made it work pretty well. She was Assistant Director of the Population Research Center under each of the titles that we had and listed in the faculty as a Research Associate of the faculty and, I think, with some professorial rank. Eventually, we were able to put her on the faculty as full professor. She became my successor as Director of the Center. She's done superb work. We did one monograph together.

VDT: The groundbreaking book on Differential Mortality in the United States: A Study in Socioeconomic Epidemiology (1973).

HAUSER: Right. Great methodology there. We both were creative but I think she did the dirty work to get it through. It's one thing to be creative; it's another thing to implement it. She did the implementation, and that's why she got the senior authorship on it. It was a very good study.

VDT: Charlie Nam told me that he and Lillian Guralnik developed the census record-matching system that you used, in a pretest for the 1960 census.

HAUSER: Right, that we worked on. I had a grant of $l,0l7,000 from the National Institutes of Health. It was a lot of money at that time, but I'll tell you something interesting. This was a study that the National Center for Health Statistics wanted to do and the Congress would not appropriate the money for the study. And so the grant we got, we gave most of the money back to the Bureau of the Census and the National Center for Health Statistics for working up the confidential census data for us.

VDT: Private enterprise really helping the government! The money came from the government and it got laundered through you.

HAUSER: It got laundered through us back to the government. And Evelyn is, of course, professor at one of the best universities, one of the best research centers.

From Jean van der Tak's interview with Mary Grace Kovar, PAA Secretary-Treasurer (1975-78) in 1989:

VDT: What about Evelyn Kitagawa? She was the first woman president we'd had after a long gap since Dorothy Thomas, in the late 1950s. From 1959 to 1977, there was no woman. Evelyn was the fourth woman president.

KOVAR: Her presidency, I thought, was long overdue for Evelyn. I think she had done outstanding work and had been doing it for years. I did not realize that there had been that long an interval between women. This is my personal opinion, obviously, but I thought she not only deserved to be president but she deserved to be president for several years before she was actually nominated and elected.

She is--you're asking my opinion of her as president--Evelyn is a conscientious, good, hard worker. If you asked Evelyn to do something and she said she'd do it, she did.

VDT: Evelyn came in the first year the president supposedly was going to be responsible for the meeting program, but the switch had come just the year before and she had done it as first vice-president. Ren Farley reminded me that she asked him to do the program. So you must have worked with Ren also that year, as program chairman. [This switch also affected Sidney Goldstein, president just before Kitagawa. He asked Charles Nam to do the program.]

THE UNIVERSITY OF CHICAGO NEWS OFFICE

Evelyn M. Kitagawa, University of Chicago Sociologist, 1920-2007

Sept. 20, 2007

Related Photo:

Evelyn M. Kitagawa, 1920-2007

Evelyn M. Kitagawa, a Professor Emerita of Sociology at the University of Chicago who did path-breaking work on demography, including work on mortality, died Saturday, Sept. 15 at the University of Chicago Medical Center. Kitagawa, 87, was a resident of Chicago's Hyde Park neighborhood.

Among her most important works was a large-scale study on the various factors related to death that she conducted with the late Philip M. Hauser, a Professor of Sociology and her mentor at the University of Chicago.

The two received a $1 million grant from the National Institutes of Health to look at the role of various personal, social and economic factors on the cause of death for 500,000 Americans who died in 1960.

The two found that there were large differences in death rates according to income and education. White men and women between the ages of 25 and 64 with less education had higher death rates, the study found. The study also determined that low-income white women had a death rate almost twice as high as those in higher-income categories.

The two researchers' work on mortality was published in 1973 in the book they co-authored, *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology.*

Kitagawa also was the author of other important books related to demography. In 1980, she published the results of a National Research Council Panel's work on population titled *Estimating Population and Income of Small Areas.* With University of Chicago sociologist Donald Bogue she co-authored two books, *Techniques of Demographic Research: A Laboratory Manual* (1964) and *Suburbanization of Manufacturing Activity with Standard Metropolitan Areas* (1955).

"Although best known for her work on mortality, she also did pioneer work in fertility, being among the first social scientists to systematically study the phenomenon of cohabitation and out-of-wedlock childbearing that began to skyrocket in the 1960s. She was among the first to study childbearing among adolescent girls particularly in low-income and ethnic neighborhoods," Bogue said.

"To her colleagues, Evelyn Kitagawa was a highly intelligent and efficient hard worker, a friend to all of her colleagues and deeply respected and admired by all," he added.

She was born Evelyn Mae Rose in Hanford, California, and received a B.A. with highest honors in Mathematics in 1941 from the University of California, Berkeley.

Kitagawa was head of the statistical analysis unit of the War Relocation Authority in Washington, D.C. from 1942 to 1946 and spent time living and working in various relocation camps, where she met her husband, Joseph Mitsuo Kitagawa. They moved to Chicago where she studied sociology at the University of Chicago, receiving her Ph.D. in 1951. That same year, she began work as a Research Fellow at the Chicago Community Inventory, a University of Chicago pioneer urban research center that had been established by Ernest W. Burgess, and then directed by Hauser. She specialized in the demography of cities and neighborhoods.

She was the organizing and managerial force in the research and preparation of the Local Community Fact Book of Chicago, published after the censuses of 1950 and 1960. "These included a lot of data (census and otherwise) for each subarea of the city. It was important for its time, allowing for rigorous investigation of a variety of topics dealing with urban phenomena — not the least being segregation, health and social class," said former colleague Stanley Lieberson, Abbott Lawrence Lowell Research Professor of Sociology at Harvard.

She served as a consultant to the Chicago Planning Commission and other local non-profit agencies on matters of labor force, housing, mortality and fertility.

In 1954, she became an Assistant Professor of Sociology. She became a Professor in 1970 and served as the Sociology Department Chair from 1972 to 1978. She was also Director of the Population Research Center from 1977 to 1987. She retired in 1989.

During retirement, she continued to serve as a consultant on research projects at the University.

She was elected as fellow in 1959 to the American Sociological Association, as a fellow in 1968 to the American Statistical Association and as a member in 1971 of the Sociological Research Association. In 1977, she was elected President of the Population Association of America. She was also President of the Sociological Research Council from 1976 to 1977. Kitagawa was a member of the Advisory Committee on Population statistics for the U.S. Bureau of the Census from 1975 to 1978 and served as its chair from 1977 to 1978.

Kitagawa was a member of the Board of Trustees of the National Opinion Research Center from 1973 to 1988, when she was named a life trustee.

She is survived by a daughter, Anne Rose Kitagawa of Boston and was preceded in death by her husband, Joseph Kitagawa, a Professor Emeritus of History of Religions and former Dean of the Divinity School at the University of Chicago.

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ON MORTALITY*

Evelyn M. Kitagawa
Department of Sociology, University of Chicago, Chicago, Illinois 60637

The relative emphasis in demographic research on the various components of population change is perhaps not unreasonably represented by the topics of the last five presidential addresses to this association. In 1972, Amos Hawley spoke on "Population Density and the City" (Hawley, 1972). During the next three years, fertility was the subject of addresses delivered by Norman Ryder, Arthur Campbell, and Charles Westoff (Ryder, 1973; Campbell, 1974; Westoff, 1975). And last year, Sidney Goldstein spoke on migration and population redistribution (Goldstein, 1976).

The relative neglect of mortality studies has been attributed, in large part, to the demographer's inclination to view the worth of research in terms of the policy relevance of its results (Preston, 1976a, p. ix). Mortality is presumed to have little relevance to policy because health and longevity are near-universal human values, and governments are expected to spend all that they can to promote the health and longevity of their citizens. However, as Preston has indicated, "The notion that mortality is outside the policy domain cannot be seriously entertained. Nations and individuals do not devote maximum amounts to health-related expenses but divert portions of discretionary funds to schools, roads, houses, and recreation—items that make life more pleasant or productive while not necessarily prolonging it. Mortality levels are inevitably a product of social and individual choice made under budget constraints and in the presence of competing alternatives. They have probably been a focus of group decision making as long as fertility levels have. Not only are mortality levels determined in part as a matter of policy, but the available policies appear to be much more effective than fertility policy in terms of demographic responsiveness" (Preston, 1976a, pp. ix–x).

On the human motivation side, it is clear that although health and longevity may be near-universal values, individuals do not orient all of their behavior to these values—especially if at the expense of alternative satisfactions. One obvious example is the unwillingness of many smokers to give up cigarette smoking despite the overwhelming evidence on its association with mortality from cancer and heart disease. Mortality research in this area has raised important policy questions concerning the sale of cigarettes and has led to various restrictions on cigarette advertisements, on smoking in public places, etc.

Policy relevance, of course, is not the only criterion for evaluating demographic research. An alternative criterion—and one that is more directly relevant to demography as a scientific discipline—is its contribution to an understanding of the causes and consequences of population trends. Today I will try to make some assessment of the results of recent demographic research on mortality using both criteria of evaluation.

HISTORICAL TRENDS

First, let us quickly review historical trends. Until the end of the 17th century, mortality rates were generally high and subject to violent fluctuations as a result

* Presented as a Presidential Address to the Population Association of America at its annual meeting in St. Louis, Missouri, April 1977.

of epidemics and famines. During the 18th century, a number of European populations began to show steady, but slow, improvements in mortality. By 1850, average life expectancy in the West (including North and West Europe, North America, and Oceania) was about 40 years. By 1900, average life expectancy in six North and West European countries and the United States had risen to 50 years, and by 1950 it was 70 years. In other parts of Europe and the Soviet Union, the decline in mortality had a comparatively late start and proceeded at a faster pace (United Nations, 1973, pp. 110–111).

Since 1950, mortality improvements in developed countries as a whole have been relatively small, especially in North and West Europe and the United States. Most of the improvement has resulted from reductions in infant mortality; the rest from smaller reductions for children and women. In a number of western countries, death rates for older males have actually increased since 1950. For example, between the early 1950s and the late 1960s (1950–1954 and 1965–1969), increases in older male mortality were responsible for decreases in male life expectancy at age 65 for nine of the 21 developed countries for which life tables were available; in five of the countries, the decrease in male life expectancy was evident by age 15, and in one country it was evident by age one (World Health Organization, 1974). In very recent years, improvements in older female mortality have slowed down in some European countries (Benjamin, 1974).

Mortality trends in the less developed countries of Africa, Asia, and Latin America have followed a different pattern. Their transition from high to low mortality did not begin until about 1900. It proceeded at a relatively slow pace until about 1940, and at a very rapid rate after World War II. Fragmentary records indicate that, in the late 1930s, life expectancy was about 30 years in Africa and Asia, and about 40 years in Latin America. By the late 1960s, the respective levels were on the order of 43, 50, and 60 (Preston, 1967b). The result has been a marked divergence in life expectancy within the less developed countries since 1940, as African countries in particular have lagged behind in mortality improvements. This is in sharp contrast to the noticeable convergence of mortality levels in the more developed countries since 1940, as virtually all of them completed the transition from high to low levels of mortality and achieved life expectancies of 70 years or longer (World Health Organization, 1974).

FACTORS IN MORTALITY

Mortality levels are determined by the complicated interplay of a variety of medical and other factors. Thus, trends in life expectancy for a given country and differences in life expectancy among countries at a given time may be influenced by differences in average levels of living, public health programs, and various social, political, and cultural factors. Similarly, patterns of differential mortality within a national population are influenced by individual differences in income, education, occupation, access to medical care, personal habits, nutrition and diet, etc. Because these factors are often closely interrelated, it is difficult if not impossible to determine the separate influence of a particular factor on mortality trends and differentials. A definitive assessment of the factors responsible for mortality variation is also greatly hampered by lack of adequate data. Reliable national death statistics are currently available for only one-third of the world's population, and in most cases the time series of reliable statistics for a country is very short indeed (World Health Organization, 1974). The coverage of statistics by cause of death—an important control variable in mortality analysis—is even more limited. As a result, much of our knowledge about current levels and past trends in mortality has been derived from incomplete or fragmentary data, or based on models of mortality patterns abstracted from available data for selected countries.

The search for a model expressing the

typical age pattern of mortality—sometimes referred to as the "law of mortality"—goes back to John Graunt, the founder of demography, who in 1662 wrote about the regularity of births and deaths and the uniformity and predictability of certain biological phenomena (Valaoras, 1973). The first systematic generalization of age patterns from empirical data was the set of model life tables derived by Notestein and his colleagues in the early 1940s to describe the average course through which mortality moved from high to low in Europe after 1870 (Notestein et al., 1944).

In the early 1950s, the United Nations constructed a set of 40 model life tables covering a wide range of mortality levels, each level characterized by its own unique age pattern (United Nations, 1955). In 1966, the scope of the models was enlarged by Coale and Demeny who constructed four different families of model life tables, representing regional differences in the age pattern of mortality (Coale and Demeny, 1966). Ledermann and Brass have also made significant contributions which introduce greater flexibility in the models of mortality (Ledermann and Breas, 1959; Ledermann, 1969; Brass et al., 1968; Brass, 1969; Brass, 1971). The United Nations and Coale-Demeny models have been widely used to estimate current mortality levels in countries with inadequate data and to project the future course of mortality.

In 1974, Preston and Nelson developed a model to portray the cause-of-death structure of mortality change in the same sense that model life tables portray the age pattern of mortality change. Regional and temporal differences in the cause structure were also identified (Preston and Nelson, 1974; Preston, 1976a).

In 1971, Omran outlined a theory of the epidemiologic transition which provides a useful framework for the analysis of the decline from high to low levels of mortality in the course of the demographic transition. "Conceptually, the theory focuses on the complex change in patterns of health and disease, and on the interactions between these patterns and their demographic, economic, and sociologic determinants and consequences" (Omran, 1971 and 1977). The theory distinguishes three successive stages in the transition, during which a long-term shift occurs in mortality and disease patterns whereby pandemics of infection are gradually displaced by degenerative and man-made diseases as the primary cause of death. The theory also differentiates three basic models of the epidemiologic transition arising from variations in the pattern, the pace, and the determinants and consequences of population change.

1. *The classical or (western) model* describes the gradual, progressive transition from high to low mortality (and fertility) that accompanied the process of modernization in most western European societies. Socioeconomic factors (defined to include health habits, hygiene, and nutrition, as well as standards of living) were the primary determinants of the western transition, augmented by the sanitary revolution of the late 19th century and medical and health progress in the 20th century. The distinguishing feature of this model is that the disequilibrating effects of explosive population growth were minimized, because mortality declined slowly enough prior to 1900 for economic growth to be sustained, and the precipitous declines in mortality after the turn of the century were tempered by declining fertility.

2. *The accelerated model* describes the mortality transition most clearly illustrated by Japan, which followed a pattern similar to the classical model but began later in time and was accomplished in a much shorter period.

3. *The contemporary (or delayed) model* describes the relatively recent transition still in process in most developing countries, where substantial declines in mortality did not set in until after World War II. Public health measures have been a factor in the extremely rapid declines in mortality that have resulted in explosive population growth in developing countries since 1950.

CONTRIBUTIONS OF RECENT RESEARCH

Recent research has extended our knowledge of the factors responsible for historical trends and differentials in mortality in a number of important respects. Time permits coverage of only a few highlights.

Cause of Death

We have learned much about the relative importance of different causes of death in the transition from high to low mortality. Preston and Nelson's model of the cause structure of mortality change decomposed the variation in standardized death rates among 165 populations (covering 43 countries and a period of 100 years) into proportionate contributions of 12 causes of death (Preston and Nelson, 1974). About 60 percent of the total decline in death rates was attributed to declining mortality from infectious diseases, distributed approximately as follows: 25 percent to influenza/pneumonia/ bronchitis, 10 percent to respiratory tuberculosis, 10 percent to diarrheal diseases, and 15 percent to other infectious and parasitic diseases. Another 25 percent was attributed to cardiovascular diseases (after adjustment for diagnostic and coding limitations of the data). In general, the causes responsible for mortality decline did not vary substantially over the range of mortality levels observed; that is, the proportionate decomposition for a decline from 30 to 20 per thousand in the age-standardized death rate was essentially the same as a decline from 20 to 10 per thousand. Preston and Nelson point out, however, that while linear relationships provide a reasonably accurate characterization of the past, they cannot be projected into the future, at least for countries currently at the lowest levels of mortality. In these countries, death rates from some causes previously responsible for mortality decline are now so low that they cannot contribute significantly to further reductions.

Regional and temporal differences in the cause structure of mortality were also identified in their model. Over time, the specific infectious diseases (respiratory TB, smallpox, measles, diptheria, whooping cough, typhoid, cholera, malaria, typhus, plague) have made a smaller and smaller contribution to a given level of mortality, suggesting a relatively faster rate of medical progress against these diseases than others. Diarrheal diseases, on the other hand, have tended to gain in prominence over time and have also maintained the most persistent regional differences. With mortality level controlled, diarrheal death rates were higher than average in South and East Europe and in the non-West, and lower than average in North and West Europe and Overseas Europe (United States, Canada, Australia, New Zealand); North and West Europe had higher mortality from respiratory tuberculosis and influenza/pneumonia/ bronchitis; Overseas Europe had higher death rates from cardiovascular diseases and violence. Factors believed to be responsible for regional variations in diarrheal death rates include malnutrition or severe undernutrition, contaminated food and water, and possibly warm climate. Higher cigarette and animal fat consumption may account for the higher cardiovascular death rates in Overseas Europe. Automobile accidents have been responsible for higher death rates from violence in Overseas Europe in recent years.

In another analysis, Preston demonstrated that differences in the cause-of-death structure are largely responsible for interpopulation differences in age patterns of mortality, holding mortality level constant. This relationship provides a useful guide to the selection of the appropriate "family" of model life tables when knowledge of the cause structure of mortality is available (Preston, 1976a, chap. 5).

Sex Differentials in Mortality

One of the most striking recent trends in mortality—especially in the West—has been the continuous divergence of male and female death rates. Stolnitz, in his classic summary of international mortal-

ity trends, showed that higher male mortality was not nearly so pervasive prior to the 1930s as was commonly believed, nor were the instances of reversal limited to the childbearing ages. Higher female death rates were fairly frequent at some ages in the West and in non-Western Europe before 1920, and also in Africa, Asia, and Latin America until the 1950s (Stolnitz, 1956). In three countries at least—Ceylon, India, and Pakistan—the most recent available data indicate that life expectancy at birth is higher for males than females (El-Badry, 1969).

Although there is persuasive evidence that biological differences between the sexes favor lower female mortality, it is also clear that a variety of environmental factors are involved. In countries where lower male mortality is common, a number of cultural and socioeconomic factors are believed to be important, including nutritional practices discriminating against female children, food allocations favoring males, and more and better medical attention to males (Preston, 1976a, pp. 147–148). There is general agreement, however, that biological and environmental factors are so interdependent that it is not possible to determine the separate influence of each. Pressat, without disagreeing with this premise, has suggested that sex differentials in life expectancy in preindustrial Europe, which were consistently less than two years and varied only slightly from country to country, may nevertheless be a useful reference point for the biological component of sex differentials (Pressat, 1973).

The latest United Nations tables indicate that life expectancy at birth is six to eight years longer for females than males in North America, Australia, and many European countries, and that it is ten years longer for females in the Soviet Union (United Nations, 1976). If we apply Pressat's reference point, it appears that environmental factors must be primarily responsible for sex differentials in mortality in western countries today. This inference is also supported by the extremely large increases in sex differentials that have occurred in recent years. In the United States, for example, the difference between female and male life expectancy increased from 3.5 years in 1930 to 7.8 years in 1975 (National Center for Health Statistics, 1974 and 1977). Biological factors cannot account for such a rapid increase because the genetic code changes much too slowly.

A recent analysis by Retherford sheds some light on environmental factors that are related to the divergent trends in male and female death rates (Retherford, 1975). First, he showed that the large increases in sex differentials in three western countries (United States, England and Wales, and New Zealand) between 1910 and 1965 were limited to persons aged 50 and older, and that cardiovascular diseases and cancer were the causes of death responsible for most of the increases. Second, he concluded that cigarette smoking accounted for about 47 percent of the sex differential in mortality between ages 37 and 87 in the United States as of 1962, and that it accounted for 75 percent of the increase in the sex differential between 1910 and 1962. Third, he demonstrated that the effect of cigarette smoking on sex differentials is felt mainly through the effect of smoking on cardiovascular diseases and cancer. These findings are consistent with an earlier study by Preston of "excess male mortality" between 1950 and 1963 for a cross section of western countries, in which he concluded that cigarette smoking was the most promising explanation of recent excesses in older male mortality and that "the diseases considered causally associated with smoking—heart disease, lung cancer, and bronchitis—apparently fully account for the deterioration of older male mortality" (Preston, 1970). Both Preston and Retherford noted possible qualifications on a "causal" interpretation of their results, however. Although the cumulative evidence leans toward a causal interpretation, it is possible that the excess mortality of smokers is due to other mortality-related characteristics by which they

differ from nonsmokers. For example, the socioeconomic status of smokers is typically lower than that of nonsmokers, and mortality is known to be negatively correlated with socioeconomic status. Stress may also be a confounding factor. As Retherford points out, the literature on stress and mortality has many problems stemming from the lack of adequate measures of stress, and Preston's finding that stress had no significant effect on excess male mortality should be regarded as suggestive but not conclusive.

In this connection, it is interesting to note that a recent study of morbidity indicates that sex differentials in morbidity are the reverse of the pattern for mortality (Verbrugge, 1976). The incidence of illness is greater for females than males, and females are more disabled when ill. Part of the difference is due to a distributional effect—diseases with a male excess being weighted heavily in mortality and those with a female excess dominating morbidity—but for several morbidity conditions females have higher morbidity but lower mortality rates. Verbrugge attributes the reversals to female interviewing and illness behavior rather than to higher illness morbidity. This suggests the interesting possibility that lower female mortality may be related to the fact that women report more illness and seek more medical attention when feeling ill, since this behavior may lead to early diagnosis and control of chronic conditions.

Social and Economic Factors in Mortality

The influence of social and economic factors on mortality has been studied on two levels: (a) in terms of the relationship between mortality change and the socioeconomic development of countries, and (b) in terms of the relationship between mortality and socioeconomic characteristics of individuals within a country.

There has been much discussion—and some dispute—concerning the relative contribution of economic development and "imported" public health programs to the extremely rapid mortality declines in developing countries since World War II (United Nations, 1973, pp. 152–155; World Health Organization, 1974, pp. 675–676). A number of studies have demonstrated a strong cross-sectional relationship between levels of mortality and economic development at a given time. Preston, for example, regressed life expectancy on three indexes of economic development in 1940 and 1970—national levels of income, literacy, and caloric consumption (Preston, 1976b). The explanatory power of the regression was the same on both dates (R^2 = .86), a finding which contradicts the general presumption that the impact of public health programs after World War II must have weakened the cross-sectional association between mortality and economic development. There was, however, a structural shift in the regression equation between 1940 and 1970, from which Preston estimated that roughly one-half of the mortality decline in developing countries during the period could be attributed to factors exogenous to economic development, primarily public health interventions. The structural shift was least important in Africa (suggesting less effective public health interventions there) and most pronounced in Latin America.

Social and economic differentials in mortality among subgroups of national populations have been documented in numerous studies, in considerable historical depth for some developed countries and more recently for developing countries. However, a precise assessment of trends in socioeconomic differentials in different regions of the world is not possible. Data prior to the present century are too fragmentary, and more recent data are seldom comparable in definition over time and space. Antonovsky, after careful review of the available data, advanced the tentative hypothesis that class differentials in mortality emerged in the West during the two centuries from 1650 to 1850, reached a peak about the time Malthus made his observations, and began to diminish during the last half of the 19th century. By the early 1950s (the latest date for which he had information), there was blurring of a

consistent class gradient although death rates in the lowest class remained substantially higher than in the other classes (Antonovsky, 1967). Since then, however, evidence has been accumulating of a reversal to increasing socioeconomic differentials in the developed countries. For example, the data for males 15–64 years old in England and Wales during 1959–1963 reveal a very large increase in class differentials and a clear class gradient, as compared with similar data ten years earlier which clearly showed the blurring of differentials in all except the lowest class. The standardized mortality ratios in 1959–1963 decreased steadily from 143 for Class I (lowest class) to 76 for Class V (highest class), as compared with ratios fluctuating from 118 (Class I) to 94 to 101 to 86 to 98 (Class V) for males aged 20–64 in 1949–1953 (Benjamin, 1974).

Similar reversals in trend have been observed in the United States. For example, our studies of socioeconomic differentials in mortality in the Chicago area over a 30-year period showed a general convergence of differentials between 1930 and 1940 but increasing differentials from 1940 to 1960. In 1960, life expectancy in the highest socioeconomic group in the Chicago metropolitan area (the Ring of the SMSA) exceeded the life expectancy in the lowest socioeconomic group by nine years among white males, by ten years among white females, by five years among nonwhite males, and by ten years among nonwhite females (Kitagawa and Hauser, 1973, p. 53). Studies by Lerner and Stutz report a widening of socioeconomic differentials in the United States between 1960 and 1970, as measured by comparisons of the ten highest and ten lowest income states; this pattern reversed a previous convergence of differentials for some decades (Lerner and Stutz, 1977). A similar widening of differentials was found in the state of Maryland between 1960 and 1970, based on comparisons of mortality in high and low income counties (Lerner and Stutz, 1976).

These observed reversals to wider socioeconomic differentials were not unexpected. As the transition to low mortality is completed, degenerative diseases become the major causes of death and such factors as access to good medical care, preventive medical action, health knowledge, and prompt medical treatment become increasingly important in combating mortality. The lower classes are at a disadvantage in these areas.

IMPLICATIONS OF RECENT RESEARCH

Time permits only a brief discussion of a few implications of recent mortality research.

1. *First, there is increasing evidence that social and economic development is an essential component of sustained rapid mortality decline in developing countries.* Preston's analysis of changes in the relationship between mortality level and socioeconomic development between 1940 and 1970 attributed roughly one-half of the gain in life expectancy in 94 developing countries to economic development factors (Preston, 1976b). A recent World Health Organization report notes that the pace of mortality decline in some developing nations slackened during the 1960s and concludes that although public health programs can speed up mortality declines to outdistance improvements in levels of living, there is a point beyond which social, political, and economic factors become increasingly operative (World Health Organization, 1974). There also is evidence of a rise in death rates in a number of poor countries during the 1970s as a result of hunger and nutritional stress (George Washington University Medical Center, 1977).

2. *Second, the very large socioeconomic differentials in mortality in the United States suggest that the most important next gain in mortality reduction in this country might be achieved through improved social and economic conditions of disadvantaged groups in the population rather than by further advances in medical knowledge.* For instance, the elimination of cancer as a cause of death in the United States would have added only 2.6 years to female life

expectancy at birth in 1964 (Preston et al., 1972, p. 771), as compared with a gain of 5.7 years in life expectancy at age 25 in 1960 if socioeconomic differentials had been eliminated and all females had experienced the age-specific death rates of white females with one or more years of college (Kitagawa and Hauser, 1973, p. 17; National Center for Health Statistics, 1963, pp. 2–10). A similar comparison for males indicates that elimination of cancer would have added 2.3 years to life expectancy at birth in 1964, as compared with a gain of 2.1 years in life expectancy at age 25 in 1960. Although comparable data are not available for the nation as a whole in 1970, evidence that socioeconomic differentials increased between 1960 and 1970 has been cited.

The persistence of large socioeconomic differentials in the United States may also account for its relatively poor ranking in mortality comparisons. In the early 1970s, more than 20 developed countries had higher male life expectancy than the United States, and at least nine had higher female life expectancy. In first-ranked Sweden, males lived 4.1 years longer on the average, and females 2.1 years longer. It is interesting to note, in this connection, that Antonovsky's comprehensive review of class differentials in mortality found only one study, for Amsterdam in 1947–1952, that could "legitimately be regarded as strongly contradictory of the link between [social] class and mortality" (Antonovsky, 1967). The Netherlands has one of the lowest death rates ever recorded, ranking second only to Sweden in 1973 (data on socioeconomic differentials are not available for Sweden).

3. *Third, recent research suggests that, as countries complete the final stage of the transition from high to low mortality, death rates will fluctuate in response to a variety of environmental, genetic, social, cultural, and personal factors that are causally related to the degenerative diseases.* The increase in older male mortality in a number of western countries beginning in the 1950s is one example.

4. *Finally, there are the policy implications of recent research on the role of environmental chemicals in cancer and other illnesses.* A number of studies have documented a positive association between air pollution level and mortality (Winkelstein et al., 1967; Lave and Seskin, 1971 and 1972). The United States Environmental Agency has publicized its commitment to underwrite an integrated assessment of population exposure to environmental carcinogens, focusing first on the relation between cancer mortality and carcinogens in air, water, and diet. Their objective in underwriting these studies is "to provide a scientific basis for the regulation of hazardous environmental chemicals" (Train, 1977).

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