Human Longevity, Individual Life Duration, and the Growth of the Oldest-Old Population

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CHAPTER 14. SOCIAL DIFFERENCES IN OLDER ADULT MORTALITY IN THE UNITED STATES: QUESTIONS, DATA, METHODS, AND RESULTS

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Introduction

Life expectancy at birth in the United States is among the lowest in the industrialized world (Population Reference Bureau 2000). In addition, variability in the timing of death seems to be much greater than in other wealthy nations (Wilmoth and Horiuchi 1999). One plausible hypothesis is that average longevity in the U.S. is relatively low because of greater inequality of health and mortality across the American population, so that the most disadvantaged groups pull down the average for the society as a whole. Thus, a thorough analysis of social differences in American mortality seems essential for a better understanding of the country's international ranking in terms of life expectancy.

Beyond the issue of international rankings, inequality in the face of death is an important topic in its own right. How large are the mortality differentials between social groups? What causes these differences, and how do they change over age and time? In this paper, we review the literature on mortality differentials across major social groups in the United States. We do not add new findings to an already vast literature. Rather, we attempt to organize the discussion about this topic and to review key results. Ours is not the first review of this complicated topic, and the interested reader should also consult the excellent articles by Feinstein (1993) and Hummer *et al.* (1998).

We focused our literature search on books and articles written since 1980, but we also reviewed earlier works if they have been cited frequently during the last two decades. We restricted the age range of our analysis to the older adult years, defined loosely as ages above 30 or 40 years, and gave preference to studies of mortality caused by major degenerative diseases, since those are the major killers in that age range. Therefore, we do not touch the vast literature on social differentials in infant and child mortality. We also do not review studies of differential mortality in young adulthood and associated causes of death, such as accidents, homicide, and maternal mortality. Although such causes contribute to mortality differentials at older ages as well, they play a relatively minor role compared to heart disease, cancer, stroke, and the other "diseases of old age."

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Finally, we chose to examine mortality differentials whose origin seems to be mostly social or environmental, rather than biological. For example, although sex differences in mortality clearly have an important social and environmental component, biology also plays a key role. Therefore, we found it convenient to exclude sex differences in mortality from this review and to concentrate on mortality differentials according to a number of social categories: 1) marital status, 2) education, 3) income, 4) occupation, 5) nativity, or place of birth, 6) race and ethnicity, and 7) place of residence. These groupings represent some of the most important dimensions of social stratification in the United States. They are also associated with significant differences in levels and patterns of mortality. In order to limit the scope of our review, we do not discuss differences in mortality by an individual's functional status, perceived health, or known risk factors, although studies of such differentials are well represented in the literature (e.g., Rogers 1995).

Questions and Hypotheses

The literature on social differences in mortality addresses a number of key issues. In this section, we review the major questions and hypotheses that give structure to this discussion. Some research has been concerned mainly with documenting and describing the mortality differentials that characterize the American population. Other inquiries have examined the causes of such differentials. Here, we offer an outline of the key topics that emerge from both of these styles of research.

MAGNITUDE OF DIFFERENTIALS

Mortality differentials are not easy to measure, and thus the first priority of research in this area has been to document the differences that exist. The methodological challenges are reviewed below. At the crudest level, the purpose of this careful measurement is merely to document the existence and measure the size of mortality differences among social categories. The description becomes much more interesting when it includes an analysis of changes in mortality differentials over age or time. A general finding has been that mortality differentials tend to diminish with increasing age, at least in relative terms (Kitagawa and Hauser 1973; Sorlie, Backlund, and Keller 1995; Elo and Preston 1996). This pattern raises important theoretical questions about the interrelated effects of selective attrition and biological ageing (see discussion below).

A key question driving descriptive analyses of mortality differentials is whether these differences have grown or diminished over time (Feldman *et al.* 1989; Pappas *et al.* 1993; Queen *et al.* 1994; Preston and Elo 1995; Duleep 1998; Schalick *et al.* 2000). The accurate measurement of temporal trends in mortality differentials is especially problematic (Duleep 1989). However, there is perhaps no more important question in this discussion than whether social groups are moving farther apart in terms of a life experience so fundamental as mortality, or whether some convergence has been achieved. It seems to be the clear ideological preference of most researchers of this topic that such differentials should diminish over time, and these sentiments echo statements issued by the American federal government (NCHS 1993, cited in Hoyert, Singh, and Rosenberg 1995). In addition, as suggested earlier, trends in mortality differentials over time may influence international rankings of life expectancy at birth.

For various reasons, then, descriptive analyses of mortality differentials are among the most important works on this topic. However, as we emphasize later in our discussion of methods, the instruments currently used to measure mortality differentials in the United States are less precise than would be desirable for a complete and accurate description of levels, patterns, and especially trends. Thus, work in this area will continue to be important.

CAUSAL EXPLANATION

It is imperative to remember that causal analyses of American mortality differentials are built on a descriptive base that is still both imprecise and incomplete. Nevertheless, even if empirical findings must be interpreted with caution, it is still useful to move beyond mere description and to ask deeper questions about the causes and meaning of these differentials. Causal analyses of mortality differentials in the United States have taken several forms. Here, we attempt to organize that discussion under several key headings.

Causes of Death Accounting for Differentials

At the most basic level of causal analysis, we may attempt to understand which causes of death account for observed differences in total mortality. Among young adults, for example, we might ask what proportion of a differential is due to external or violent deaths (accidents, homicide, and suicide), and what proportion is due to disease. Among older adults, the topic of this review, we may ask the same question, but the answer is likely to be less revealing since most deaths at older ages are the result of disease. Instead of focusing on the distinction between external mortality and disease, we must ask which major diseases account for the largest part of total mortality differentials at older ages.

A plausible hypothesis is that mortality differentials at older ages result mostly from two sorts of diseases: those for which individual behaviours like smoking and diet play a crucial role, and those for which there exist effective but expensive treatments. Thus, we might expect to find large differences across social groups in mortality from lung cancer due to differences in smoking habits. We might also find differences in various forms of cardiovascular disease (CVD) due not only to differences in smoking and diet but also to differences in access to medical intervention. If therapies for CVD become more effective over time but access is limited to certain groups, mortality differentials could widen; if access becomes more egalitarian, differentials could narrow. On the other hand, for ailments that are not obviously linked to personal behaviours and for which no effective treatment is currently available (such as some forms of cancer and most neurological disorders), mortality differentials should be small and relatively stable over time.

Correlation Among Different Mortality Differentials

One question that has been widely discussed in the literature is the correlation among the various dimensions of mortality differentials. The purpose of such analyses is to discover one or more primary dimensions of social differentiation and to interpret other forms of mortality variation as the product of differentiation along these primary dimensions. For the United States, the most common version of this question involves mortality differentials by race. Are these differentials due to different behavioural practices (and possibly genetic differences), or do they merely reflect the socioeconomic disadvantage of certain racial

groups, especially Blacks, in American society? Or are both sets of factors important, perhaps because they are interrelated?

Statistical analyses are often used to show that race becomes less important as an explanatory variable once socioeconomic indicators (e.g., education, income, wealth) are included in a regression equation (Sorlie *et al.* 1992; Rogers 1992; Menchik 1993; Sorlie, Backlund, and Keller 1995; Lantz *et al.* 1998; Hummer *et al.* 1999; Rogers, Hummer, and Nam 2000). But how should such a result be interpreted? It does not mean that race is unimportant, because race may itself be a cause of the socioeconomic disadvantage of certain groups. Likewise, it does not mean that socioeconomic status is more important than the personal behaviours that are characteristic of different racial groups, since such variables are usually missing from the same regression equation.

Rather, such analyses should be interpreted as mere comparisons of the predictive power of different variables. A plausible hypothesis is that income and other indicators of an individual's socioeconomic status are better predictors of subsequent mortality than is his or her race. An equally plausible hypothesis is that personal behaviours are even better predictors of mortality than is socioeconomic status. However, neither statement, even if true, implies that race or socioeconomic status is unimportant in the causal sequence that culminates in higher mortality among certain racial groups in the United States.

In short, the results of any regression analysis of mortality differentials must be interpreted in light of a broader causal model. In the case of racial differentials, a reasonable model is that race affects both an individual's socioeconomic status (because of opportunity structures and racial prejudice) and his or her personal behaviours (because of cultural differences). In turn, socioeconomic status affects access to medical care and has further effects on personal behaviours (for example, excess smoking and drinking may be a response to the stress associated with a disadvantaged social status). Personal behaviours and access to care in times of need are the immediate causes of mortality differentials by race, but both race and socioeconomic status contribute, directly or indirectly, to the final outcome. Questions for research concern the relative strengths of the different causal relationships in the model.

Socioeconomic status is related in complex ways to other mortality differentials as well. For example, mortality differentials by nativity, or place of birth or origin, may reflect partly the socioeconomic conditions of migrants. Relatively high or low mortality among migrants may be unsurprising once we account for the social position of the groups involved. On the other hand, mortality differentials by nativity may be contrary to what would be predicted based on socioeconomic status, emphasizing the need to look for other sorts of explanations. Indeed, sex differentials in mortality are quite the opposite of what would be expected based on the relative economic position of men and women.

Education, income, and wealth tend to be highly correlated, and thus mortality differentials in terms of these three variables are interrelated as well. Again, the paths of causation are complex, and it is difficult to assert that one or another dimension of social variation predominates. Family income and wealth during childhood influence educational levels, and educational attainment in turn affects adult earnings and accumulated assets. Income and wealth may have direct positive effects on health, since they can be used to purchase medical care and healthy leisure activities. However, they may have negative effects as well because they make more affordable some unhealthy habits like excessive meat consumption, smoking, and drinking.

Educational attainment influences a person's income and wealth during adulthood and thus has an indirect impact on mortality. Education may have direct impacts on mortality as well, because it brings knowledge about health risks and may help open the doors to a complex and sometimes intimidating medical care system. Although income and wealth make health care more affordable, education may give people the vocabulary and the confidence needed to communicate effectively with doctors and other medical personnel. For people faced with multiple choices about how to spend their money, education may also encourage more healthy choices (for example, fish instead of red meat, exercise instead of television, red wine instead of hard liquor) and thus may mitigate some of the negative health effects associated with high levels of income and wealth.

Personal Behaviours and Cultural Practices

From a public health perspective, perhaps the most important issue is whether differences in mortality between social groups are due to personal behaviours and cultural practices that could be modified through education and outreach. Interventions to alter smoking, drinking, or dietary habits may be important means of reducing mortality differentials. Medical care utilization is another aspect of personal behaviour that may account for mortality differentials and that could possibly be altered through public programs.

However, it is unclear whether interventions to alter the health habits of certain groups can reduce mortality differentials significantly. If group differences in mortality are highly correlated with socioeconomic differences, then targeted interventions seem less likely to be successful. In that case, improving the health of certain groups may require a broader strategy of social reform that reduces socioeconomic inequality in general. But if it can be shown that a significant part of mortality differentials is attributable to individual behaviours tied to cultural traditions rather than to socioeconomic deprivation, successful interventions may be possible without disrupting the existing social order. Furthermore, even if such behaviours are predicted (in a statistical sense) by the disadvantaged social status of certain groups, it may still be possible to intervene using education and outreach to mitigate self-destructive habits.

When faced with specific examples, it may be difficult to distinguish between behaviours that result from socioeconomic disadvantage and those clearly tied to cultural traditions. For example, sub-optimal dietary habits may be a cultural adaptation to a history of socioeconomic deprivation. Even if the historical pattern of social inequality is corrected over time, the group may retain its culinary traditions as an important part of its identity. Likewise, alcoholism in certain groups may not be a longstanding cultural tradition, but rather a recent response to social marginalization in a changing world. In this framework, however, it does not matter whether a cultural practice is new or old, only that it can be identified as a cause of excess mortality and modified through a targeted intervention.

Temporal Ordering of Differentiation

A recurrent issue in the analysis of some mortality differentials is whether individuals are more likely to die because they belong to certain groups, or whether they belong to certain groups because they are less healthy and thus more likely to die. In other words, does differentiation by social affiliation precede or follow differentiation by health status and mortality risk? Both causal pathways are interesting and worthy of study, although they imply different emphases. If individuals with poor health (or with family histories of health problems) are less likely to marry, then health status becomes an important factor in the analysis of marriage patterns.[1] On the other hand, if the health of individuals is unrelated to their probability of marriage, then mortality differentials by marital status provide important evidence about the pros and cons of married life.

The problem of making such distinctions using available data is quite difficult, and few analysts have dared to tackle this problem directly (Goldman 1993). Nevertheless, it is sufficiently general and must be considered as a possible explanation for many commonly observed mortality differentials. Aside from marital status, other examples that may result from selection into social categories on the basis of health status include mortality differentials by income, employment status, nativity, and even religious affiliation. Low income or unemployment may be either the result or the cause of poor health status and elevated mortality risks (Menchik 1993; Chapman and Hariharan 1994; Smith 1999). Migrants may have lower mortality because healthy people are more likely to immigrate, and perhaps also because some foreign residents return to their homelands when they become ill. Religious groups that eschew modern medical care may be selected for individuals with a lower propensity for ill health.

In each of these cases, it is possible and even likely that causation operates in both directions. For example, marriage markets may select for good health at the outset, but marriage itself may also lower mortality risks because of the protective effects of a stable and supportive family life. However, these two processes can also have opposite effects on mortality. For example, migration may select for good health prior to departure, but the challenges and prejudices that immigrants face in a new land may have adverse health impacts.

Concern about reverse causation has led many analysts to favour the use of educational attainment as the primary dimension of social variation used in the study of differential mortality. Unlike income and wealth, educational attainment is relatively fixed throughout life, and an individual's maximum level of education is usually obtained years before the adverse health events that cause the vast majority of deaths in modern society (Kitagawa and Hauser 1973; Preston and Taubman 1994).

Nevertheless, it is also possible that some individuals may simply be inclined to favour long-term over immediate gratification of desires. This sort of predisposition might encourage some people both to pursue educational goals successfully and to avoid risky health

[1] Lee and Panis (1996) suggest that there could also be *adverse* selection into marriage on the basis of health, if less healthy individual recognize the benefits of marriage and choose to marry (or to remarry more quickly) as a result.

behaviours, whereas those who favour the present over the future might be less successful as students and more prone to risk-taking in general (Fuchs 1986:214–42). On the other hand, since education is only partly an individual choice and is heavily constrained by family pressures and social norms, personality factors may have a limited influence on the level achieved. In that case, educational attainment would indeed offer an excellent opportunity for causal analysis of mortality differentials, relatively free from the effects of reverse or mutual causation.

Age Patterns of Mortality Differentials

As noted earlier, a common finding is that mortality differentials tend to diminish with age, at least in relative terms. The cause of this recurrent pattern is uncertain, however. One possibility is that selective attrition removes individuals who have the greatest mortality risks from all groups, but the cumulative force of selection is greatest for the most disadvantaged groups. Therefore, at older ages only the most robust members of disadvantaged groups remain, and these individuals compete more successfully with the remaining members of advantaged groups. In extreme cases, a "crossover" of the two mortality patterns may occur, whereby a disadvantaged group displays higher mortality at younger ages and lower mortality at the end of life (Manton and Stallard 1981). However, apparent mortality crossovers may also be the result of imperfect data, so caution is required in evaluating such empirical patterns (Coale and Kisker 1986; Elo and Preston 1994).

Selective attrition is not the only plausible explanation for the convergence of (relative) mortality differentials over age. Alternatively, the social factors that contribute to such differentials at younger ages may diminish at older ages. Elderly persons may reduce risky behaviours, like excess smoking and drinking, and retirement may bring a reduction in work-related stress (House, Kessler, and Herzog 1990). Such lifestyle changes may bring greater benefits to the groups most adversely affected at younger ages, leading to a narrowing of mortality differentials. In addition, government programs providing income support and medical care for the elderly may "level the playing field."

Thus, some important differences between social groups may be left behind as individuals grow older. Furthermore, due to the shared challenges of biological ageing, individuals may grow more alike as they age. Whereas the causes of mortality at younger ages are amenable to considerable social and environmental manipulation, it is possible that the problem of physiological ageing, which is the dominant cause of mortality at older ages, is much less tractable. The imprint of social disadvantage at younger ages probably remains and continues to exercise some influence at older ages, but these differences may eventually be overwhelmed by the biological forces of deterioration that affect all humans, regardless of social class.

Another force that may promote convergence, and even cross-over, of mortality rates at older ages might be called "social hormesis." In biology, "hormesis" refers to the positive aftereffects of exposure to mild levels of stress, which appears to increase average longevity in several non-human organisms (for a review, see Minois 2000). The most common example is exposure to low levels of radiation; but heat shock is another stressor that is known to raise the life expectancy of survivors in some laboratory settings. It seems at least plausible that a similar mechanism could operate in human populations among individuals

who are faced with relatively high levels of social stress during early and adult life. Survival at older ages may be enhanced by coping skills developed at younger ages in response to social adversity, in keeping with the familiar saying, "if it doesn't kill you, it makes you stronger."[2]

Community Effects

Mortality differentials by place of residence are commonplace. These differences may be due in part to differences in the socioeconomic status of individuals residing in these locations. However, community of residence may have direct effects as well for a variety of reasons. In general, the social and economic conditions of a community are related to the quality of housing stock, the availability of local medical resources, the presence of environmental contaminants, and the acceptability of certain behaviours (Anderson *et al.* 1996). All of these factors could have health effects on individuals in the community independent of their own socioeconomic status.

The health effects of community of residence may be especially pronounced in cases of residential segregation of economically disadvantaged and/or minority populations. Social isolation in these situations may lower self-esteem and promote feelings of alienation, thus encouraging personally and socially destructive behaviours. Daily contact with similarly disadvantaged individuals may promote feelings of hopelessness and discourage a positive outlook on the future (Sanders-Phillips 1996; Williams 1999; Polednak 1996). The geographic concentration of disadvantaged persons may also favour the development of social pathologies like crime, violence, and excess drinking and smoking. Indeed, one outcome of residential segregation in the U.S. is that low-status Blacks seem to experience qualitatively inferior residential environments compared to low-status Whites (Massey and Fong 1990). For these reasons, residential segregation may contribute to mortality differentials by race in the United States.

Another possible connection between community of residence and mortality differences is the potential psychological effect of relative deprivation. As discussed already, absolute deprivation, or poverty, is often thought to contribute to mortality differentials. However, it has also been suggested that individuals may suffer stress-related health problems due to a sense of inferiority created by relative deprivation (Wilkinson 1992, 1996). If correct, the hypothesis suggests that mortality differentials will never disappear so long as economic inequality exists in a society. Conversely, if economic inequality diminishes, mortality differentials should decrease as well.[3]

If relative deprivation is a cause of mortality differences in a population, it may seem that residential segregation of disadvantaged groups could help to ease the stresses of

[2] Friedrich Nietzsche apparently wrote, "That which does not kill me makes me stronger" (in *Beyond Good and Evil*). We thank Steve Austad for pointing this out.

[3] However, it is worth noting that even in the absence of economic differences, individuals would still be ranked (formally or informally) according to a variety of criteria (beauty, intellect, athletic ability, political power, holiness, etc.). Thus, some degree of psychological stress due to feelings of inferiority could remain.

perceived inferiority by minimizing contact with advantaged groups. However, given the ubiquitous images of wealth and glamour provided by television and other mass media, it seems unlikely that disadvantaged groups, even if geographically isolated in their place of residence, could be unaware of their relative position in society.

Summary of Data Sources

There are at least three types of data for use in computing mortality rates by social category (Valkonen 1993). The first draws information from two independent sources, typically death records and census data, each containing breakdowns for a given social category (e.g., sex, race, educational attainment). The second source of data on mortality differentials is longitudinal follow-up studies. These are studies where information is collected on individuals at multiple time points (e.g., some health and nutrition surveys) not merely for the purpose of mortality follow-up. The third format consists of cross-sectional survey or census data linked to subsequent death records, but with no other follow-up except to ascertain mortality status. This third type can be based on a single baseline data set with mortality follow-up over some period, or there can be several baselines at different times (e.g., annual health surveys) with mortality follow-up until a common endpoint.

In this section, we provide an overview of data sources for studying differential mortality in the United States and discuss general strengths and weaknesses associated with each type of data. We draw heavily on a review of this topic by Hoyert *et al.*(1995).

DATA FROM TWO INDEPENDENT SOURCES

Vital statistics and census data for any national population are typically classified according to some key social categories. Minimally, such data are broken down by age and sex. In the United States, racial categories are also a common feature of aggregate data. Using such information, it is possible to construct age-specific death rates by sex or race merely by dividing deaths for some group by population counts for the same group.

The advantages of such an approach are that it is simple to apply and that the results usually pertain to the entire population (not a sample). The major disadvantage is that the classification schemes used in the two sources may not be fully comparable, resulting in "numerator-denominator bias." In the case of sex, we can safely assume that the two classification systems will be almost identical. However, an individual's race may be recorded differently in the two data sources, especially for individuals belonging to less numerous racial groups (Rogers, Carrigan, and Kovars 1997).

In the United States, it appears to be more likely that an individual's race or ethnicity will be recorded as Asian and Hispanic in a census than on a death certificate. Census data are derived from self-reported information and thus tend to reflect a great diversity of race and ethnicity, whereas death certificates completed by doctors or medical examiners are more likely to classify individuals as either Black or White. Such errors probably have only minor

effects on mortality estimates for larger groups, like Whites and Blacks,[4] but are known to produce an important downward bias in death rates for smaller race/ethnic categories, like Asians and Hispanics (Rosenwaike, Hempstead, and Rogers 1991; Rosenberg *et al.* 1999). Caution in the interpretation of calculated mortality rates is especially appropriate in the case of Hispanics, since some detailed studies of death certificates have found that a substantial fraction of decedents with Spanish surnames were not recorded as being of Hispanic origin (Polednak 1995; Sorenson 1998).

Mortality rates along dimensions other than sex and race are even less reliable. In some cases, the two classification schemes are clearly not compatible. For example, the census asks individuals about current occupation, whereas the death certificate contains information on "usual occupation" (Hoyert *et al.* 1995). Even if the two data sources appear to contain a breakdown by the same variable, it is important to be skeptical about whether the meaning and interpretation of the variable were in fact the same. In other cases, one of the data sources (typically the death certificate) may simply lack the desired categories.

"Followback surveys" are an attempt to address some of these difficulties. Such surveys collect census-type information from next-of-kin for a sample of decedents in the population of interest. For example, the National Mortality Followback Survey (NMFS) of 1986 was based on a 10% sample of death certificates. Next-of-kin were contacted about six months after a death and were asked to complete a questionnaire about the decedent (Hoyert *et al.* 1995). A more recent NMFS was conducted in 1993. One advantage of such surveys is that they can be designed to provide more information about the characteristics of decedents than is typically available on a death certificate. The other advantage is that the information is collected through reports by next-of-kin, which should be closer in meaning to the self-reports of individuals than are death certificates completed by medical personnel. In theory, at least, followback surveys should minimize numerator–denominator bias, but since the data for numerator and denominator are drawn from two different sources, it is still possible that they are not fully comparable.

LONGITUDINAL STUDIES (COMPLETE FOLLOW-UP)

A number of studies follow groups of individuals over a period of several years and collect information at several time points. In these cases, mortality follow-up is not the primary intention of the longitudinal study design. Nevertheless, mortality information is often available either indirectly (when it is known merely that an individual died during some given interval) or directly (when exact information on date, and possibly cause, of death is obtained for decedents). Routine follow-up of study participants may be supplemented by searches in the National Death Index (NDI) or Medicare beneficiary records. Using multiple sources for mortality follow-up helps to ensure the completeness and accuracy of information about the timing and circumstances of death.

[4] The measurement of Black–White mortality differences is probably affected more by differential accuracy in age reporting (see Elo and Preston 1994; Hill, Preston, and Rosenwaike 2000) than by inconsistent racial classification.

Another advantage is that longitudinal data of this sort are well suited to the analysis of the causal processes that influence mortality differentials by social group. It is possible in some cases to study whether social differentiation precedes or follows divergence in mortality risks. For example, does lower income result in increased mortality, or does poor health lead simultaneously to increased mortality risks and lower income? With data on all three variables (health, income, and death) over several years of a person's lifetime, it is possible to address such questions directly (Menchik 1993; Chapman and Hariharan 1994). Without such information, it is impossible to do more than just speculate about reverse and mutual causation.

A potential disadvantage of data in this format is their limited scope. If the data refer to a single cohort, they do not permit an analysis of temporal change in the variables of interest. Even if multiple cohorts are followed, studies of mortality change over age and time may be awkward, since cohorts are observed over different age ranges. But perhaps the most significant difficulty with data in this format is that sample sizes may be too small to measure mortality differences between relatively small social groups.

MATCHED DATA (FOLLOW-UP FOR MORTALITY STATUS ONLY)

A third source of information on mortality differentials consists of cross-sectional survey or census data that have been linked to subsequent death records. In these studies, there is no follow-up of participants other than to ascertain the date and, possibly, the cause of death.

The first data set of this type was the classic Matched Records Study, which linked a national sample of death records during four months, May–August 1960, to Census records from April of the same year (Kitagawa and Hauser 1973:184). Given the limited computer technology of the time, records had to be matched manually by searching through files of Census records organized by block. The restriction of the sample of deaths to a short time period soon after the Census enumeration was intended to facilitate the matching procedure, on the theory that the address on the death certificate should usually fall within the block where the person was residing a few weeks or months earlier. Nevertheless, only about 77 percent of the deaths in the sample were successfully matched to a corresponding Census record (Kitagawa and Hauser 1973:187). Then, as now, data analysis had to rely on an assumption that the probability of a successful match was unrelated to the covariates of interest.

Today, computers are used to perform a similar form of record linkage. Now, however, instead of linking backward from a sample of deaths to an earlier census (like Kitagawa and Hauser), the usual strategy is to link forward from a sample survey to a complete list of deaths. The National Death Index (NDI) was created for this purpose and covers all deaths occurring in the United States from 1979 onward. A researcher can submit a list of names and other identifying information to the National Center for Health Statistics (NCHS), which searches for possible deaths among individuals on the list over a specified range of years. The search returns only a list of potential matches (zero, one, or more) for each individual on this list. For each potential match, the NDI provides information about the closeness of each match, as well as the date of death, the state where the death occurred, and the death certificate number (NCHS 1990). The researcher has the responsibility of

cleaning this list to eliminate "false positives" and of contacting the various states to obtain copies of death certificates, if needed.

For the United States, the most widely used data set based on this type of match procedure is the National Longitudinal Mortality Study (NLMS). There are two versions of the NLMS: one that can be analysed only within the U.S. Census Bureau and another that has been released to the public. The public-use sample in its current form consists of five rounds of the Current Population Survey (CPS) during 1979, 1980, and 1981, linked to death records during 1981–1989 using the NDI. The version that is not available to the public contains linked records from more rounds of the CPS over a broader range of years, plus an "enumeration sample" from the 1980 Census (Rogot *et al.* 1992b).

A disadvantage of the public-use NLMS is that users cannot know when an individual entered the study, because the date of survey has been suppressed by the Census Bureau in order to minimize the possibility that participating households could be identified (in conformity with Title XIII regulations, which apply to all data produced by the Census Bureau, including the CPS). For this reason, the public-use NLMS refers to a fuzzy nine-year time period beginning around mid-1980 and ending in mid-1989. The NLMS was the data source for numerous studies during the past decade (e.g., Rogot *et al.* 1992b; Sorlie, Backlund and Keller 1995; Elo and Preston 1996; Anderson *et al.* 1996; Backlund, Sorlie, and Johnson 1996; Johnson, Sorlie, and Backlund 1999). These data have been used to assess the magnitude of mortality differences in the U.S. during the 1980s and to describe their age pattern. However, the NLMS design is not well suited to an analysis of temporal trends in mortality differentials unless combined with results from other studies that may not be fully comparable (Preston and Elo 1995).

In addition to the NLMS, the annual National Health Interview Survey (NHIS) for years 1986–1994 has been linked to death records through the end of 1995 using the NDI to create the NHIS-NDI. These data have been used in a comprehensive analysis of mortality differentials made by Rogers *et al.* (2000). Unlike the NLMS, the NHIS-NDI provides complete information about when an individual enters and exits the study population. Because the NHIS is conducted by NCHS and not by the Census Bureau, the same measures to safeguard the identity of participants are not required (nevertheless, a breach of confidentiality seems highly unlikely). For this reason, the NHIS-NDI may ultimately prove to be more useful than the NLMS, especially for the study of trends in mortality differentials.

The major advantages of data in this form are their size and their potential temporal breadth. The baseline surveys used (such as the CPS or the NHIS) are relatively large and provide detailed information about the national population. The NDI is a relatively inexpensive means of follow-up and can theoretically be used to follow survey participants until all of them have died. When annual surveys (like the NHIS) are combined into a single data set, with mortality follow-up through the NDI, researchers have the opportunity to track the evolution of mortality differentials over time and across a broad age range.

There are, however, some unresolved problems with this kind of data for the United States. It has been reported in the literature that approximately 93% of deaths can be identified successfully using the NDI. Our unpublished results, based on both the NLMS and the

NHIS-NDI, suggest that this figure may be significantly lower in some population sub-groups. Obviously, differential success in linkage would bias estimates of mortality differences. Another problem is that most of the baseline surveys used in these studies (e.g., CPS, NHIS) exclude the institutionalized population. At older ages, residents of nursing homes (part of the institutionalized population by definition) are among the most likely to die (McConnel and Deljavan 1982). In addition, the frequency of institutionalization differs across population groups and over age, further complicating an analysis of mortality differentials using such data.

Some of these problems with the data source may eventually be resolved. It should be possible to improve the linkage procedure and to expand coverage to include the institutionalized population. However, a more fundamental disadvantage of this sort of data is that they offer little opportunity to address issues of causation at an individual level. When faced with possible reverse or mutual causation, researchers can do little to resolve the issue using such information. However, other data sources may also be used for mortality follow-up, such as Social Security administrative records (Olson 1999). Such data offer richer analytic possibilities, because they contain information on individuals over the life course (e.g., earnings histories) in addition to vital status.

Methods of Analysis

Many methods can be used for the quantitative analysis of mortality differentials. We will not attempt a complete review of this topic but only offer some highlights of important techniques and issues.

MEASURES USED FOR COMPARISON

In any comparative study, the first task is to choose an appropriate measure. Three common choices for a comparative analysis of mortality are death rates, probabilities of dying, and life expectancies.

Death Rates

By definition, death rates are the number of deaths over some interval of age and time divided by the person-years of exposure over the same interval. Such values can be computed for the population as a whole or for sub-groups, if deaths and exposures are classified according to the same categories. When the characteristics of deaths are found through a matching procedure, the matched sample of deaths can be used to classify all decedents in the population according to the characteristics of interest by assuming that the matched decedents are representative of the population as a whole. If this assumption is correct, the procedure yields unbiased estimates of mortality rates by social group.

Probabilities of Death or Survival

Demographers often consider death rates to be the fundamental unit of analysis, but two alternative measures are probabilities of death or survival. Although these measures are easier to understand for non-specialists, they also present some conceptual difficulties in comparative studies of mortality. A fundamental problem is that all mortality differences

expressed in terms of probabilities of death or survival disappear if the time interval is sufficiently short or long, since these probabilities then approach zero or one by definition. On the other hand, death rates measure the intensity of dying at any moment, or over some interval of age and time, and do not in general converge toward zero or one as the interval shrinks or expands.

Furthermore, according the standard mathematical model, discrete probabilities of death or survival are functions of the continuous hazard curve over some age interval, not the other way around. Therefore, an analysis of differences in death rates, which are discrete approximations to the underlying continuous hazard function, brings us closer to the causal processes producing mortality differentials than does a study of differences in probabilities of death or survival. We conclude that, at least from the specialist's point of view, there are sound reasons for viewing mortality differentials in terms of rates rather than probabilities. These same arguments apply to the odds of death or survival, which are merely functions of probabilities.

Life Expectancies

Yet a third alternative is to measure mortality differences in terms of life expectancy (at birth or at some later age). This approach also appeals to non-specialists, who can understand a concrete concept like the average duration of life in a population better than an abstract notion like a death rate.

METHODS OF COMPARISON

In addition to choosing the metric (rates, probabilities, life expectancies), it is also necessary to choose a method of comparison. Perhaps the simplest, and certainly the most common, technique is to compute the ratio of death rates (or probabilities or life expectancies) for the two groups being compared. Alternatively, one may merely compute the difference in these values for the two groups. This distinction in methods of comparison is sometimes referred to as "relative versus absolute" differences, or "proportional versus absolute" differences (see also Mackenbach and Kunst 1997).

As in the previous section, the distinctions outlined here are not merely academic. There are published examples where the magnitude of a mortality difference either increases or decreases over age or time depending on the measure and method of comparison (Martikainen and Valkonen 1998). There is probably no answer in general to the question of which method of comparison is the correct one.

MULTIVARIATE ANALYSIS

Multivariate statistical methods have been widely used in the study of mortality differentials, mostly as a means of understanding the interrelated effects of alternative explanatory variables. As mentioned earlier, a common approach has been to introduce socioeconomic variables (like income, wealth, education, and occupation) into a regression equation already containing the variable of primary interest, such as race, ethnicity, marital status, or nativity. If the coefficient of the primary variable diminishes after such controls are

introduced, it is said that socioeconomic status accounts for some part of the observed mortality differential (by race, etc.).

There are a number of familiar multivariate methods, and the choice amongst them depends mostly on the dependent variable used in the analysis. Logistic regression is used when the dependent variable is a probability (of death or survival), whereas Poisson regression is used to model death rates. Hazards models, or survival analysis, are especially useful when data are available in the form of survival times for individuals.

Notably, all three of these methods express mortality differentials in relative terms: the coefficients of a Poisson regression are an estimate of the relative death rate in a social category (compared to some reference group); logistic regression yields estimates of the relative odds of dying; finally, the most common form of hazards modelling (the proportional hazards model) assumes that the relative risk of dying is constant across the age range, and coefficients are estimates of the magnitude of that relative risk (for a particular social category compared to some reference group).[5] Alternative multivariate approaches that express and analyse mortality differentials in absolute terms may be worth exploring as well.

There are also important choices about the variables used in a multivariate analysis. Ideally, micro-data should be used to depict individual characteristics that may be correlated with mortality differences, but in many data sets only a limited number of variables are measured at an individual level. In such situations, one strategy is to link the micro-data to aggregate data from another source, and to use the aggregate information as a proxy for individual characteristics. For example, if a micro-data set contains a race variable for all individuals but no information about income, it may be possible to obtain an income proxy in the form of the mean or median income of the census tract where an individual resides. However, this approach often results in biased estimates of individual-level effects and must be applied with caution (Geronimus, Bound, and Neidert 1996).

ALL-CAUSE VS. CAUSE-SPECIFIC MORTALITY

Most studies of mortality differentials in the United States have focused on all-cause mortality. However, for testing various hypotheses, it is essential to analyse differences in cause-specific mortality as well. Since the total mortality rate at a given age is merely the sum of cause-specific rates, including information on cause of death does not usually require a fundamentally different method of analysis.

At younger adult ages in the United States, it is particularly important to consider the contribution of violent causes of death (accidents, homicide, suicide) to difference in mortality by social class. At older adult ages, these causes may be important as well, but their importance is eclipsed by the major causes of death at older ages. Since much of the

[5] As noted already, mortality differentials tend to converge at older ages. This fact makes the proportional hazards assumption questionable in general, although it may still be useful and appropriate for analyses over a limited age range.

mortality decline of recent decades is attributable to the reduction in cardiovascular disease mortality at older ages (CDC 1999), it is especially important to analyse the contribution of mortality from heart disease and stroke to trends in mortality differentials among the elderly.

Summary of Key Results

In this section we summarize key results from studies of differential mortality in the United States. We also highlight the main points of controversy and suggest directions for future research.

EXPLANATIONS OF DIFFERENTIALS BY MARITAL STATUS

Mortality is lower among married people than among their single, divorced, or widowed counterparts in many countries. In general, the mortality differential by marital status is larger for men than for women and, when expressed as a relative risk, tends to diminish with age. Divorced persons usually experience the highest levels of mortality compared to married, single, or widowed individuals (Hu and Goldman 1990). Unmarried persons who are poor experience higher mortality risks than would be predicted on the basis of their marital status and economic conditions alone (Smith and Waitzman 1994).

Mortality differentials by marital status have been attributed to the "protective" and the "selective" effects of marriage, or to a combination of these two factors. Married people may have lower death rates (1) because they enjoy greater social support in general and especially in times of need, (2) because the presence of family members may dissuade risky behaviours like excess smoking and drinking, and/or (3) because the social networks provided by marriage and family may improve access to information about health and to medical services (Gove 1973; House, Karl, and Umberson 1988; Smith and Waitzman 1994; Murray 2000). In addition, departures from the married state, through either widowhood or divorce, are often stressful and may have negative health impacts (Goldman, Korenman, and Weinstein 1995; Lillard and Waite 1995).

However, it is also possible that mortality differentials by marital status result from healthy people being more likely to get married. Some studies have examined the correlation between the proportion of the population that is single or divorced and the level of excess mortality for these groups (compared to married persons). It has been argued that a negative correlation provides evidence for selection, since a low proportion of single or divorced suggests that individuals in these states are highly selected (Livi-Bacci 1985; Kisker and Goldman 1987). Although this finding has been replicated in large international comparisons, this particular interpretation is dubious, since it is also possible that the rarity of single and divorced persons in a society contributes negatively to their health status by increasing their social isolation (Hu and Goldman 1990).

In the analysis of marital mortality differentials, the problem of making accurate causal inferences about the relative contribution of selection versus protection based on aggregate data is probably insurmountable (Goldman 1993). An alternative strategy is to examine

mortality differentials by marital category in longitudinal studies that include measures of health status. By controlling for an individual's health status at baseline, it is possible to determine the magnitude of mortality differentials by marital status corrected for the selective effects of marriage on health.

A longitudinal study of elderly persons in the United States during 1984–1990 based on the Longitudinal Study of Aging (LSOA, a nationally representative sample of the non-institutionalized population aged 70 and over in 1984) found that marital status usually had only modest effects on mortality. An exception was found among widowed males, who showed a measurably higher risk of death (Goldman, Korenman, and Weinstein 1995). Surprisingly, never-married elderly women were found in this study to have better health outcomes than their married counterparts. This finding says little about the selective effects of marriage, since most marriages in this population had occurred many years earlier. The authors note, however, that "elderly singles have a distinct advantage over their formerly married counterparts since they have not experienced the stress and changes in the social and economic environment associated with both divorce and widowhood" (Goldman, Korenman, and Weinstein 1995:1727). It is also possible that long-time single people are better prepared to cope with the loneliness and social isolation of old age compared to their counterparts who were married for most of their lives.[6]

Lillard and Panis (1996) employ the longitudinal Panel Study of Income Dynamics (PSID) to address a complicated set of questions about the relationships between marriage and health. They consider not only the possibility that marriage may select positively for good health, but also that there may be adverse selection if less healthy people seek out marriage because of its perceived benefits (social support, care giving, etc.). In an analysis using structural equations modelling, they conclude that both types of selection occur commonly in the male marriage market (they consider men only). Positive selection dominates overall and among never-married men in particular, and thus it usually accentuates the mortality advantage due to the direct effects of marriage. However, adverse selection dominates among divorced men over age 50 who remarry, and thus it counteracts to some extent the favourable effects of marriage on the observed mortality differential.

TRENDS IN DIFFERENTIALS BY EDUCATION

Ever since the study by Kitagawa and Hauser (1973), studies of mortality differentials in the United States have favoured the use of educational attainment as the primary indicator of social class. Some advantages of this choice have been mentioned already. Obviously, educational attainment influences other key socioeconomic variables, such as income and wealth. Furthermore, educational attainment is relatively stable after early adulthood and is influenced very little by later changes in health status. For these reasons, a key question has been whether mortality differentials by educational level have been increasing or decreasing in recent decades in the United States.

[6] Grundy (this volume) suggests that the apparent reversal of health status at older ages between never-married and married older women may be due in part to the exclusion from the analysis of the institutionalized population.

Feldman *et al.* (1989) address this question by comparing the NHEFS[7] data for years 1971–84 to Kitagawa and Hauser's results for 1960. There are some differences in the type and quality of the data underlying the two studies. Kitagawa and Hauser's data were characterized by relatively poor match rates (see above) but are based on a sample of the entire U.S. population. Although the NHEFS data have better match rates, they exclude the institutionalized population. Feldman *et al.* conclude that, in general, educational differences in mortality increased over this time period for White men but did not change for White women. However, the educational differentials derived from the NHEFS have fairly large standard errors (due to a small sample size), and these errors are nearly large enough to obscure the existence of an educational gradient in mortality altogether. This, combined with the fact Kitigawa and Hauser did not report standard errors, makes any trend analysis based on these two sources rather problematic.

Pappas *et al.* (1993) use the larger NMFS (numerator) and NHIS (denominator) data sets from 1986 to ask the same question, again in comparison to Kitigawa and Hauser's results. Similar to Feldman *et al.*, they find that an index of educational dissimilarity in mortality rates increased over time for all population subgroups. This uniform increase leads to the conclusion that educational differences in mortality have widened over time. However, no standard errors are reported, so again the statistical certainty of the finding is unclear.

Preston and Elo (1995) attempt to settle this question by using the much larger NLMS data set for 1979–1985 (i.e., version one of the public-use NLMS). They point out that the Pappas *et al.* data are subject to numerator–denominator bias. However, NLMS data are also subject to biases due to exclusion of the institutionalized population and, possibly, differences in rates of linkage (using the NDI) between social groups. Leaving aside these non-sampling errors, the much larger sample size allows for greater precision in the estimation of mortality rates.

The estimates of Preston and Elo generally lie within the confidence region of the Feldman *et al.* rates but have much lower standard errors. On the other hand, these estimates yield lower educational differences than the Pappas *et al.* estimates. Considering several measures of educational inequalities in death rates, Preston and Elo conclude that differences have probably narrowed over time for White women but widened for White men. The authors express doubts about the exact magnitude of these changes because of the imprecision of Kitagawa and Hauser's results (due to the relatively low match rate). However, they conclude that the direction of the trends would not change given plausible adjustments to the earlier data.

Whereas most studies express mortality differences in terms of the relative risk of death, some authors have computed educational differences in life expectancy at different ages. Rogot *et al.* (1992a), using the full NLMS for 1979–1985, compute life expectancy by

[7] NHEFS is part of the National Health and Nutrition Examination Study (NHANES) and stands for NHANES Epidemiologic Followup Study.

educational attainment for both men and women. Based on a graphical comparison to the findings of Kitagawa and Hauser, these authors concur with Preston and Elo that there was a slight widening of differentials for White men and a slight narrowing for White women. Curiously, the magnitude of life expectancy differences by education varies widely depending on the study. For instance, Rogot *et al.* (1992a) show differences in life expectancy at age 65 (from lowest to highest educational group) of 3.3 years for men and 2.4 years for women. However, comparable differences based on an analysis of the National Long-Term Care Study were 2 years for men but a remarkable 7.6 years for women (Manton, Stallard and Corder 1997).

Probably the most interesting finding that emerges from this body of literature is the differing trends in educational differences in mortality by sex. Why has the gradient increased for men but decreased for women? No satisfactory explanation seems to be available in the literature, so this topic remains an important area for future research.

TRENDS IN DIFFERENTIALS BY INCOME

The traditional focus on trends in mortality differentials by educational attainment may or may not be warranted. While education is undoubtedly a primary determinant of economic status through its impact on occupation and wages, it is debatable whether educational attainment is indeed the proximal cause of social differentiation affecting mortality risks. Why not examine income or wealth? Wealth measures are notoriously difficult to obtain and measure, so the majority of evidence about mortality differentials pertains to differences in income. Nevertheless, two studies seem to indicate that mortality differences by wealth may be at least as important as those by income, suggesting that further research would be warranted (Menchik 1993; Attanasio and Hoynes 2000).

Duleep (1989) uses CPS data linked to records from the Social Security Administration (CPS-SSA) to compare relative mortality differences by income during 1973–78 to those documented in Kitagawa and Hauser. Due to data limitations, results are limited to white males aged 25–64. Relative mortality differences by income appear to have widened over the period. Whether absolute differences and differences in life expectancy widened over the period cannot be determined from the article.

Pappas *et al.* (1993) and Schalick *et al.* (2000) use the NMFS (numerators) and NHIS (denominators) for various years to examine whether mortality differences by income have increased or decreased over time. As noted previously, data that draw deaths and exposures from different sources are subject to numerator–denominator bias, but if this bias is similar for the two periods, the trend should be unaffected. Using Kitagawa and Hauser's estimates for 1960 as a baseline, the Pappas *et al.* study concludes that relative mortality differences by income widened between 1960 and 1986 for all major population subgroups (by race, sex, etc.). Again, issues of statistical uncertainty are unresolved, if only because we do not know the reliability of the 1960 results of Kitagawa and Hauser. The study by Schalick *et al.* uses data from the 1967 and 1986 versions of the NMFS and the NHIS. Their findings are sensitive to the measure of inequality employed. Using an absolute measure it appears that mortality differences by

income have decreased, whereas using a relative measure such differences have increased.[8] Since mortality declined over the period, even a moderately large widening of relative differences can be consistent with a narrowing of absolute differences.[9]

EFFECTS OF SPECIFIC OCCUPATION ON MORTALITY RISKS

An early study of occupational differences in mortality showed substantially higher mortality levels among labourers than among non-labourers, but it found few differences between large (and heterogeneous) groups of non-labourers (Moriyama and Guralnick 1956). Kitagawa and Hauser (1973) also documented substantial mortality differences by occupation, but their data did not permit a multivariate analysis that would have separated the effects of income, education, and other factors from those due directly to a person's occupation.

More recent studies have investigated more thoroughly the relationship between occupational mortality differentials and other socioeconomic factors (Moore and Hayward 1990; Johnson, Sorlie, and Backlund 1999). A key finding is that most mortality differences by occupational category can be explained by accompanying levels of income and education. However, some specific occupations present elevated risks beyond those predicted by a person's socioeconomic position: taxi drivers, cooks, longshoremen, and transportation operatives. Other occupations present unusually low risks: lawyers, natural scientists, teachers, farmers, and a variety of engineers (Johnson, Sorlie, and Backlund 1999).

Moore and Hayward (1990) make an important substantive and methodological point about the need to study occupational careers rather than merely a person's current occupation. "For example, men in physically demanding and debilitating occupations may move to other less risky occupations before dying" (p. 32). Aside from this selection mechanism, there may be other factors at work as well. The authors find, for example, that a person's mortality is negatively related to the substantive complexity of his longest occupation and to the physical and environmental demands of his current or latest occupation.

Another study about the effects of specific occupations concluded that "writers die young" compared to comparable professions (Kaun 1991). The study, which was based on a random sample from an unnamed source, found that writers have a mean life span about ten years less than other creative artists (artists, cartoonists, composers, conductors, dancers, musicians, singers, painters, and photographers). However, this difference reduces to about seven years after adjustment for sex and date of birth, which is still quite remarkable. The author attributes the elevated mortality risk to the fundamental character of a writer's work, which "provides little, if any, short-term satisfaction, and to the contrary, some fair amount of pain" (p. 397).

[8] Their absolute measure was the "slope index of inequality," while their relative measure was the "relative index of inequality" (see also Preston and Elo 1995).

[9] Preston and Taubman (1994) suggest that we should discuss mortality in terms of "goods," such as survivorship, rather than "bads," such as mortality. Given that the absolute difference in mortality rates narrowed, we can infer that the ratio of survival rates also narrowed.

DIFFERENTIALS BY NATIVITY, OR PLACE OF BIRTH

In the United States, mortality among the foreign-born tends to be lower than among native-born persons, but not in all cases. Because some race/ethnic groups are composed of a high fraction of foreign-born persons, observed race/ethnic mortality differentials are strongly influenced by nativity status. Hummer *et al.* (1999) provide an excellent overview of the relationship between nativity and race/ethnic mortality differentials.

Among non-Hispanics, foreign-born adults (above age 25) have been found to have a lower risk of death across the age range, especially among men. Using the full NLMS, Sorlie *et al.* (1993) show that non-Hispanic foreign-born adult men have death rates that are from 16 to 55 percent lower than their age-matched native-born counterparts. For women, this differential is smaller (from 7 to 28 percent) and is statistically significant only above age 45.

On the other hand, among Hispanic adult men a foreign-born mortality advantage exists only above age 45, and foreign-born Hispanic women show a statistically significant mortality advantage (compared to native-born Hispanics) only in the 45–64 year age range. However, adult Hispanics as a whole tend to have relatively low mortality compared to non-Hispanics, except perhaps among young adults (Sorlie *et al.* 1993). Thus, although there is less difference between native-born and foreign-born Hispanics in terms of their mortality profiles, both groups are advantaged compared to the general population at older adult ages.

Most Blacks in the U.S. are native-born and the mortality differential between them and most other population groups is relatively large (see below). However, the minority of foreign-born Blacks appears to be a highly advantaged group. In their study of Black and White residents of New York City, Fang *et al.* (1997) found that foreign-born Blacks had the lowest adult mortality of the four groups considered (foreign- and native-born Whites and Blacks). Along other indicators of social status, such as employment rates and educational attainment, foreign-born Blacks were again found to be the most favoured of these four group.

An important aspect of the relationship between nativity and mortality is that it tends to differ across the age range. Hummer *et al.* (1999) report that foreign-born Hispanics tend to have higher death rates at ages 15–44, even after controlling for socioeconomic factors, but that this relationship reverses itself at older ages. Rosenwaike (1983) found such a reversal among Puerto Rican immigrants living in New York City, and a similar pattern has been noted among the foreign-born in France as well (Brahimi 1980).

It seems likely that foreign-born individuals are selected for a relatively high health status at the time that they immigrate. Swallen (1997) finds that foreign-born residents in the U.S. have low mortality compared to persons in their homeland, even for sending countries like Japan or the United Kingdom where health conditions are favourable. Nevertheless, young immigrants face a variety of disadvantages as they attempt to establish themselves in the labour market and in other social arenas. Therefore, it is primarily at older ages, when the disadvantages of social and economic integration have been overcome, that the selective effects of immigration are reflected fully in the mortality profile of the foreign-born.

EXISTENCE OF DIFFERENTIALS BY RACE OR ETHNICITY

Mortality differentials by race have been known for a long time in the United States. The Black-White difference is especially large but has diminished in recent decades. National life tables (based on vital statistics and census data) show a difference in life expectancy at birth of 8.3 years between Blacks and Whites around 1950, which dropped to 6.9 years around 1990.[10] At age 30, the Black-White difference in life expectancy fell more modestly, from 5.9 years around 1950 to 5.6 years around 1990 (NOVS 1954; NCHS 1997). The existence and general magnitude of these differences are well confirmed, at least for recent decades, by studies using matched records (e.g., Kaufman *et al.* 1998; Elo and Preston 1997; Behrman *et al.* 1991; Sorlie *et al.* 1992; Rogers 1992). Furthermore, mortality differentials by race are accompanied by well-documented differences in health and morbidity (Manton and Stallard 1997).

The causes of mortality differences by race are only partially understood, and some empirical characteristics of these differences are still open to dispute. For example, the difference in mortality between Blacks and Whites diminishes with age and, arguably, may even reverse directions at older ages (see discussion of the mortality crossover below). Across the age range, this racial difference in mortality is accounted for at least in part by differences in socioeconomic status (SES) between Blacks and Whites, but the fraction of the entire differential that should be attributed to inequality in SES is uncertain (see below). There is no strong evidence that racial mortality differentials in adulthood are due to genetic differences between racial groups (Neel 1997).

It is difficult to measure mortality for racial or ethnic groups in the United States other than Blacks and Whites because of inconsistencies in the reporting of these designations in vital statistics and census data, resulting in the numerator–denominator bias mentioned earlier. For these groups, the only reliable means of estimating mortality is linked records, which ensure that race or ethnicity is classified by the same method in both numerator and denominator. Since such studies were rare before 1980, reliable information on mortality levels among Hispanics and Asians is available only for the most recent decades (Barringer, Gardner, and Levin 1993; Sorlie *et al.* 1993; Liao *et al.* 1998). Reliable mortality estimates for Native Americans are still difficult to obtain (Young 1997; Snipp 1997).

Much like Blacks, most Native Americans were born in the United States. Their cultures have a long history of economic marginalization and mixed success in assimilation. There are several notable features that emerge from the study of Native American mortality. First, the data problems are fairly severe (Snipp 1997; Young 1997). Native Americans are not easily identified in national health statistics, and mortality estimated from such sources is severely underestimated (Sorlie *et al.* 1992, as reported in Young 1997). For this reason, the most widely used source of information is data collected directly by the Indian Health Service (IHS), which only covers those Native Americans living on reservations and using the IHS. However, since many Native Americans do not live on reservations, the IHS data

[10] Technically, data for 1950 are for non-Whites.

are not representative of the entire population and thus mortality estimates from this source may well be biased.

The Native American mortality experience is in many ways similar to that of Blacks. Young adult mortality is much higher than the White population, principally due to more deaths due to external causes, especially accidents (Young 1997; Kunitz 1990). Pathologies associated with alcohol and diabetes are also well documented. At middle and older ages, where these causes of death are less important in relative terms, Native American mortality converges to that of Whites. At the highest ages (above 65 years), measured mortality is around 80% of White levels due to exceptionally low death rates from cancer and heart disease among Native Americans. However, the same issues of age misstatement that apply to the Black population are important here as well (see below).

Both the Hispanic and Asian populations contain a significant share of first- or second-generation immigrants from a variety of nations. Thus, they probably form more heterogeneous groups than Black Americans. As noted already, Hispanic and Asian mortality must be considered alongside differences in mortality by nativity (Hummer *et al.* 1999; Rogers, Hummer, and Nam 2000).

The mortality difference between Hispanics and non-Hispanics is especially intriguing because it seems to defy the conventional wisdom, which states that socially disadvantaged minority groups should have higher mortality than the majority population. However, mortality estimates based on matched records demonstrate that Hispanics in the United States enjoy low levels of mortality in spite of their socioeconomic disadvantage, a phenomenon that has been called "an epidemiological paradox" (Markides and Coreil 1986). For example, during 1979–1987 mortality above age 25 among Hispanic Whites was about 74 percent (men) or 82 percent (women) of the level for non-Hispanic Whites (Sorlie *et al.* 1993). In the same study, the Hispanic mortality advantage was observed for major causes of death like cancer and cardiovascular disease, although Hispanics were more likely to die from diabetes and homicide (men only).

The Hispanic mortality advantage is most pronounced at middle and older ages. At younger adult age (25–44 years), Hispanic status is associated with no apparent advantage (Sorlie *et al.* 1993) or higher mortality (Hummer *et al.* 1999; Rogers, Hummer, and Nam 2000). Some authors have proposed that Hispanic mortality at older ages is lower than for the rest of the U.S. population because sick immigrants may return to their home countries to die (Rosenwaike 1983). However, this hypothesis seems to be contradicted by the fact that U.S.-born Mexican Americans also display lower old-age mortality than other native-born non-Hispanic Whites (Hummer *et al.* 1999). Likewise, Cuban Americans, who are unlikely to return to Cuba, show lower mortality as well (Sorlie *et al.* 1993).

Mortality among Asian Americans is generally lower than among Whites (Barringer, Gardner, and Levin 1993; Hummer *et al.* 1999; Gardner 1980; Rogers, Hummer, and Nam 2000). This has been known at least since the study by Kitagawa and Hauser (1973), who documented the lower mortality of Japanese and Chinese Americans. This advantage has been observed over all adult ages and for all major categories of cause of death. Although there is considerable SES diversity among Asian sub-populations (e.g., recent

Hmong immigrants versus native-born Japanese Americans), Asians have on average similar levels of income and higher levels of education compared to Whites, which should yield at least a slight mortality advantage (other factors being equal). Furthermore, since mortality (at least at older ages) tends to be lower for the foreign-born, and since a substantial fraction of Asians living in the United States are immigrants, lower mortality among Asians is not surprising. Nevertheless, even after conditioning on socioeconomic factors and nativity, an Asian mortality advantage is still evident (see below).

THE BLACK–WHITE MORTALITY CROSSOVER

Even though Blacks are more likely to die at younger ages, their death rates at older ages appear to be lower than for Whites. Two explanations for this phenomenon, known as the Black–White mortality crossover, have competed for acceptance during the past few decades. One perspective holds that the crossover is merely an artifact of faulty age data. Indeed, it is well known among demographers that consistent age exaggeration (i.e., an upward bias in reported age) results in a downward bias in estimated death rates at older ages (Coale and Kisker 1990). It has also been shown, however, that even random age misreporting (with no consistent upward or downward bias) also results in underestimates of death rates at these ages. This occurs because misclassification of deaths from younger to older ages is numerically more significant than misclassification in the other direction, due to the rapidly diminishing tail in the age distribution of deaths (Preston *et al.* 1996). Because age data for elderly Blacks are known to be less accurate than for elderly Whites, this explanation of the origin of the mortality crossover is at least plausible.

Nevertheless, another plausible explanation suggests that the Black–White mortality crossover is real and not merely an artifact of poor measurement. According to well-specified mathematical models, it is possible that the elderly survivors of a high-mortality group can be more robust (and thus have lower mortality) than the survivors of a low-mortality group if three conditions hold true: (1) the two populations are internally heterogeneous in terms of mortality risks (i.e., members of each group have variable risks of dying across the age range); (2) the relative mortality risk for an individual (compared to some average mortality profile) is constant over age; and (3) mortality levels differ widely at younger ages. Under these conditions, the ordering of the two groups in terms of their mortality experience could reverse itself at older ages, yielding a crossover such as observed for Blacks and Whites in the United States (Manton and Stallard 1981).

An important criticism of this explanation is that such models do not usually account for the potential effects of scarring on the elderly survivors of the high-mortality group, even though some research points toward the impact of early life conditions on old-age mortality (e.g., Barker 1998; Costa 1998). Nonetheless, it seems at least possible that the effects of scarring could diminish with age or be dominated by the effects of mortality selection. Another possibility is that the survivors of a group that is disadvantaged in younger life could enjoy lower mortality at older ages because of better coping skills developed in the face of adversity—an example of social hormesis, as mentioned earlier. In short, the existence of mortality crossovers by race in the United States remains an intriguing though unproven hypothesis.

Indeed, it is difficult and perhaps impossible to find positive proof for the mortality selection hypothesis, since there is no simple means of measuring the differential risks of mortality among individuals within population sub-groups. For this purpose, it would be necessary to obtain *in vivo* physiological measurements that are complete and reliable predictors of subsequent mortality risks. With such measurements, it would be possible to compare the relative importance of mortality selection and physical scarring in the aftermath of adverse health conditions during early life. Unfortunately, studies of this sort are difficult to conduct, and a clear link between specific physiological parameters and individual mortality risks has been only partially established. Therefore, we lack a direct and convincing demonstration that the Black–White mortality is in fact due to mortality selection, even though mathematical models demonstrate that such an outcome is theoretically possible.

On the other hand, there have been important empirical studies addressing the hypothesis that the crossover may result from faulty data. A careful study of beneficiary records from the Social Security Administration suggested that the Black–White crossover was real at least above age 90 (Kestenbaum 1992). It is believed that age reporting in the Social Security records is more accurate than in vital statistics and census data, so the presence of a mortality crossover in these data is important evidence that the crossover phenomenon may be real.

Other researchers have studied the accuracy of age reporting on death certificates during the 1980s by matching these records to census information for the same individuals from the late nineteenth or early twentieth centuries (Preston *et al.* 1996; Hill, Preston, and Rosenwaike 2000). This technique suggests that inaccurate measurement is the cause of the Black–White mortality crossover at least below age 95. At the very highest ages, however, the crossover remains even after cleaning the data by this method. The authors suggest that this apparent difference may be real, or it may be due to random variation given the small number of cases above age 95 (Hill, Preston, and Rosenwaike 2000).

In conclusion, the issue of the Black–White mortality crossover remains an open question, although the extent of the phenomenon, if it exists at all, is surely much smaller than originally claimed. A narrowing of racial differentials in mortality at older ages is not implausible, since governmental support systems in the United States help alleviate social inequality in general among the elderly (much more so than at younger ages). However, a reversal of the differential requires that mortality selection and social hormesis dominate physical scarring in terms of their combined impacts on old-age mortality. Unfortunately, the mechanics of such a theory are difficult, if not impossible, to test empirically.

ROLE OF SOCIOECONOMIC STATUS IN RACIAL OR ETHNIC DIFFERENTIALS

As noted earlier, Blacks tend to have relatively high levels of mortality compared to Whites or to the American population in general. Conversely, Asians and Hispanics seem to have a more favourable mortality experience. The literature appears to indicate that these racial differences interact with socioeconomic status in different and somewhat unpredictable ways, so we treat these issues separately in this section.

A common finding is that the level of excess mortality among Blacks decreases after controlling statistically for socioeconomic status. In other words, when the level of income

or educational attainment is held constant, the mortality difference between Blacks and Whites is smaller than for the two groups as a whole. This reflects the fact that Blacks tend to have lower levels of income and education, which are associated with higher levels of mortality. In addition, it has been noted that mortality differences by income and education are generally larger than those by race (Navarro 1991).

Existing studies do not agree on the question of whether Black–White mortality differences can be explained by variation in socioeconomic status. Some authors (Menchik 1993; Lantz *et al.* 1998) find that excess mortality among Blacks disappears (i.e., becomes statistically insignificant) after taking account of differences in SES. On the other hand, some studies with larger samples (Sorlie *et al.* 1992; Rogers 1992; Sorlie, Backlund, and Keller 1995; Hummer *et al.* 1999) find that Blacks have a statistically significant mortality disadvantage even after controlling for SES. Nevertheless, the magnitude of this disadvantage is reduced by at least 40% when socioeconomic variables are taken into account.

This issue is even more complicated, however, if we consider the interaction between race and SES for different major causes of death. For example, the Black–White mortality differential for homicide and some forms of cancer can be explained largely by differences in SES (Onwuachi-Sanders and Hawkins 1993; Greenwald *et al.* 1996). On the other hand, cardiovascular mortality differences by race contain an important residual element that cannot easily be attributed to SES or observable risk factors (Escobedo, Giles, and Anda 1997). Even within a single cause of death, such as stroke, the size of the racial mortality difference may differ according to income level (Casper, Wing, and Strogatz 1991).

It is important to note that the Black–White mortality differential diminishes with age whether or not one controls for SES. Thus, studies that lump together broad age groups tend to underestimate the excess mortality of Blacks at younger ages and overestimate this quantity at older ages. Similarly, the income-mortality gradient appears to be less steep for Blacks than for Whites, so that the racial differential can vary depending on the income class being studied (Kaufman *et al.* 1998).

If the Black–White mortality gap cannot be explained by differences in socioeconomic status, a portion of this differential must be due to race-specific personal behaviours and characteristics, and another portion to social factors, such as racism and segregation. Relevant personal behaviours and characteristics may include diet, smoking, driving patterns, exercise, and weight, which vary across race/ethnic groups as shown in Table 1 (note that the values in this table are not adjusted for either age or SES).

Although Asians as a group have lower mortality than the White population, they also have a more favourable distribution of SES characteristics.[11] This suggests that the Asian mortality advantage will be reduced if SES is taken in account. However, even after accounting for

[11] If nativity is considered part of SES, then Asians have a profile that definitely favours lower mortality compared to Whites, since a higher proportion of Asians are foreign-born. On the other hand, if nativity is not included in SES, Asians have slightly lower incomes but much higher levels of education compared to Whites, so on balance Asians still have a slight advantage compared to Whites in terms of SES.

Table 1. Percent who report particular risky health behaviours, by race-ethnic group and nativity, United States, 1990.

	Asian Americans		Mexican Americans		Other Hispanics		Non-Hispanic blacks		Non-Hispanic whites	
	FB	NB	FB	NB	FB	NB	FB	NB	FB	NB
No breakfast	15.4	15.3	14.3	28.7	13.6	28.1	17.4	22.9	18.7	23.1
Overweight	6.2	11.7	31.2	32.4	27.2	24.0	29.4	36.9	22.0	24.5
No seat belt	6.3	5.5	15.3	16.4	16.8	16.7	12.8	17.3	12.4	17.1
Light smoker	13.2	3.8	13.4	18.2	15.8	16.3	9.6	17.4	9.6	9.2
Heavy smoker	4.0	7.2	3.8	6.0	6.6	8.9	1.2	9.0	11.6	16.4
Heavy drinker	4.0	5.8	12.1	14.5	8.4	11.4	6.2	7.2	7.7	9.8
No exercise	58.7	47.5	71.8	57.9	70.5	58.3	71.0	65.4	70.5	57.8

Note: FB = Foreign Born; NB = Native Born.
Source: NCHS, 1993 (reproduced from Hummer *et al.* 1999)

differences in SES and nativity, Asian mortality is still about 30% lower than among Whites (Hummer *et al.* 1999). Therefore, just as SES and other factors work together to create higher mortality in the Black population, the two sets of effects combine to produce lower mortality in the Asian population.

The Hispanic mortality advantage is more complex. Hispanics have lower mortality in general, in spite of less favourable socioeconomic characteristics. Therefore, the Hispanic mortality advantage after controlling for SES is in fact larger than the observed mortality differential (Sorlie *et al.* 1993; Hummer *et al.* 1999).

How important are personal behaviours and characteristics in accounting for the mortality differentials by race and ethnicity that remain after controlling statistically for differences in SES? On average, the health behaviours and characteristics of Blacks resemble those of Whites, with the notable exceptions of a higher prevalence of obesity and a lower frequency of exercise. Asians seem to have more favourable health behaviours than Whites (see Table 1). It is unclear whether smoking is more or less common among Hispanics compared with non-Hispanic Whites (as summarized by Sorlie *et al.* 1993). Mexican Americans are more likely to be overweight and heavy drinkers than non-Hispanic Whites (see Table 1).

Lantz *et al.* (1998) find that controlling for health practices has only a minor impact on that portion of the Black–White mortality difference not attributable to SES. For this reason, it would be somewhat surprising if health practices explain the Asian or Hispanic mortality advantage (net of SES). Feldman *et al.* (1989) include health behaviours in proportional hazard regressions where heart disease is the outcome variable and where education is a key explanatory variable. If these health behaviours were important channels through which education affected heart disease, their inclusion in the regression should reduce educational differences in the outcome variable. However, including health behaviours in the analysis has only a minor impact on measured educational effects. This finding seems to suggest that the risks and benefits linked to a person's educational level, and possibly to

other SES characteristics, race, and nativity as well, operate through channels other than the commonly observed health behaviours, as summarized in Table 1.

If personal characteristics do not explain the non-SES effects of race/ethnicity on mortality, then perhaps social factors operating at an aggregate level are at play. The most obvious social factor affecting all such minority groups in the United States is a shared experience of segregation and discrimination. Indeed, race-based residential segregation has often been cited as a contributing factor in the excess mortality of Blacks (Williams 1999; Polednak 1993; Polednak 1996; Potter 1991; Jackson *et al.* 2000), as discussed in the next section.

EFFECTS OF COMMUNITY, OR PLACE OF RESIDENCE

It is well established that the risk of death is related to the socioeconomic characteristics *of individuals*. However, it is also possible that the average socioeconomic status *of a community* has a direct impact on the health and mortality of individuals residing in that community. The average socioeconomic well-being of the community may affect the quality of the housing stock, the availability of medical care, the level of exposure to environmental toxins, and various risky behaviours (e.g., drinking, smoking, drugs, violence).

An analysis of data from the National Longitudinal Mortality Study suggests that, although the effects of individual socioeconomic position are clearly more important, the socioeconomic status of the community "makes a unique and substantial contribution to mortality" in the United States (Anderson *et al.* 1996). Similarly, in a study of mortality by census tracts, Guest *et al.* (1998) document a positive correlation between unemployment, non-completion of high school, and community mortality levels. Finally, Polednak (1993) finds that the Black–White mortality ratio is positively related to the Black–White poverty ratio and the degree of residential segregation across metropolitan areas.

On the other hand, Daly *et al.* (1998) find that the income distribution of a community has little effect on the mortality risks of individuals after controlling for personal SES. Likewise, Jackson *et al.* (2000) study the association between the degree of segregation[12] in a census tract and individual mortality risks controlling for family income. However, it is difficult to interpret their findings, which indicate that the relationship between segregation and mortality is positive for some groups and negative for others.

The relationship between community of residence and mortality is by no means a simple one. It was noted already that residential segregation and associated social pathology could be contributing factors in the relatively high mortality of Blacks in the United States. On the other hand, Hispanic communities in the U.S. are thought to provide a supportive social environment that contributes to good health. These two results may seem contradictory. Apparently, social isolation of groups can lead to either worse or better health outcomes depending

[12] Jackson *et al.* (2000) measure segregation as the proportion of the census tract that is Black. A high percentage of Blacks in a census tract is taken as evidence of higher segregation, suggesting that an all-White neighbourhood is perfectly integrated whereas an all-Black neighbourhood is wholly segregated. In spite of these objections, it is possible that the measure serves as a good proxy for segregation in general and for the potentially negative effects of segregation on health and longevity in particular.

on the circumstances. We speculate that the difference hinges on the causes of the social isolation experienced by the group in question. For African Americans, residential segregation is the result of a long history of social and economic marginalization by the mainstream society, whereas for Hispanics (and other recent immigrant groups) it reflects a social cohesiveness associated with trying to become part of a new land. There are many subtleties to be considered in both cases, and this contrast should be explored in greater depth.

Another example of the effect of community, or place of residence, is the urban-rural mortality difference. In their landmark study, Kitagawa and Hauser (1973) showed that age-adjusted total mortality rates in 1960 were 5 percent higher in urban counties compared to rural ones, with the highest risk of mortality being found in urban counties that contain a central city. This finding seems anomalous in view of the fact that residents of rural areas tend to be poorer and less well educated on average than city dwellers. In addition, medical services are more widely available in cities. Nevertheless, other factors may help to make city life less conducive to good health. For example, urban social relationships may be more formal and impersonal, leading to anomie, social isolation, and psychological stress; in addition, overcrowding may contribute to the spread of infectious disease (Miller, Voth, and Danforth 1982).

Data from the NLMS confirm that a rural mortality advantage was still present in the United States during the 1980s (Smith *et al.* 1995). After adjusting for age and sex, it was estimated that rural residents had death rates that were about 7–9 percent lower than residents of central cities. This relationship did not change substantially after further adjustment for race/ethnicity, income, education, and marital status. In contrast, residents of suburban districts or urban areas outside central cities also showed an age-sex-adjusted mortality advantage of about 4–7 percent compared to those in central cities, but these differences become smaller and lose statistical significance once controls are added for the same socioeconomic variables.

Thus, residents of central cities experience higher mortality risks compared to all other residential groups in the United States, but these differences with respect to other residents of non-rural areas (i.e., suburbs and outside the central city) appear to be mostly a function of socioeconomic status. The rural mortality advantage, on the other hand, does not disappear after adjusting for socioeconomic differences and thus requires another explanation. Possible protective effects of rural residence may include a more favourable social and physical environment and fewer risky behaviours. Another possibility is that people with serious health problems move from rural areas to cities in order to be closer to health facilities (Smith *et al.* 1995). To our knowledge there has been no adequate empirical confrontation of these two hypotheses (which can again be labeled "protection" versus "selection") in the published literature.

The interaction between poverty, race, and place of residence is well documented by Geronimus *et al.* (1999) in their study of mortality at ages 15–64 among poor Black and White communities in the urban North and the rural South.[13] At least two key findings

[13] The authors acknowledge that their study design cannot distinguish simultaneously between the effects of North vs. South and urban vs. poor because of population size requirements.

from their analysis are worth noting here. First, poor Blacks in the rural South display a much smaller mortality disadvantage compared to poor Blacks in the urban North at comparable levels of socioeconomic status (even after adjustment for differences in the cost of living). A similar but reduced finding is reported for poor Whites. As a result, the magnitude of the rural mortality advantage among the poor appears to be much larger than among the population as a whole. Whereas the latter is usually less than 10 percent, the study by Geronomius *et al.* suggests that the former is at least 20 percent and sometimes more than 100 percent. Second, perhaps contrary to expectations, the excess mortality of these poor communities (compared to Whites nationwide) is not due primarily to the widely publicized causes of homicide and AIDS. Rather, most of the excess mortality in these areas in the age range of 15–64 was due to higher levels of chronic disease, in particular cardiovascular disease.

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