Sources of Increasing Differential Mortality among the Aged by Socioeconomic Status

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Several studies have attempted to explain the rising mortality differential by SES for the general population. One of the largest studies is by Steenland et al. (2002), which reported a comparison of cause-specific death rates by educational level in two surveys sponsored by the American Cancer Society. The surveys, covering deaths in 1959-72 and 1982-96, showed an inverse correlation between schooling and age-adjusted death rates among those 45 and older and all causes of death except breast cancer. More important, the authors found increasing mortality differences by educational attainment across the two surveys, and the growing differences were particularly marked for coronary heart disease among men. However, results for a variety of cancers were more mixed. Cutler et al. (2010) examined the role of behavioral risk factors in accounting for the increased education differential. They found that smoking, obesity, high cholesterol, and hypertension played only a small role. Their results were particularly striking given that smoking and obesity are the two leading behavioral causes of death and are, of course, linked to SES. In sum, the source of the increased mortality differential remains uncertain and controversial.

We use the longitudinal data from the HRS to explore the relationship between SES and the different causes of death as they relate to older persons. The HRS also includes responses to a set of behavioral questions, similar to those used in the study by Cutler et al. (2010), the respondent's self-reported health status, and the age of the parents at death. From previous work,

¹ Studies of the correlation between SES and mortality are particularly numerous dating back to Kitagawa and Hauser (1968), but the evidence of a causal relationship is more limited and controversial. For example, see the discussion in Cutler, Deaton, and Lleras-Muney (2006) and a recent study of the effects of education on mortality by Clark and Rover (2013) for Great Britain.

we know that the pattern of increasing mortality differentials among older Americans can be seen in the HRS sample. The HRS interviews and associated data files contain a great deal of information on SES, work history, health status, causes of death, and other items that are crucial to identifying the links between SES, on the one hand, and causes of poor health and death, on the other.

First, we develop a basic empirical model of mortality that takes account of birth year, age, income, and education as alternative measures of SES. The interaction of birth year and SES is used to measure the change in differential mortality over time. Second, we employ the model to measure the relationship between SES and the different causes of death. The model is estimated as a simple logit relationship and as a multinomial logit regression. Third, we add the behavioral measures, parental life expectancy, and initial health status to determine if these additional variables can account for the pattern of increasing differential mortality.

Our results replicate previous findings with regard to increases in relative mortality differences among Americans with high and low socioeconomic status. Whether SES rank is measured by educational attainment, actual average career earnings between ages 41 and 50, or predicted career earnings between 41 and 50, we find that the more recent birth cohorts have relatively larger mortality differences favoring high-SES individuals compared with earlier cohorts. Second, our results indicate that the diverging mortality experiences of high- and low-SES Americans are not attributable to growing differences in just one or two causes of death. Among women in the HRS, for example, the relative mortality of low-income and low-education women climbed compared with women in higher SES groups because of relative increases in deaths attributable to heart conditions, digestive system issues, and several other causes. Thus, it is hard to point to a single cause of death that has produced the majority of the relative decline in mortality among high-income women.

Third, when we include indicator variables that reflect individual differences in genetic endowment, in behaviors such as smoking and alcohol consumption linked to early mortality, and respondents' health status at the time of enrollment in the HRS sample, we find these variables are highly significant in predicting individual mortality. With only one exception, however, these additional variables do not materially reduce the estimated size of increases in mortality differences by SES group. That is, even though most of the variables are highly significant in accounting for individual differences in mortality, they do not explain the growth

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in relative mortality differences over time. An important exception is the variables that reflect respondents' health or disability status at the time of their enrollment in the HRS. When these variables are included, the apparent increase in relative mortality among low-SES groups is reduced. Of course, respondents' health status at enrollment is itself linked to socioeconomic status.

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