Illness, Not Age, Is the Leveler of Social Mortality Differences in Old Age

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Objectives. Although mortality differences between groups of different socioeconomic status (SES) are known to decrease at higher age, the reasons for this are unknown. This study reviews the existing arguments for convergence versus divergence of SES mortality differences in old age, differentiates between increasing age and worsening health, and discusses explanations for the observed convergence.

Methods. Register data of all Danish men aged ≥59 years between 1980 and 2002 (n = 938,427), and survival analysis, are used to examine mortality differences between income groups, controlling for seven variables. Interaction models describe the change of SES mortality differences with age or health, days in hospital being a proxy for the latter.

Results. Large mortality differences were found with relative risks of 0.28–0.36 for income groups above the 25th income percentile compared with those below the 10th percentile. Mortality differentials are stable across age groups but converge when health deteriorates.

Discussion. The idea of “age as leveler” has to be revised because it is illness and not age that levels SES mortality differences. That the transition from poor health to death is independent from SES suggests that social mortality differences originate in early stages of the health deterioration process. Intensive treatment for very sick people will not decrease SES mortality differences.

Key Words: Denmark—Diversity in aging—Event history methods—Mortality—Socioeconomic status.

Although differences in health and mortality between socioeconomic groups exist in all age groups, most studies report that relative differences decline in old age (e.g., Andersen & Laursen, 1998; Martelin, Koskinen, & Valkonen, 1998; Elo & Preston, 1996). However, a simple age pattern of mortality differences is prone to bias. The question as to whether (and why) mortality differences between socioeconomic groups decline in old age is related to several highly debated theoretical and methodological factors that are briefly addressed subsequently; for more details, see Hoffmann (2008). The present study focuses on one aspect of this broader question by exploring whether increasing age (age as leveler) or declining health (illness as leveler) lie behind the convergence. An answer to this question will allow better evaluation of the factors that determine the age pattern of socioeconomic mortality differences.

At least four arguments have been proposed to support a mortality convergence at old age. First, physical decline works as a leveler of social differences because biological processes assume dominance over social determinants and, eventually, everybody dies regardless of social class (Liang et al., 2002). The underlying assumption is that mortality in old age is generally more biologically and genetically determined than in young ages (Mayer & Wagner, 1996). Second, the welfare state reduces differences in socioeconomic status (SES) in old age through social policy and benefits that favor the elderly population, thereby contributing to some redistribution between social groups (Bassuk, Berkman, & Amick, 2002; Knesebeck, Lüschen, Cockerham, & Siegrist, 2003). This effect of the welfare system decreases health inequality either fundamentally by reducing social inequality or at the level of health outcome only through health care itself. This has been claimed by Preston (1984) and further discussed by Dupre (2007) for programs such as Medicare in the United States. Third, the impact of earlier stratifying and relevant health experiences (e.g., working conditions) fades out at old age because most elderly people disengage from the main stratifying systems (House, Kessler, & Herzog, 1994). For example, Marmot and Shipley (1996) report a decrease in social mortality differences after retirement, rather than related with age. Fourth, declining differences are observed on the aggregate level only because the surviving population is more homogenous due to unobserved heterogeneity and selective mortality (Vaupel, 2001). The compositional change in the surviving population (that includes strong survivors from the lower SES groups) creates a downward bias of SES mortality differences because mortality in the low SES group then becomes similar to that in the high SES group. This is one possible explanation for mortality convergence (House et al., 1994) but the methodological implications are not addressed in the present study (see Discussion). It is noteworthy that the selection argument differs from the other three, in that it does not claim a decreasing impact of SES on health at the individual level but advocates to differentiate between the individual and the aggregate level (Dupre, 2007).
Arguments against mortality convergence also need to be considered. First, vulnerability increases in old age and makes differential exposures more harmful. The impact of health risks may thus increase, outbalancing decreasing differences in exposures (House et al., 1994; Stronks, 1997). Second, past experiences (e.g., education) accumulate and may interact with other factors, such as economic and social capital. The health outcome of this accumulation is incorporated in the “health stock.” Dannefer (1987, 2003) has elaborated on this principle and described a social theory of cumulative advantage, also called the “Matthew effect” (Beckett, Goldman, Weinstein, Lin, & Chuang, 2002; Lynch, 2003). Third, the impact of past unhealthy experiences (e.g., bad working conditions or smoking) is postponed until older ages, implying a time lag between an experienced disadvantage and its effect on health inequalities (House et al., 1994; Lauderdale, 2001).

Compared with numerous findings of convergence, few studies have found divergence or at least stable differences. For example, Otterblad Olausson (1991) found increasing mortality differences, whereas stable mortality differences were reported by Fox, Jones, and Moser (1985) and Huisman and colleagues (2004). Increasing educational health differences were found by Ross and Wu (1996) and Lynch (2003), and stable health differences were shown in some countries by Huisman, Kunst, and Mackenbach (2003). There are several reasons for these inconsistent findings: Otterblad Olausson admits that her results might be influenced by a cohort effect, that is, the older cohorts in her sample experienced more economic inequality in their childhood. Besides cohort effects (also with regard to health behavior), differing results can be due to (a) the use of different measures for predictors or outcome variables, for example, health versus mortality, (b) different study populations whereby each health care system may have a different impact on the age pattern of health and inequality, and (c) different levels of inequality at the starting age, which may determine the move toward convergence or divergence. The age pattern of the observed SES mortality differences might be the net effect of several of the above-mentioned mechanisms working simultaneously.

In the present study, we first present the pattern of socioeconomic mortality differentials over age for Denmark. Then, interaction models are applied to reveal whether the observed convergence of social mortality differences is attributable to increasing age or to worsening health. The answer to this question will allow more valid evaluation of the above arguments for convergence, in particular those relying on increasing age (welfare state policy and fading out) versus the argument of physical decline. In the light of these findings, new interpretations are suggested for health policy and for understanding the interplay between SES, health, and aging.

### Table 1. Descriptive Statistics of the Sample (Frequencies in Percent of Total Person-Years)

<table>
<thead>
<tr>
<th>Income</th>
<th>Days in hospital</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentiles</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>0–10</td>
<td>4.5a</td>
<td>0–3</td>
</tr>
<tr>
<td>10–25</td>
<td>11.4</td>
<td>4–7</td>
</tr>
<tr>
<td>25–50</td>
<td>20.6</td>
<td>8–14</td>
</tr>
<tr>
<td>50–75</td>
<td>26.4</td>
<td>15–30</td>
</tr>
<tr>
<td>75–90</td>
<td>20.2</td>
<td>31–61</td>
</tr>
<tr>
<td>90–100</td>
<td>16.8</td>
<td>&gt;61</td>
</tr>
<tr>
<td>Not known</td>
<td>0.1</td>
<td>Not known</td>
</tr>
</tbody>
</table>

Note: The percentages of income for men do not correspond to the size of the percentile groups because the limits for the income groups are calculated from the total sample including both men and women.

### Methods

Data were used from the Danish Demographic Database that combines different national registers from 1980 onward. Danish registers can be linked by an individual person identification number, they cover the entire population, and provide annual information (Petersen, 2000). Our time to event data set includes individual information on 938,427 men aged 59 years and older, observed between 1980 and 2002, that is, for 23 years (Table 1). The data set for each individual includes multiple entries and an indicator variable in case of death. Because men turning 59 years after 1980 are included, the present sample includes cohorts born between 1874 and 1939.

The present study is restricted to the analysis of men only because (a) in these specific cohorts, women have a much lower likelihood of having their own income and (b) higher SES mortality gradients for men suggest that relevant dimensions of SES and their mechanisms differ slightly between gender (detailed analysis of gender differences in the social differentiation of aging, health, and mortality is beyond the scope of this study). Income is used as an indicator for SES. This decision is based on data showing that income is more strongly associated with mortality than with education (e.g., Herd, Goesling, & House, 2007), especially in higher ages (Goldman, Korenman, & Weinstein, 1995). In addition, earlier results emerging from the Danish data (Hoffmann, 2008) show that the largest mortality differences exist between income groups and only small differences exist between educational and/or occupational status groups. Moreover, the relatively small mortality differences between educational groups are substantially reduced by controlling for income, whereas the rate ratios for income are robust against the inclusion of other dimensions of SES in the model (results not shown). Furthermore, the variance in
education for people born in Denmark before World War II is low and formal degrees of education are difficult to interpret. Finally, after retirement, occupational status as a measure for SES is not a good alternative to income. For the present study, individual gross annual income is used and measured in six percentile groups (0–10, 11–25, 26–50, 51–75, 76–90, and 91–100). The limits of these groups are defined by actual amounts of income in Danish Kroner, calculated from the income distribution of each of the 23 years of observation. Because income distribution changes considerably with age, age-adjusted income percentiles were also used which, however, had no effect on the results (results not shown).

The seven control variables are operationalized as follows: “Education” is measured based on years of schooling, divided in four categories (0–7, 8–11, 12, and 13+ years). “Occupational status” is measured in six categories as helper, unskilled manual, skilled manual, nonmanual, self-employed, and unknown. The number of “children” is a binary indicator for children living in the household. “Source of main income” has the categories pension, early retirement, self-employed, and unknown. The number of “children” is measured in six percentile groups (0–10, 11–25, 26–50, 51–75, 76–90, and 91–100). The limits of these groups are defined by actual amounts of income in Danish Kroner, calculated from the income distribution of each of the 23 years of observation. Because income distribution changes considerably with age, age-adjusted income percentiles were also used which, however, had no effect on the results (results not shown).

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Risk of dying. All models control for age and include interaction effects between income and age or between income and health. The baseline for age covers the age range 59 years up to the highest age, whereas the observation period is only 23 years (1980–2002). Thus, these are (in part) synthetic cohorts; cases coming under observation older than 59 years of age are left-truncated, implying that they are observed only after other members of the same cohort have already died. Stata takes this into account by distinguishing between “time under risk” (starting at age 59 years) and “time under observation” (starting at the individual age of entry; Gutierrez, 2002). No bias due to period or cohort effects was found; this was tested by including a variable for period and, in a second test, by including a variable for the birth year (both in 5-year categories). In these alternative models, no significant change was found in the magnitude of mortality inequality or in its pattern over age.

RESULTS

Table 2 presents mortality rate ratios for the Danish elderly population. Very large differences in mortality between income groups exist mainly in the poorer part of the income distribution. In the upper 75% of the income distribution (the four richest groups), only relatively small mortality differences are seen, as well as some changes in the association between income and mortality. Figure 1 shows these mortality differences in interaction with age. All age groups show a large mortality advantage in the richer groups compared with the poorest group: The upper 75% of the income distribution have a homogenous mortality level that is 40–80% lower than the poorest 10% (reference group). The second poorest group is in the middle. Only the poorest 25% of Danish elderly men have a clear mortality disadvantage. However, for the question addressed in the present study, it is important to note that mortality differences clearly narrow with increasing age.

Although the x-axis in Figure 1 (age) implies both increasing age and worsening health, the model for Figure 2 includes the variable “days in hospital” and, therefore,
partly controls for health status and health deterioration. It can be seen that the convergence virtually disappears and mortality differences between income groups are almost stable across age groups. This means that increasing age alone, while controlling for days in hospital, does not cause SES mortality differences to converge in old age. Figure 3 shows the complementary model, an interaction between income and days in hospital, and shows the effect of declining health on SES mortality differences while controlling for age. As can be seen, the SES mortality differences converge with worsening health. The more sick a person is, the less impact the social status has on mortality. After separating the two dimensions of aging (increasing age and worsening health), it turns out that illness, and not increasing age, is a leveler for SES mortality differences.

**DISCUSSION**

This study shows that, among elderly men in Denmark, mortality differences between income groups are very large and that these differences narrow with increasing age. However, the main factor behind this narrowing is the decline in health and not increasing age. In the situation of a poor health status, income has almost no influence on the transition to death. A similar result was reported by Klein and Unger, that is, the impact of income on mortality is much higher in a good health status than in poor health (Klein & Unger, 2001). The main implication of this finding is that the notion of age as leveler (Dowd & Bengtson, 1978; Dupre, 2007; Ferraro & Farmer, 1996) is not specific enough because it is not age that levels SES mortality differences but illness.

Even if it is plausible to assume that increasing age is generally related to worsening health, these two dimensions should be kept separate for analytical purposes, as was advocated by Kelley-Moore (2010). Age increases for everyone but decline in health varies widely between different social groups. Although both these factors increase mortality, they have different consequences for the impact of social status on mortality. A meaningful interpretation of this finding is not that health levels social differences in general but that it only does so after health has been influenced by SES to a certain extent. In many cases, health decline is not exogenous but is dependent on SES. This perspective leads us to regard declining health as a process open to social and other influences. However, the more health deteriorates, the less this process is influenced by SES. Our finding does not confirm the plasticity of aging and mortality that was claimed for very high age groups by Vaupel, Carey, and Christensen (2003); these authors found that after German reunification, in Eastern Germany even people aged 90+ years experienced mortality improvement due to improved nutrition, pensions, and health care. However, Vaupel and colleagues also examined age groups—and not health groups. Thus, it seems important to differentiate between high age and poor health when studying the impact of SES on mortality.

Although our results allow tentative evaluation of the four arguments presented in the introduction, the findings and related interpretations emerging from this Danish context cannot be simply extrapolated to other countries. Our findings do not support the hypothesis of an equalizing effect of welfare state policy on mortality inequality among the elderly population (the second argument for convergence) because increasing age alone does not seem to be a factor that causes mortality differences to converge. For the same reason, the temporal distance to unequal health experiences (e.g., during working life) and a fading out of the effect of these unequal factors (the third argument for convergence) is not plausible. We found that mortality differences between income groups are stable across age and that it is poor health that levels social mortality differences, possibly as a result of a universal shift from social to biological determinants of mortality when health declines (the first argument for convergence). It follows that arguments based on age alone as a leveling factor are not confirmed whereas the idea that, once an illness has developed then social differences become much less important, appears to be correct. With regard to the selection hypothesis (the fourth argument...
for convergence), the present study did not aim to test this hypothesis and more complex methods are needed for this (see Beckett, 2000; Beckett & Elliott, 2001; Crimmins, Jung, & Seeman, 2009; Dupre, 2007; Ferraro & Farmer, 1996; Hoffmann, 2008). Nevertheless, our findings suggest that the convergence shown in Figure 1 is not due to mortality selection because the convergence is removed by controlling for days in hospital (Figure 2). If the convergence was due to selection, it would still be visible in Figure 2 because the sample in the higher age groups in Figure 2 is just as selective as the sample in Figure 1. Admittedly, it is not possible to simply verify or rule out the various arguments by the use of one empirical example. In the present study, mortality selection may well have a converging influence, and the parallel lines in Figure 2 would diverge without mortality selection (which, in turn, would support the arguments for divergence). The mechanisms described in the introduction are not mutually exclusive; for example, accumulating social differences and the dominance of poor physical conditions over social conditions can occur simultaneously, and we may be observing the net effect of both mechanisms. However, the clear difference in results when controlling and not controlling for days in hospital reveals the important role of worsening health.

Two conclusions relevant for policymaking can be drawn from this study. First, if SES does not have a large impact on the mortality of sick people but we observe large overall SES mortality differences, this means that SES has its effect early on in the process of health deterioration. Socioeconomic differences in health and mortality originate in the period of prevention and the treatment of mild illnesses; therefore, this is the period in which interventions will be the most promising. Or, in relation to items measured in the present study (i.e., income), salutary things that “money can buy” for healthy people in everyday life in order to stay healthy seem to be important. On the other hand, differences in the quality of medical services for serious illnesses and access to costly treatment and “high-tech” solutions do not seem to play an important role with regard to inequality in mortality.

A second possible interpretation of Figure 3 refers to the mutual reaction between the medical system and the patient, depending on the patient’s health status. In a good or medium health status, patients have no (or only occasional/voluntary) contact with medical services. At this stage, SES plays a greater role in the process of health decline and subsequent mortality (left side of Figure 3) because effective prevention and early treatment is not the standard case. The patient might have to request and pay for early treatment. On the other hand, those with serious illnesses who have been hospitalized for many days eventually receive the full range of high-quality services irrespective of their social background. This might be because even noncompliant patients finally undergo the necessary treatments or because health insurance companies only pay out and service providers only earn sufficient money, when the patient is already very sick. This interpretation would imply a serious dysfunction of incentives and a misallocation of resources in a medical system that underestimates the value of prevention.

One limitation of the present study is the health indicator used. Although Danish register data are generally considered among the best data sets available for mortality studies, days in hospital is not the best health indicator. Other variables, such as self-rated health or physical functions, are desirable but not available in the Danish registers. However, tests and comparisons with data from the Health and Retirement Study (HRS) from the United States show that days in hospital are a sufficient health indicator. First, they strongly predict mortality with and without control for SES and, second, in the HRS data, the variable days in hospital provides results very similar to self-rated health and objective health measures based on functional limitations (results not shown). Strictly speaking we can neither exclude nor measure a potential bias based on less access and less use of hospitals by poor people. However, this social difference in hospital use is likely to be small in Denmark because hospital admission does hardly depend on individual wealth due to a health care system with universal and comprehensive coverage. Van Doorslaer and colleagues (2000) show that in Denmark people with low income spend more days in hospital but when one controls for health status, this difference disappears completely. Given the clear difference between Figures 1 and 2 and the strong convergence shown in Figure 3, it is unlikely that this potential bias can explain our results. After additional testing, we found that, although hospital days generally decrease over time, our results are not biased by period effects in this measure of health.

In conclusion, this study reveals an important dimension regarding the impact of SES on health, namely a person’s health status. Health deterioration is an important continuum for the medical perspective on physiological processes, where more advanced health deterioration seems to imply increased independence from external factors. In addition, from a sociological perspective health deterioration is also a continuum that starts in private (healthy) life (where the individual is sovereign but preventive resources depend on SES) and ends in severe illness and submission to a wide range of medical treatments and services. Based on the present study, the explanation for SES health differences lies in earlier stages of this process. Additional studies are needed to test the empirical basis of these findings and to confirm the accuracy of these interpretations and conclusions.

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References


